**Autoimmune Diseases and Human Health: An updated insight**

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**Running Title:** Autoimmune diseases and human health

**Abstract**

Autoimmune diseases and disorders are not the same thing. While the underlying aetiology of each illness is different, the immune system dysfunction that underpins both disorders may produce similar symptoms. One important distinction is that autoinflammatory disorders are characterised by a breakdown of the innate immune system, whereas autoimmune illnesses are characterised by an obstruction of the adaptive immune system. The hallmark of autoimmune/inflammatory diseases is systemic or organ-specific inflammation that causes tissue destruction. The prior categories for autoimmune/inflammatory conditions were auto inflammatory and autoimmune illnesses.Certain disorders, like lupus, which exhibit family aggregation and may suggest a genetic predisposition, are suggested to be the result of a complex interplay between genes and environment. There is evidence linking autoimmune diseases to clinical symptoms. Measuring auto antibodies in patients may help with diagnosis and severity analysis, which might be important for treatment. This chapter has primarily addressed treatment strategies for several autoimmune conditions, including systemic lupus erythematosus, rheumatoid arthritis, and myasthenia gravis.

**Keywords:** Autoimmune diseases, diabetes, rheumatoid arthritis, lupus erythematous, auto antigen

1. INTRODUCTION

Autoinflammatory illnesses and autoimmune disorders belong to different classes. The immune system failure that underlies both conditions might result in comparable symptoms including weariness, rash, and swelling, but the underlying etiology of each disease is distinct (Scherlinger et al., 2020). One significant difference between autoimmune diseases and autoinflammatory disorders is that the former are defined by a breakdown of the immune system's capacity to adapt, while the latter are characterised by an obstruction of the innate immune system

(Leo et al., 2020). The hallmark of autoimmune/inflammatory illness is systemic or organ specific inflammation that causes tissue destruction (Evert et al., 2003). Autoinflammatory and autoimmune disorders were the previous classifications for autoimmune/inflammatory condition (Watad et al., 2017). But it soon become evident that the world is far more complicated than that, with autoimmune and autoinflammatory conditions essentially acting as extremes at either end (Tang et al., 2012).On the other hand, autoimmune disorders were described as being brought on by an immune system that adapts and, as a result, characterized by the presence and pathophysiology involvement of self-antibodies and/or populations of lymphocytes that are reactive to their own substances.(Fig. 1) (Satoh et al., 2016).



**Figure 1.** Different autoimmune diseases and their target organs.

The term "classical" autoimmune illnesses and monogenic autoinflammatory conditions represent opposite ends of a spectrum that represents systemic autoimmune/inflammatory disorders (Croft et al., 2019).

As a result of the constantly expanding list of known autoimmune/inflammatory circumstances, the previously discussed inter-individual fluctuations in phenotypes and results (sometimes even between individuals who have the same assessment and/or within family members), and the recognition that originally separate conditions may move together the inflammation frequency from, for example, a basically auto-inflammatory to an autoimmunity characteristics (this can occur repeatedly in grown-up Still's disease and systematic JIA, for example), the molecular mechanism of autoimmune/inflammatory circumstances is complicated and only partially understood.(Swinkels et al., 2018). Based on current knowledge, autoimmune and inflammatory diseases are caused by either complicated genetic predispositions (more common) or monogenic disease causes (less common). Both types of diseases are influenced by both individual and environmental factors, which can change how the disease manifests itself and/or how specific phenotypes and outcomes are affected.(Wang et al., 2015).

Although the exact cause of autoimmune diseases is unknown, it is probably complicated and involves both genetic and environmental components (Fallah et al., 2014). A complicated interaction between genes and environment is implied in the etiology of some diseases, such as lupus, which show familial aggregation and may indicate a hereditary predisposition(Hainer et al., 2019). However, other cases have been linked to viral triggers or environmental variables.Epigenetic processes, a class of gene regulatory mechanisms, control the accessibility of chromatin to transcriptional regulatory proteins, hence modifying gene expression without changing the underlying sequence of DNA (Meers et al., 2019). Epigenetic processes are fluid but also heritable, can be influenced by the environment, and eventually account for a significant amount of variation among tissues and cells within an organism, even if all cells are diploid and have the same genotype (Simeonov et al., 2019). Examples of epigenetic processes include DNA methylation, non-coding RNA expression, and histone protein post-translational modifications (West et al., 2018).

2. HISTORICAL BACKGROUND OF AUTOIMMUNE DISEASES

Medical microbiology saw the emergence of immunology as a new specialty. Robert Koch's studies on the origins of infectious diseases—especially tuberculosis—and Louis Pasteur's findings, which validated the germ theory of infectious diseases, raise important questions.Does the host have a strong defence system or is it helpless against dangerous bacteria?(Lo et al., 2020). Working at Koch's Institute for infectious illnesses in Berlin, Behring and Ehrlich identified antibodies as essential counterparts to bacterial toxins, while Metchnikoff, who had been employed at the Pasteur Laboratory in Paris since 1888, found the essential function of bacterial infection and intracellular kill in host defence (Conigliaro et al., 2019).

Medical historians regard the mid-twentieth century as the point at which the scientific and medical societies recognized the presence of autoimmune illness (Ling et al., 2019). Several illnesses, including sympathetic ophthalmia andendophthalmitis phacoanaphylactica, were previously identified as autoimmune disorders (Kumagai et al., 2020). During the first part of the century, autoimmune illness was considered biologically improbable (Schreiber et al.,2019). The phrase "horror autotoxicus" was coined by Paul Ehrlich to emphasize how autoimmunity would contradict nature's tendency toward self-harm (Khan et al., 2020).

As to Fässler et al. (2019), the immune system's capacity for self-harm was initially demonstrated by the identification of allergies and anaphylaxis. Understanding the etiology of autoimmunity was hampered by a significant stumbling block: the immune system's ability to distinguish between self and foreign substances; this ability was eventually named immunological tolerance (Fig. 2) (Finkel et al., 2021).Investigators of sympathetic ophthalmia and endophthalmitis phacoanaphylactica were positioned to disprove horror autotoxicus, but there was insufficient convincing experimental and clinical data to do so (Zhou et al., 2020). In the 1950s, autoimmune illness gained widespread recognition following seminal investigations of chronic thyroiditis and a succession of clinical laboratory advances (Rendeiro et al., 2020). The difficulties experienced by ophthalmology scientists provide insights into how medical concepts develop (Hajishengalis et al., 2019). We examine how ocular immunology had a role in developing the notion of autoimmune illness and why it took time to gain popularity (Chua et al., 2020).

**Figure 2.** Background of autoimmune diseases. Cross reaction, mutation and intolerant are the main hallmarks.

As stated by Paul R. Ehrlich in 1901, an immune response that targets the "self" may cause harmful antibodies to be produced in the person (Schulte-Schrepping et al., 2020). The renowned "horror autotoxicus" he described showed that the body needed a defense mechanism to protect itself against the dangers of self-antibodies (van der Made et al.,2020).

Paul Ehrlich (1900) is regarded as one of the pioneers of contemporary immunology. He developed the side-chain hypothesis, which postulated that pathogens engage with cells' side chain receptors (Combes et al., 2021). His notion of an expanding antibody molecule with many binding sites for complement and antigen activation was the first one proposed (Shin et al., 2019). The concept of clonal selection was created in 1957 by Frank Burnet and David Talmage, after Astrid Fagraeus' 1948 identification of plasma B cells as primarily involved in antibody production (Defendi et al., 2020).

In the 1940s, fluid substances that attach to the radioactive antigenic and immunoglobulin G were discovered, leading to the creation of two types of antagonists (ANAs and RF)..There is evidence linking autoimmune diseases to clinical symptoms. Measuring autoantibodies in patients can help with diagnosis and severity analysis of the disease, which could be important for treatment (Bekkeringet al., 2018). Pauling's instructive theory from 1940, which held that the antigen serves as an illustration for the antibodies, was challenged by this argument, which stated that cells create just one antibodies component that determines the antibody before they encounter an antigen (van der Meer et al., 2016). Donath-Landsteiner haemolytic anaemia is an autoimmune illness that was first identified by Donath and Landsteiner (Wrap et al., 2020). One patient who developed paroxysmal cold hemoglobinuria following exposure to coldness in the arms or legs had an antibody, a blood component that attaches and destroys self-red blood cells (Hoey et al., 2019). August Wassermann observed in Berlin that syphilis patients' sera reacted with extracts of both sick and normal tissues, allowing autoantibodies to be identified (Lung et al., 2019).

3. CLASSIFICATION OF AUTOIMMUNE DISEASES

Numerous factors can be used to categorize autoimmune disorders.The site of the autoimmune attack is one of them, this criterion is used to differentiate between systemic and organ-specific autoimmune disorders(Bettacchioly et al., 2021). This artificial classification structure serves a practical purpose in helping patients and primary care providers find the right expert (Table 1) (Abu-Rumeileh et al., 2020).

**Table 1:**Autoimmune diseases with target organ and auto stimulate antigens.

|  |  |  |
| --- | --- | --- |
| Disease | Target Organ | Known auto antigens |
| Thyroiditis (autoimmune) | Thyroid | ThyroglobulinThyroperoxidase |
| Grave’s disease | Thyroid | Thyroid-stimulating hormone receptor |
| Type 1 diabetes | Pancreatic Beta cells | Insulin, GAD, IA-2 |
| Addison’s disease | Adrenal | 21OH hydroxylase |
| Gastritis | Stomach | 17OH hydroxylase |
| Celiac disease | Small bowel | H+/K+ ATPaseIntrinsic Factor |
| Vitiligo | Melanocytes | Transglutaminase |
| Multiple sclerosis | Brain, spinal cord | TyrosinTyrosinase-related protein-2 |
| Pemphigus | Skin | Myelin basic proteinProteolipid protein |
| Hepatitis (autoimmune) | Liver | Hepatocyte antigensCytochrome; p450-1 A2 |
| Myasthenia gravis | Muscle | Acethylcholine receptor |
| Primary biliary cirrhosis | Liver bile ducts | 2-Oxoacid dehydrogenase complexes |

3.1. Systemic Specific

When autoantigens are present in nearly every kind of cell in the body, such as DNA-protein complexes, the conditionisreferred to as systemic autoimmune disease (EASL et al., 2017). Consequently, a wide variety of organs and tissues are affected by the pathological damage. Common systemic autoimmune diseases include rheumatoid arthritis, scleroderma, dermatomyositis, and systemic lupus erythematosus (Nydegger et al., 2016).

3.2. Organ Specific

Immune system attacks that target a single organ or tissue preferentially are known as organ- specific autoimmune disorders.The skin in individuals with vitiligo, the beta cells of the endocrinefew examples.

### 3.3. Graves' disease

### Graves' disease is characterized by the development of self-antibodies targeting thyroid stimulating hormone (TSH) receptors (TRAb). Anxiety, irritability, a rapid heartbeat, and weight loss are examples of stimulatory effects that can result from excessive thyroid hormone production as well as release caused by the attachment of TRAb self-antibodies to the TSH receptor (Arulraj et al., 2021).

3.4. Diarrheal illness with inflammation

Under the general category of autoimmune bowl sickness (IBD), which is characterised by ongoing swelling of the digestive system, include Crohn's illness and ulcerative colitis (Annett et al.,2020).

### 3.5. Multiple sclerosis

### Multiple sclerosis (MS) is a form of neurodegenerative illness that destroys myelin, the coating that protects the fibers of nerves in the brain and spinal cord. It causes the immune system to target myelin cells (Soltani et al., 2019).

### 3.6. Rheumatoid arthritis

### The main target of rheumatoid arthritis (RA) is the joint, where it causes chronic inflammation that leads to discomfort and damage to the joints (Levine et al., 2018). It frequently has symmetry, so if one hand or knee has it, the other one also does.

3.7. Lupus erythematosus systemic

### An autoimmune disease called lupus, also known as systemic lupus erythematosus, affects the kidneys, liver, skin, joints, and neurological system, among other organs (Stojan et al., 2016). A generalized lack of immunological tolerance is one of its defining features.

### 3.8. Type 1 diabetes

Attacks by the body's immune system on the pancreas beta cells that make insulin elevate blood sugar levels and cause the disease referred to as type 1 diabetes (Lu et al., 2019). Among the symptoms include more thirst, more frequent urination, or unexplained weight loss..

3.9. Gluten sensitivity

A reaction of the immune system to the protein gluten, which is found in the grains rye, barley, and wheat, is termed celiac disease. When gluten is consumed, the small intestine may have an immune response that results in damage to the villi that line the gut and facilitate food absorption. (Zhang et al., 2017). Due to its transit through the gastrointestinal tract, which includes the oesophagus, the gastrointestinal tract, small and large intestines, rectum, and anus, eating gluten can increase the chance of developing gastrointestinal cancer (Alpert et al., 2019).

3.10. Psoriatic arthritis and psoriasis:

Rapid cell development that causes scaling on the skin's surface is a hallmark of the skin condition psoriasis. There is a lot of inflammation and redness around the scales (Mamoshina et al., 2019). Certain individuals with psoriasis may experience joint pain, stiffness, and oedema from psoriatic arthritis.

### 3.11. Sjögren's syndrome

### Sjögren syndrome, also known as SjS or SS, is an ongoing condition of autoimmune origin that primarily affects the salivary and lacrimal glands in the body. It also often has significant effects on other organ systems, including the kidneys, neurological system, and lungs.

3.12. Undifferentiated Disease of the Connective Tissue

### When a person does not fit the diagnostic criteria for any particular connective tissue disease, but still displays signs of the disease, such as blood test results and physical characteristics, this condition is known as undifferentiated connective tissue diseases (Rockwood et al., 2011). Over time, 30–40% of people get a specific connective tissue condition.

3.13. MS, or multiple sclerosis,

### MS is an autoimmune illness that damages the insulating coverings of nerve cells in the brain and spine.Damage to the nervous system can impair signal transmission and cause physical, mental, and psychiatric symptoms.Symptoms may include double vision, visual loss, eye discomfort, muscular weakness, and loss of sensation/coordination (Lehallier et al., 2019).The reason is unknown;

###  however, it is likely due to immune system damage or myelin-producing cell failure,there is currently no recognized cure for multiple sclerosis.Current therapies include disease-modifying drugs to reduce inflammation and the symptoms that accompany acute flares, as well as to prevent future episodes.Physical and occupational therapy, as well as patient-centered symptom management, can improve functional capacity.

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### Figure 3. Pathophysiology and different factors of autoimmune diseases.

4. MYASTHENIA GRAVIS

Myasthenia gravis is a fast and voluntary controllable muscle weakening and weariness.Antibodies that create a breakdown in nerve-muscle communication are the cause of the disorder. One of the most prevalent conditions affecting neuromuscular transmission is myasthenia gravis. It is one of the autoimmune illnesses that is now most understood and defined. A number of ophthalmic muscles, bulbar functions, limbs, and breathing muscles are affected, and it is marked by cyclical weakness and fatigue that results from an immune reaction to the postsynaptic membranes of the neuromuscular junction (Cohen Tervaert et al., 2018). Myasthenia gravis is diagnosed usinga combination of clinical and serological testing. With the help of the available treatment options, the illness can be successfully managed and even completely remitted.

5.1. Epidemiology

The body's immune system attacks neuromuscular junction components in myasthenia gravis (MG), a rare and persistent autoimmune disease that affects sensory to muscle signal transmission

(Imbach et al., 1981). Myasthenia gravis is characterised by weakness and fatigue in the active skeletal muscles, particularly in the face, jaw, eyes, neck, and limbs.

5.2 Incidence

In 55 investigations conducted throughout 1950 and 2007, the frequency rate of MG varied from 1.7 to 21.3, resulting in an entire pooled occurrence ratio for 5.3 per 1,000,000 persons annually. 29 additional research conducted between 2007 and 2019 discovered that the annual incidence rate of MG varied between 0.15 through 61.33 per 1,000,000 people (Sultan et al., 1984).2021 study estimates that for every 1,000,000 persons, there are between 4.1 to thirty instances of MG annually.

5.3 The occurrence

Although the stated prevalence of MG varies by place, there's has been an upsurge during the past 50 years. Globally, the prevalence rates range from 150 to 200 instances per million people.

* 1. Pathophysiology

 The most well-understood autoimmune condition is myasthenia gravis (MG), and research on the condition has contributed to a fundamental understanding of the mechanics behind neuromuscular transmission. Antibodies directed against the acetylcholine receptor (AChR) are the cause of myopathy (MG). By impairing the end-plate potential, these antibodies lower the safety factor for effective synaptic transmission

(Tian et al., 2021). It is evident that complement activation is necessary for the postsynaptic surface to be destroyed by AChR antibodies.Patients with MG who do not have antibodies against the AChR have been identified to have an antigenic target that is specific to muscle kinase. Although it is unknown how tolerance breaks down, T-cells are required for the development of autoantibodies in MG.

There is an intriguing variation in the participation of muscle groups in MG, with the extraocular muscles being particularly involved. This article examines the processes underlying the autoimmune process of myasthenia gravis (MG), normal neuromuscular transmission, and the varying susceptibilities of the ocular muscles to MG.

Classification of dogs and cats and congenital myasthenic disorders

One can either inherit or acquire myasthenia, a syndrome marked by reduced neuromuscular transmission. A group of genetic diseases with an early onset that impair the neuromuscular junction, also called the NMJ, of skeletal muscle are collectively known as congenital myasthenic syndromes (CMSs). These illnesses vary in therapy. Autoantibodies against the NMJ cause myasthenia gravis (MG), an acquired autoimmune disease. Regarding treatment and result, it is critical to recognise both illnesses as diseases.Reviewing the literature on MG and CMSs in pets such as cats and dogs, and then making recommendations for a classification system upon a comparison with existing human classifications for MG and CMSs in pets of all kinds (Signore et al., 2018). First, myasthenia gravis can present as acute fulminating, localized, or widespread in nature. The basis for subclassification is either the underlying cause of autoimmune disease or seronegativity.Cats receiving thiourylene medication or having a thymoma or not are associated with the underlying cause of autoimmune illness.

Congenital myasthenic disorders are classified according to the affected NMJ component, the involved mutant gene, the affected protein, and the biological cause of the neurological transmission failure. Intending to facilitate identification of the disease categories for both illnesses by presenting this classification of MG and CMSs as well as direct care, improve prognosis, and offer a foundation for more research on these disorders.

Causes

In order to communicate with your muscles, your nerves release chemicals known as neurotransmitters that attach to receptor sites on your muscle cells. This process is known as the nerve-muscle junction. When you have myasthenia gravis, your immune system makes antibodies that block or harm a lot of the acetylcholine (as-uh-teel-KOH-leen) receptors that are located in your muscles. Less accessible receptor locations mean fewer nerve impulses reach your muscles. This leads to weakness. Antibodies can also block a protein called MuSK (muscle-specific receptor tyrosine kinase; TIE-roh-seen KIE-nays). This protein helps to create the nerve-muscle junction(Ding et al., 2021). Antibodies designed to neutralize this protein may cause myasthenia gravis. Antibodies targeting lipoprotein-related polypeptide 4 (LRP4) may make this sickness worse. Through research studies, more antibodies have been found, and this number is likely to rise in the future.

Antibodies that block the LRP4, MuSK, or cholinergic do not cause myasthenia gravis to develop in certain people (Vulto et al., 2017). This form of the illness is known as seronegative myasthenia gravis, which is also occasionally called antibody-negative myasthenia gravis

Generally speaking, researchers continue to believe that autoimmunity contributes to this type of myasthenia gravis; they have simply not yet found the causative antibodies.

Thymus gland

One component of your immune system is the thymus gland. This gland is situated beneath the breastbone in the upper chest. The antibodies that obstruct acetylcholine are thought to be produced by the thymus gland or to have assisted in their production.In healthy adults, the thymus gland is small, but in newborns it is enormous. However, the thymus gland is larger than normal in some adults with myasthenia gravis. Thymomas, or tumours of the thymus gland, are another condition that some myasthenia gravis patients develop. Thymomas, also called malignant, are rarely cancerous. However, thymomas can develop into malignancy.

Among the things that can exacerbate myasthenia gravis are:

• Weary

• Disease or illness.

• Surgery.

• Tension.

• Certain medications, including beta blockers, antibiotics, quinidine gluconate, quinidine sulphate, quinine (Qualaquin), phenytoin (Dilantin), and several anaesthetics.

• Maternity.

• Menstrual cycles.

Symptoms

With usage of the affected muscle, myasthenia gravis induces muscular weakness that worsens. Since most symptoms of muscle weakness go better with rest, they can occur intermittently (Rajewsky et al., 2019). However, the symptoms frequently get worse over time. The worst of the illness usually strikes a few years afterwards it first manifests.

Myasthenia gravis could be your condition, and it could harm any controllable muscles in your body. The frequency of afflicted muscle groups varies.

Muscles within the eye

More than half of cases of myasthenia gravis typically start with ocular symptoms. A few of the signs and symptoms include

• The word for one or both pupils drooping is ptosis.

• Double vision, known as diplopia, happens if one of the eyes is closed and can be either upward or horizontal. It can either improve or disappear.

The facial and throat muscles

Roughly 15% of cases involve the face and throat muscles in the earliest symptoms of myasthenia gravis. These signs and symptoms may:

* Difficulty in speaking.
* Difficulty in swallowing
* Impact the chewing process; the chewing muscles may begin to hurt midway through a meal. This is especially true for those who have been eating tough foods like steak (Weinreichet al., 2020).
* Alter your facial expressions.
* The leg and neck muscles:

In addition, myasthenia gravis may cause weakness in the neck, arms, and legs. A person's gait may be affected by leg weakness. Weak neck muscles make holding up the head difficult.

Diagnosis

Neurological test

Testing could be one way your doctor examines your neurological health

• Adaptations.

• Strength of muscles.

• Tone of muscles.

• Touch and visual senses.

• Sync.

• Equilibrium.

The following tests could be performed to support a myasthenia gravis diagnosis

Test with an ice pack

Your healthcare professional may apply an ice bag to your eyelid if it is drooping. Your healthcare practitioner takes out the bag after two minutes and assesses your drooping eyelid for potential improvement.

Blood examination:

A blood test may identify abnormal antibodies that obstruct the protein receptor sites where your muscles receive contraction signals from your nerves.

Stimulating nerves repeatedly

In this nerve conduction study, the testing muscles' skin is covered with electrodes that the physicians place there. Electricity is applied in tiny pulses across the electrodes (Kohler et al.,2019). These pulses gauge the nerve's ability to communicate with the muscle.The nerve is put through multiple tests during this examination to see whether weariness impairs the nerve's capacity to convey impulses. The test's findings help identify myasthenia gravis.

Electromyography, also known as single-fiber

This examination looks at the electrical signals that travel from your brain to your muscles. It is necessary to put a small wire electrode via the skin and through a muscle in order to evaluate a single muscle fiber.

Visualising

prescribe an MRI or CT scan.

Tests for pulmonary function

These tests determine whether breathing is being affected by your disease.

Treatment

Medications

* Antagonists of cholinesterase

Medication like pyridostigmine (Mestinon, Regonal)enhances the nerve-muscle transmission. While these medications don't cure anything, they can help some people's muscles contract more forcefully and stronger.Intense salivation and perspiration, diarrhoea, nausea, and upset stomach are possible adverse effects.

* Corticosteroids

The corticosteroid prednisolone (Rayos) lowers the immune system's ability to produce antibodies and inhibits it (Adamson et al., 2019). On the other hand, prolonged use of corticosteroid might have major adverse consequences. These include diabetes, weight gain, deteriorating bones, and an increased risk of some infections.

* Immunosuppressive medicines

It is also possible for your doctor to recommend additional medications that alter your immune system. These drugs may include tacrolimus as (Astagraf XL, Prograf, among others), methotrexate (Trexall), selenium (Sandimmune, Gengraf, among others), or azathioprine (Azasan, Imuran). These drugs can be taken with corticosteroids; the half-life of the former is several months. Immunosuppressant side effects can include increased infection risk as well as liver or renal damage (Terwiel et al., 2019).

 Intravenous treatment

 The following treatments are typically administered briefly to address symptoms that worsen out of the blue or prior to surgery or other treatments

Plasmapheresis, pronounced "plaz-muh-fuh-REE-sis;

This method uses a filtering procedure that is similar to dialysis. A machine that removes antibodies from your blood stops messages from your nerve terminals from reaching your muscles. Nevertheless, the benefits of this surgery typically wear off within a few weeks (Llanos et al., 2019). Finding veins for therapy can become difficult after multiple surgeries.

Plasmapheresis carries some risks, such as bleeding, cramping in the muscles, irregular heartbeat, and blood pressure decline. In certain people, the solutions used to substitute the plasma cause an allergic reaction.

Intravenous immunoglobulin (IVIg)

Your body receives normal antibodies from this therapy, which modifies the reaction of your immune system. Benefits can last three to six weeks and are typically noticeable in less than a week. Chills, headaches, vertigo, and fluid retention are among the moderate side effects that are occasionally experienced.

* Antibody that is monoclonal

Myasthenia gravis patients are administered eculizumab (Soliris) and rituximab (Rituxan) intravenously. These drugs are typically utilised in cases where no other therapy is effective. They may cause major adverse consequences.

Operation

There are some myasthenia gravis sufferers who have thymus gland tumours. If you suffer from a tumor called a thymoma, you need to have the thymus gland removed, or a thymectomy. Eliminating the thymus gland may help alleviate symptoms even in the absence of a tumour. It may take years for this operation to start showing results, though.

There are two types of thymectomy procedures: minimally invasive and open. To access the chest and remove the thymus gland, an open surgical procedure involves the physician splitting the sternum, the middle portion of the breastbone. Minimally invasive surgery involves the removal of the thymus gland through tiny incisions.

6. RHEUMATOIDARTHRITIS

The term" Arthritis" means joint inflammation, joints are places where two bones meet. There are various types of arthritis present, Rheumatoid arthritis is one of them.Rheumatoid arthritis is a disease which causes irreversible joint damage and disability (Starshinova et al., 2018). Generally diagnosis is done by combination of both clinical and laboratory features. Patient's suffering from polyarthritis ofjoints of the hands and feet generally faces problems like early morning stiffness and sometimes constitutional symptoms. Rheumatoid factors are proteins that our immune system produces that might target healthy tissue within our body. In autoimmune illnesses including Sjogren syndrome and rheumatoid arthritis, elevated levels in the blood of rheumatoid factor are frequently observed. The normal range for rheumatoid factor levels in blood is 0–20 units per milliliter.

Risk factor

Factors influencing high risk of rheumatoid arthritis

1. Gender

 Women are mostly affected to Rheumatoid arthritis as compared to men.

1. Age

Generally, occurs in middle age.

1. Smoking

Smoking cigarettes increase high risk of rheumatoid arthritis,if one havea genetic disposition for the development of the disease.

1. Overweight

People with excess obesity may face high risk of developing rheumatoid arthritis.

Pathophysiology

* Antigen presentation to T cells.
* T and B cell Multiplicity in the synovial lining angiogenesis.
* Build up of neutrophils in synovial fluid.
* Initial formation of Pannus ( a sheet of granulation tissue containing inflammation, known as the Pannus, proliferates from the synovial membrane and invades the joint
* Erosion of subchondral bone.
* Invasion of cartilage by Pannus proliferation of chondrocytes.
* Ligament laxity.
* Reduced range of motion, contractures, joint instability and systemic problems.

Difficulties

* Osteoporosis risk is increased by rheumatoid arthritis, including medication-assisted treatment (weakening of bones that increases the risk of fracture).
* Arthritis nodules- Usually, the elbows and other pressure areas are where these rigid masses of tissue form. However, the body's nodules can form anywhere, including the heart and lungs.
* Many medications used to treat rheumatoid arthritis can decrease the immune system, which raises the risk of pathogens.Get immunized to protect yourself from illnesses including influenza, shingles, pneumonia, and COVID-19.
* Carpal tunnel syndrome - If rheumatoid arthritis affects our wrists, it may constrict the nerve that supplies most of our hands and fingers.
* Lung condition - Breathlessness that worsens over time is a possible consequence of lung tissue inflammation and scaring in rheumatoid arthritis patients.
* Thyroid cancer - Rheumatoid arthritis is linked to an increased risk of lymphomas, a family of blood cancers that start in the lymphatic system (Bhattacharya et al., 2024).

Indications and Manifestations

* Symptoms and indicators of rheumatoid arthritis can include:
* heated, swollen, and delicate joints
* stiffness in the joints, which usually grows worse after sitting still and in the morning
* Fatigue, fever, and loss of appetite
* Early-stage rheumatoid arthritis usually affects smaller joints first, notably those that connect your fingers and toes to your foot.Early-stage rheumatoid arthritis usually affects smaller joints first, notably those that connect your fingers and toes to your foot. Usually, the identical joint on each side of your body give you trouble.
* About 40% of people with rheumatoid arthritis also have non-joint indications and symptoms. Areas that could be impacted include:
* Seeing
* Respiratory
* Skin
* Coronary
* Glandular glands
* Nervous tissue
* The bone marrow
* Blood vessels

Rheumatoid arthritis symptoms and indicators can vary greatly in severity and occasionally come and go. Relative remissions, or periods when the illness's pain and swelling are reduced or eliminated, alternate with flare-ups, or periods of increased disease activity. Over time, rheumatoid arthritis may cause joints to shift and swell (Adhikary et al., 2024).

Diagnose

The most frequent systemic inflammatory arthritis to be diagnosed is rheumatoid arthritis. The most common groups afflicted are women, smokers, and anyone with a family history of the condition. Having at least one joint with noticeable swelling that cannot be attributed to another illness is a requirement for diagnosis. The more minor joints impacted, the higher the chance ofbeing diagnosed with rheumatoid arthritis. An inflammatory arthritis patient's elevated C-reactive protein level, erythrocyte sedimentation rate, rheumatoid factor, or anti-citrullinated enzyme antibody all suggest rheumatoid arthritis. Among the first laboratory evaluations should be a full blood count with differential and testing for hepatic and renal function. It is advised that individuals taking biologic medications get tested for hepatitis B, hepatitis C, and TB. Early treatment with disease-modifying antirheumatic medications is possible with an earlier diagnosis of rheumatoid arthritis.

Treatment

To manage the disease, drug combinations are frequently employed. The first-line treatment for rheumatoid arthritis is usually methotrexate. Tumour necrosis factor inhibitors, for example, are considered biologic medicines and can be combined for dual therapy. The goals of treatment include minimizing joint pain and swelling, preventing radiographic deterioration and visible deformity, and continuing with daily activities and employment (Starshinova et al.,2020). Joint replacement is recommended for patients with severe joint degeneration who have symptoms that are not adequately managed with medication.

7. SYSTEMIC ERYTHEMATOSUS LUPUS

Lupus, also known as systemic autoimmune lupus erythematosus (SLE), is a chronic (long-term) autoimmune disorder that can affect almost any part of the body, with the most frequent areas being the skin, joints, kidneys, liver, heart, lungs, skeletons, blood, and brain. An individual with systemic lupus erythematosus has an autoimmune illness, which means that their own immune system targets and damages their own healthy cells and tissues. No two persons with lupus have the same exact form of the disease because it can affect any organ system. Nonetheless, the majority of individuals suffering from lupus or systemic lupus erythematosus (SLE) report experiencing remissions or times when their symptoms appear tobe minimal or nonexistent, and flare-ups or relapses, which are marked by increased inflammation (Table 2).

**Table 2:**Various antigens their nature, prevalence and association with systemic lupus erythematosus

|  |  |  |  |
| --- | --- | --- | --- |
| Antigen | Nature | Prevalence in SLE | Association |
| Hep-2 cell nuclei | ANA | > 95% | Numerous autoimmune diseases |
| dsDNA | Native, double-stranded DNA | 40%-60% | High specificity for lupus, titers correlate with disease activity |
| Histones |  | 50%-70% | Drug-induced lupus |
| Sm | Small nuclear RNAs complexed with protein | 20% - 30% | High specificity for lupus |
| Nuclear RNP (U1 RNP) | Small nuclear RNAs complexes with protein | 30% -40% | $$MCTD$$ |
| SS-A (Ro) | Protein associated with RNA | 30% -50% | $Sjögren sy$ndrome, subacute cutaneous lupus, neonatal lupus with heart block, SLE with interstitial pneumonia |
| SS-B(La) | Protein bound to small RNA | 10% -15% | $Sjögren sy$ndrome |
| Ku | DNA binding proteins | 10% -39% | MCTD, scleroderma, primary pulmonary hypertension |
| Ki | Nuclear protein | 8%- 31% | Arthritis, pericarditis, and pulmonary hypertension in patients with SLE |
| PCNA/cyclin | Cell cycle protein | 3% |  |
| Hsp90 | Heat shock protein | 50% | Polymyositis |
| p ribosomal protein, rRNP | Ribosomal phosphoprotein | 10% | Neuropsychiatric SLE |
| ssDNA | Single-stranded DNA | 70% |  |
| β2-glycoprotein1 | Anionic proteins, cardiolipin | 25% | Lupus anticoagulant, arterial and venous thromboses, neurologic disease |

A systemic lupus genetic predisposition may be associated with several risk factors, such as the -DR3 haplotype, HLA-B8 haplotype, ultraviolet ( UV ) rays from sunlight, viral infection, particularly Epstein-Barr virus infection, hormones, toxins, and tobacco smoking.

The prevalence of mild along with more severe SLE is increased in smokers. Smoking also decreases the effectiveness of other therapies, such as antimalarial drugs.

Systemic lupus erythematosus types

1. Erythematosus lupus systemic - Systemic lupus erythematosus is the most common kind of lupus (SLE)
2. Cutaneous lupuserythematosus (CLE)

Skin-specific lupus or cutaneous lupus erythematosus (CLE) (which comprises subacute cutaneous form of lupus erythematosus (SCLE) and discoid lupus erythematosus (DLE) Systemically involved or not can be the presentation of skin lupus erythematosus (CLE).

1. Drug-induced erythematosus of the lupus (DILE)

A condition similar to lupus that is brought on by some pharmaceutical medications. Characteristics of drug-induced lupus erythematosus (DILE) set it apart from typical (idiopathic) SLE. For instance, DILE appears concurrently with drug exposure and disappears after treatment is finished.

1. Neonatal lupus erythematosus

A rare disorder known as neonatal lupus erythematosus affects newborns of lupus-affected women. Neonatal lupus refers to lupus that affects infants born to women who have the disease. Antibodies from the mother against anti-Sjögren's syndrome-related antigen A (also called anti-Ro; SSA/Ro) or anti-Sjögren's syndrome-related antigen B (also called anti-La; SB/La) cross the placenta and result in neonatal lupus, which is characterized by inheritable heart block, sensitivity to light rash, cytopenia, and liver anomalies (Nydegger et al., 1974).

1. Systemic lupus typically has onset in children or adolescents

Juvenile-onset systemic lupus erythematosus (JSLE) or child-onset lupus erythematosus. SLE, often known as "child-onset lupus" or "juvenile-onset systemic lupus," can also affect children. It has been separated into a distinct class because of the clinical distinctions between usual adult-onset SLE and child-onset SLE.

Complications from Lupus:

Acute atherosclerosis, or arterial blockage, brought on by lupus disease, especially when it's active, can cause cardiac events, kidney failure, and strokes in young women. As a result, in addition to treating their illness, lupus sufferers must exercise and cut down on additional risk factors for heart disease like high blood pressure, smoking, and excessive cholesterol. One of the most prevalent and dangerous symptoms of lupus is inflammation of the kidneys. Dialysis and renal failure may result if it remains undiagnosed. By obtaining treatment as soon as kidney disease manifests, you can contribute to preventing these dangerous consequences. Among these indicators are:

* Elevated blood pressure
* Swollen hands and feet
* Swollenness surrounding your eyes
* Urinating more frequently at night, having bleeding with foam in the urine, or experiencing discomfort or difficulty urinating are examples of changes in urination.)

Many parts of your body, including your: can become inflamed as a result of systemic lupus erythematosus (SLE).

Renal system

Lupus can cause significant kidney damage, including lupus nephritis, and failure of the kidneys constitutes one of the leading reasons of mortality for those with the condition.

The spinal nerve apparatus includes the cerebellum: The central nervous system and the brain. Lupus can cause brain damage that manifests as headaches, vertigo, altered behaviour, visual issues, strokes, or seizures. A lot of people with lupus experience loss of memory and may have trouble expressing their ideas.

The bloodstream and blood vessels

Lupus can cause cardiovascular and blood flow problems, anemia, or a decrease in red blood cell counts, and blood clotting or bleeding more frequently. Vasculitis, or inflammation of the blood vessels, is another effect it may have.

Lungs

Breathing difficulties may result from an inflammation of the lining of the chest cavity, which is more likely in people with lupus. Pneumonia and lung bleeding are also potential outcomes.

Heart

 Inflammation of the heart muscle (myocarditis), arteries (arteritis), or cardiac membranes (pericarditis) can all be brought on by lupus. There is also a significant rise in the risk of heart attacks and cardiovascular disease.

## Lupus Causes

Your immune system defends your body against infections and cancer when it is in good health. An autoimmune disease called lupus causes the immune system in the body to attack healthy tissue. Your genetic makeup and environmental factors most likely have a combined role in your development of lupus. But neither the aetiology nor the factors influencing the disease's variable manifestation are understood. Physicians are aware that a variety of elements are necessary, such as the "correct" genetic composition, exposure to the environment, and features unique to each organ. In addition, lupus patients may have trouble getting rid of ageing and damaged cells from their bodies, which constantly stimulates the immune system and causes an aberrant reaction.

In those with a genetic predisposition to the condition, exposure to environmental stimuli appears to be able to bring on a flare-up of the illness. It seems that exposure to environmental cues can trigger a flare-up of the sickness in persons who have a hereditary predisposition to the disorder.

Sunshine: In sensitive individuals, sun exposure may cause an internal reaction or cause lupus skin lesions.

Diseases: For some patients, an infection might start their lupus or lead to a relapse.

Remedy: Antibiotics, anti-seizure drugs, and several blood pressure meds can all cause lupus. When a drug-induced lupus patient stops taking the medicine, they typically recover.

Rarely, even when the medication is stopped, symptoms could still exist.

Symptoms and indicators of lupus

Non-lupus-specific symptoms are frequently seen by lupus sufferers.

Among them are fever, tiredness, blood clots, weight loss, and uneven or hairline-related hair loss. They might also have stomach pain, heartburn, and circulation problems in the fingers and toes. Miscarriages can happen to pregnant women. However, over 90% of people with the condition have cutaneous symptoms. The most common locations for skin lesions associated with systemic lupus erythematosus are listed below:

* Face, in particular the nose and cheeks
* Photosensitivity—an heightened sensitivity to sunlight—resulting in sun-exposed skin on the limbs, supports the palms, the upper portion of the chest, and upper back.
* The nails and fingers
* The nose or mouth

• The Scalp

One of the defining cutaneous characteristics of systemic lupus erythematosus is a butterfly rash, often known as malar blush. After being exposed to the sun, redness around the cheekbones and nose bridge may arise weeks before other symptoms do. A rash may appear on skin that has been photo-distributed, or has been subjected to the sun, particularly the backs of hands and fingers. The arms and trunk may also be affected by this rash, which takes the form of red, scaly areas. It is possible for tiny, painless ulcers to develop in the nose or mouth, usually on the upper part of the mouth. Hair loss is a possible symptom of lupus that affects the scalp skin. Patchiness or thinning throughout the scalp, particularly at the temples, are possible

Prevention of Lupus

Nobody is aware of a preventative measure for systemic lupus erythematosus (SLE) because its cause is unclear. By minimising sun exposure (wearing hats, long sleeves, and sunscreen), getting enough sleep, and taking prescribed medicine, flares of lupus may be lessened. The possibility of osteoporosis can be reduced by consuming vitamin D and calcium supplements.

The diagnosis of lupus

A single test cannot identify lupus. The diagnosis is made using the results of the physical examination, indications and symptoms, and blood and urine testing. strange blood tests, such as

* Low blood vessel counts, including anemia, low white blood cell counts, and low platelets.
* A positive result for antinuclear antibodies (ANA): Specific abnormal antibodies as well as like anti-double-strand DNA (also called anti-dsDNA), anti-Smith (sometimes called anti-Sm), or antiphospholipid immunoglobulins are present in almost all lupus patients and have the capacity to set off the body's own self-attack If, based on your symptoms, your doctor suspects you have lupus, they will order many tests for blood to confirm the diagnosis. The ANA blood screening test is the most significant. A negative ANA indicates the absence of lupus. If your ANA test results are positive, you may have lupus and require more testing. These blood tests include particular antibodies, including anti-dsDNA and anti-Sm, that are used to diagnose lupus. Antiphospholipid antibodies indicate an increased risk for specific problems, such as blood clots or miscarriage. Physicians may also assess blood levels of specific complement proteins, which are components of the immune system, in order to help identify and monitor the disease (Bogdanos et al., 2017).

## Lupus Treatment

The lupus is a long-term illness. Remission induction is the goal of treatment (Song etal., 2020). The kind and severity of your symptoms will determine how you are treated. Choosing which medications to take and whether to treat the signs and symptoms you are experiencing requires a careful discussion of the benefits and risks with your doctor. It’s possible that you and your doctor will need to adjust your medicine or dosage as your symptoms come on and off. The medications listed below are utilised most frequently to treat lupus:

Alternative names for nonsteroidal anti-inflammatory drugs (NSAIDs)

Over-the-counter NSAIDs such as naproxen sodium (Aleve) and ibuprofen (such as Motrin IB, and others) can be used to alleviate fever, edoema, and discomfort associated with lupus (Adhikary et al., 2021; Adhikary et al., 2022). Stronger NSAIDs can be obtained through prescription. NSAID adverse reactions include haemorrhaging in the stomach, kidney difficulties, and an increased risk of cardiac problems (Banerjee et al., 2022).

Prior to using any over-the-counter (OTC) medication for your lupus, always get your doctor's approval.

Immunosuppressive medicines

In severe lupus patients, immune-suppressive medications may be beneficial. Trexall, mycophenolate mofetil (CellCept), and azathioprine (Imuran, Azasan) are a few examples. The severe kidney ailment linked to the disease known as lupus nephritis has recently been treated with mycophenolate mofetil. A greater chance of infection spreading, liver damage, infertility, and cancer are examples of potential side effects.

Combination therapy

To manage lupus and avoid tissue damage, medical professionals may mix and match a few different drugs. Every therapy has advantages and disadvantages. The majority of immune-suppressive drugs have potential negative effects and need to be closely watched. Some side effects of these drugs include an increased risk of infections, diarrhea, nausea, and vomiting, loss of hair, hypertension, and osteoporosis (weak bones). When a medication has negative effects or the disease enters a remission, rheumatologists may decide to discontinue it altogether or reduce the dosage. Because of this, it's critical to have thorough and regular physical examinations as well as laboratory testing to monitor your symptoms and adjust your care as necessary.

Steroids

Other corticosteroids, such as prednisone, aid in lowering inflammation brought on by lupus. High doses of steroids are commonly used to treat severe brain and kidney illnesses, such as methylprednisolone (A-Methapred, Medrol) (Chowdhury et al., 2022). Among the negative effects are weight gain, bruising easily, diabetes, high blood pressure, osteoporosis (thinning bones), and an increased risk of infection. Larger dosages and longer treatment regimens carry a larger risk of adverse effects (Gupta et al., 2023).

8. CONCLUSION

Symptoms of inflammation have been linked to autoimmune diseases, and autoantibody counts in patients may help with diagnosis and severity assessments of the condition, which may be important for treatment. Myasthenia gravis is the most well-understood autoimmune disease; studies on the disease have advanced our knowledge of the basic principles underlying neuromuscular transmission. Rheumatoid arthritis and several medications used to treat it may decrease the immune system, which raises the risk of infections. When treating arthritis with rheumatoid arthritis, methotrexate is frequently the first medication used. For dual treatment, biologic drugs like cancer necrosis factor inhibitors may be combined. The -DR3 haplotype and other genetic predispositions to systemic lupus are associated with risk factors such as sunlight exposure, UV radiation from HLA-B8, viral infection (particularly Epstein-Barr virus hormones), pollutants, and cigarette smoking. Over-the-counter NSAIDs, such as ibuprofen (Advil, Motrin IB, and others) and naproxen sodium (Aleve), may be used to treat fever, edoema, and pain associated with lupus. Extended treatment plans and larger dosages are associated with an increased risk of side effects.

**REFERENCES**

1. Scherlinger M., Mertz P, Arnaud L.Worldwide trends in all-cause mortality of auto-immune systemic diseases between 2001 and 2014. *Autoimmune. Rev.*2020;19(8) [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32234406)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Autoimmun.+Rev.&title=doi:+arj:+worldwide+trends+in+all-cause+mortality+of+auto-immune+systemic+diseases+between+2001+and+2014&author=M.+Scherlinger&author=P.+Mertz&author=L.+Arnaud&volume=19&issue=8&publication_year=2020&)]

2. Leo A., Leung P.S.C., Hirschfield G.M., Gershwin E.M. The pathogenesis of primary biliary cholangitis: a comprehensive review. *Semin. Liver Dis.*2020;40(1):34–48. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31537031)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Semin.+Liver+Dis.&title=The+pathogenesis+of+primary+biliary+cholangitis:+a+comprehensive+review&author=A.+Lleo&author=P.S.C.+Leung&author=G.M.+Hirschfield&author=E.M.+Gershwin&volume=40&issue=1&publication_year=2020&pages=34-48&pmid=31537031&)]

3. Evert J., Lawler E., Bogan H., Perl’s T. Morbidity profiles of centenarians: survivors, delayers, and escapers. *J GerontolABiolSci Med Sci.*2003;58(3):232–237. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/12634289)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J+Gerontol+A+Biol+Sci+Med+Sci&title=Morbidity+profiles+of+centenarians:+survivors,+delayers,+and+escapers&author=J.+Evert&author=E.+Lawler&author=H.+Bogan&author=T.+Perls&volume=58&issue=3&publication_year=2003&pages=232-237&pmid=12634289&)]

4. Watad A., Bragazzi N.L., Shoenfeld Y. Autoimmunity in the elderly: insights from basic science and clinics - a mini-review. *Gerontology.*2017;63(6):515–523. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/28768257)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Gerontology&title=Autoimmunity+in+the+elderly:+insights+from+basic+science+and+clinics+-+a+mini-review&author=A.+Watad&author=N.L.+Bragazzi&author=Y.+Shoenfeld&volume=63&issue=6&publication_year=2017&pages=515-523&pmid=28768257&)]

5. Tang D., Kang R.…Lotze M.T. PAMPs and DAMPs: signals that spur autophagy and immunity. *Immunol. Rev.*2012;249(1) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3662247/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/22889221)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Immunol.+Rev.&title=PAMPs+and+DAMPs:+signals+that+spur+autophagy+and+immunity&author=D.+Tang&author=R.+Kang&author=M.T.+Lotze&volume=249&issue=1&publication_year=2012&)]

6. Satoh T., Akira S. Toll-like receptor signalling and its inducible proteins. *Microbiol.Spectr.*2016;4(6) [[PubMed](https://pubmed.ncbi.nlm.nih.gov/28084212)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Microbiol.+Spectr.&title=Toll-like+receptor+signaling+and+its+inducible+proteins&author=T.+Satoh&author=S.+Akira&volume=4&issue=6&publication_year=2016&)]

7. Croft A.P., Campos J., Buckley C.D. Distinct fibroblast subsets drive inflammation and damage arthritis. *Nature.*2019;570(7760):246–251. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6690841/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31142839)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nature&title=Distinct+fibroblast+subsets+drive+inflammation+and+damage+arthritis&author=A.P.+Croft&author=J.+Campos&author=C.D.+Buckley&volume=570&issue=7760&publication_year=2019&pages=246-251&pmid=31142839&)]

8. Swinkels M., Zhang J.H., Clark S.J. C-reactive protein and pentraxin-3 binding of factor H-like protein 1 differs from complement factor H: implications for retinal inflammation. *Sci. Rep.*2018;8(1) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5786067/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/29374201)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Sci.+Rep.&title=C-reactive+protein+and+pentraxin-3+binding+of+factor+H-like+protein+1+differs+from+complement+factor+H:+implications+for+retinal+inflammation&author=M.+Swinkels&author=J.H.+Zhang&author=S.J.+Clark&volume=8&issue=1&publication_year=2018&)]

9. Wang S.S., Vajdic C.M., Smedby K.E. Associations of non-Hodgkin Lymphoma (NHL) risk with autoimmune conditions according to putative NHL loci. *Am. J. Epidemiol.*2015;181(6):406–421. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4402340/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/25713336)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Am.+J.+Epidemiol.&title=Associations+of+non-Hodgkin+Lymphoma+(NHL)+risk+with+autoimmune+conditions+according+to+putative+NHL+loci&author=S.S.+Wang&author=C.M.+Vajdic&author=K.E.+Smedby&volume=181&issue=6&publication_year=2015&pages=406-421&pmid=25713336&)]

10. Fallah M., Liu X.… K. H: autoimmune diseases associated with non-Hodkin lymphoma: a nationwide cohort study. *Ann. Oncol.*2014;25(10):2025–2030. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/25081899)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Ann.+Oncol.&title=K.+H:+autoimmune+diseases+associated+wih+non-Hodkin+lymphome:+a+nationwide+cohort+study&author=M.+Fallah&author=X.+Liu&volume=25&issue=10&publication_year=2014&pages=2025-2030&pmid=25081899&)]

11. Hainer S.J. TG. F: high-resolution chromatin profiling using CUT&RUN. *CurrProtocMol Biol.*2019;126(1) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6422702/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30688406)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Curr+Protoc+Mol+Biol&title=TG.+F:+high-resolution+chromatin+profiling+using+CUT&RUN&author=S.J.+Hainer&volume=126&issue=1&publication_year=2019&)]

12. Meers M.P., Bryson T.D., Henikoff J.G., Henikoff S. Improved CUT&RUN chromatin profiling tools. *Elife.*2019 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6598765/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31232687)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Elife&title=Improved+CUT&RUN+chromatin+profiling+tools&author=M.P.+Meers&author=T.D.+Bryson&author=J.G.+Henikoff&author=S.+Henikoff&publication_year=2019&)]

13. Simeonov D.R. A. M: CRISPR-based tools in immunity. *Annu. Rev. Immunol.*2019;37:571–597. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30698999)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Annu.+Rev.+Immunol.&title=A.+M:+CRISPR-based+tools+in+immunity&author=D.R.+Simeonov&volume=37&publication_year=2019&pages=571-597&pmid=30698999&)]

14. West E.E., Kolev M., Kemper C. Complement and the regulation of T cell responses. *Annu. Rev. Immunol.*2018;36:309–338. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7478175/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/29677470)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Annu.+Rev.+Immunol.&title=Complement+and+the+regulation+of+T+cell+responses&author=E.E.+West&author=M.+Kolev&author=C.+Kemper&volume=36&publication_year=2018&pages=309-338&pmid=29677470&)]

15. Lo M.W., Kemper C., Woodruff T.M. COVID-19: complement, coagulation, and collateral damage. *J. Immunol.*2020;205(6):1488–1495. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7484432/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32699160)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Immunol.&title=COVID-19:+complement,+coagulation,+and+collateral+damage&author=M.W.+Lo&author=C.+Kemper&author=T.M.+Woodruff&volume=205&issue=6&publication_year=2020&pages=1488-1495&pmid=32699160&)]

16. Conigliaro P., Triggianese P.…Chimenti M.S. Complement, infection, and autoimmunity. *Curr.Opin.Rheumatol.*2019;31(5):532–541. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31192812)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Curr.+Opin.+Rheumatol.&title=Complement,+infection,+and+autoimmunity&author=P.+Conigliaro&author=P.+Triggianese&author=M.S.+Chimenti&volume=31&issue=5&publication_year=2019&pages=532-541&pmid=31192812&)]

17. Ling M., Murali M. Analysis of the complement system in the clinical immunology laboratory. *Clin. Lab. Med.*2019;39(4) [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31668271)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Clin.+Lab.+Med.&title=Analysis+of+the+complement+system+in+the+clinical+immunology+laboratory&author=M.+Ling&author=M.+Murali&volume=39&issue=4&publication_year=2019&)]

18. Kumagai S., Togashi Y.… H. N: the PD-1 expression balance between effector and regulatory T cells predicts the clinical efficacy of PD-1 blockade therapies. *N*at. Immunol.2020;21(11) [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32868929)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nat.+Immunol.&title=H.+N:+the+PD-1+expression+balance+between+effector+and+regulatory+T+cells+predicts+the+clinical+efficacy+of+PD-1+blockade+therapies&author=S.+Kumagai&author=Y.+Togashi&volume=21&issue=11&publication_year=2020&)]

19. Schreiber K.H., Apelo Si Arriola.… DW. L, .NCJd: a novel rapamycinanalog is highly selective for mTORC1 in vivo. *Nat. Commun.*2019;10(1) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6642166/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31324799)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nat.+Commun.&title=DW.+L,+.+NCJd:+a+novel+rapamycin+analog+is+highly+selective+for+mTORC1+in+vivo&author=K.H.+Schreiber&author=Arriola+Apelo+Si&volume=10&issue=1&publication_year=2019&)]

20. Khan S., Gerber D.E. Autoimmunity, checkpoint inhibitor therapy and immune-related adverse events: a review. *Semin. Canc. Biol.*2020;64:93–101. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6980444/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31330185)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Semin.+Canc.+Biol.&title=Autoimmunity,+checkpoint+inhibitor+therapy+and+immune-related+adverse+events:+a+review&author=S.+Khan&author=D.E.+Gerber&volume=64&publication_year=2020&pages=93-101&)]

21. Fässler M., Diem S.…Flatz L. Antibodies as biomarker candidates for response and survival to checkpoint inhibitors in melanoma patients. *J Immunother Cancer.*2019;7(1) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6383238/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30786924)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J+Immunother+Cancer&title=Antibodies+as+biomarker+candidates+for+response+and+survival+to+checkpoint+inhibitors+in+melanoma+patients&author=M.+F%C3%A4ssler&author=S.+Diem&author=L.+Flatz&volume=7&issue=1&publication_year=2019&)]

22. Finkel P., Xia W., Jefferies W.A. Beyond unconventional: what do we really know about group 2 innate lymphoid cells? *J. Immunol.*2021;206(7):1409–1417. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33753565)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Immunol.&title=Beyond+unconventional:+what+do+we+really+know+about+group+2+innate+lymphoid+cells?&author=P.+Finkel&author=W.+Xia&author=W.A.+Jefferies&volume=206&issue=7&publication_year=2021&pages=1409-1417&pmid=33753565&)]

23. Zhou L., Huntington K.…El-Deiry W.S. Natural Killer cell activation, reduced ACE2, TMPRSS2, cytokines G-CSF, M-CSF and SARS-CoV-2-S pseudovirus infectivity by MEK inhibitor treatment of human cells. *bioRxiv.*2020, aug 3 [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=bioRxiv&title=Natural+Killer+cell+activation,+reduced+ACE2,+TMPRSS2,+cytokines+G-CSF,+M-CSF+and+SARS-CoV-2-S+pseudovirus+infectivity+by+MEK+inhibitor+treatment+of+human+cells&author=L.+Zhou&author=K.+Huntington&author=W.S.+El-Deiry&publication_year=2020,+aug+3&)]

24. Rendeiro A.F., Casano J.…Inghirami G. Profiling of immune dysfunction in COVID-19 patients allows early prediction of disease progression. *Life Science Alliance.*2020 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7768198/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33361110)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Life+Science+Alliance&title=Profiling+of+immune+dysfunction+in+COVID-19+patients+allows+early+prediction+of+disease+progression&author=A.F.+Rendeiro&author=J.+Casano&author=G.+Inghirami&publication_year=2020&)]

25. Hajishengalis G., Xiaofei L.…Chavakis T. Trained innate immunity and its implications for mucosal immunityand inflammation. *Adv. Exp. Med. Biol.*2019;1197:11–26. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6986364/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31732931)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Adv.+Exp.+Med.+Biol.&title=Trained+innate+immunity+and+its+implications+for+mucosal+immunityand+inflammation&author=G.+Hajishengalis&author=L.+Xiaofei&author=T.+Chavakis&volume=1197&publication_year=2019&pages=11-26&pmid=31732931&)]

26. Chua B.A., Van Der Werf I., Jamieson C., Signer R.A. Post-transcriptional regulation of homeostatic, stressed, and malignant stem cells. *Cell Stem Cell.*2020;26(2) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7158223/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32032524)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Cell+Stem+Cell&title=Post-transcriptional+regulation+of+homeostatic,+stressed,+and+malignant+stem+cells&author=B.A.+Chua&author=I.+Van+Der+Werf&author=C.+Jamieson&author=R.A.+Signer&volume=26&issue=2&publication_year=2020&)]

27. Schulte-Schrepping J., Reusch N.… (DeCOI). SLDC-OI: severe COVID-19 is marked by a dysregulated myeloid cell compartment. *Cell.*2020;182(6):1419–1440. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7405822/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32810438)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Cell&title=(DeCOI).+SLDC-OI:+severe+COVID-19+is+marked+by+a+dysregulated+myeloid+cell+compartment&author=J.+Schulte-Schrepping&author=N.+Reusch&volume=182&issue=6&publication_year=2020&pages=1419-1440&pmid=32810438&)]

28. van der Made C.I., Hoischen A.…Ikeno Y. Primary immunodeficiencies in cytosolic pattern-recognition receptor pathways: toward host-directed treatment strategies. *Immunol. Rev.*2020;297(1):247–272. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32640080)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Immunol.+Rev.&title=Primary+immunodeficiencies+in+cytosolic+pattern-recognition+receptor+pathways:+toward+host-directed+treatment+strategies&author=C.I.+van+der+Made&author=A.+Hoischen&author=Y.+Ikeno&volume=297&issue=1&publication_year=2020&pages=247-272&pmid=32640080&)]

29. Combes A.J., Courau T.…Krummel M.F. Global absence and targeting of protective immune states in severe COVID-19. *Nature.*2021;591:124–130. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8567458/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33494096)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nature&title=Global+absence+and+targeting+of+protective+immune+states+in+severe+COVID-19&author=A.J.+Combes&author=T.+Courau&author=M.F.+Krummel&volume=591&publication_year=2021&pages=124-130&pmid=33494096&)]

30. Shin J.I., Lee K.H.… Kronbichler A**: inflammasomes and autoimmune and rheumatic diseases: a comprehensive review.** *J Autoimmmun.*2019;103 [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31326231)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J+Autoimmmun&title=Kronbichler+A:+inflammasomes+and+autoimmune+and+rheumatic+diseases:+a+comprehensive+review&author=J.I.+Shin&author=K.H.+Lee&volume=103&publication_year=2019&)]

31. Defendi F., Thielens N.M.…Dumestre-Pérard C. The immunopathology of complement proteins and innate immunity in autoimmune disease. *Clin. Rev. Allergy Immunol.*2020;58:229–251. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31834594)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Clin.+Rev.+Allergy+Immunol.&title=The+immunopathology+of+complement+proteins+and+innate+immunity+in+autoimmune+disease&author=F.+Defendi&author=N.M.+Thielens&author=C.+Dumestre-P%C3%A9rard&volume=58&publication_year=2020&pages=229-251&pmid=31834594&)]

32. Bekkering S., Arts R.J.W.…Netea M.G. Metabolic induction of trained immunity through the mevalonate pathway. *Cell.*2018;(1–2):135–146. J.W. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/29328908)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Cell&title=Metabolic+induction+of+trained+immunity+through+the+mevalonate+pathway&author=S.+Bekkering&author=R.J.W.+Arts&author=M.G.+Netea&issue=1%E2%80%932&publication_year=2018&pages=135-146&pmid=29328908&)]

33. van der Meer J.W., Simon A. doi: ro: the challenge of autoinflammatory syndromes: with an emphasis on hyper-IgD syndrome. *Rheumatology.*2016:ii23–ii29. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/27856657)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Rheumatology&title=doi:+ro:+the+challenge+of+autoinflammatory+syndromes:+with+an+emphasis+on+hyper-IgD+syndrome&author=J.W.+van+der+Meer&author=A.+Simon&publication_year=2016&pages=ii23-ii29&pmid=27856657&)]

34. Wrap D., De Vlieger D., McLellan J.S. Structural basis for potent neutralization of beta coronaviruses by single-domain camelid antibodies. *Cell.*2020;181(5) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7199733/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32375025)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Cell&title=Structural+basis+for+potent+neutralization+of+betacoronaviruses+by+single-domain+camelid+antibodies&author=D.+Wrapp&author=D.+De+Vlieger&author=J.S.+McLellan&volume=181&issue=5&publication_year=2020&)]

35. Hoey R.J., Eom H., Horn J.R. Structure and development of single domain antibodies as modules for therapeutics and diagnostics. *Exp. Biol. Med.*2019;244(17):1568–1576. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6920669/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31594404)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Exp.+Biol.+Med.&title=Structure+and+development+of+single+domain+antibodies+as+modules+for+therapeutics+and+diagnostics&author=R.J.+Hoey&author=H.+Eom&author=J.R.+Horn&volume=244&issue=17&publication_year=2019&pages=1568-1576&)]

36. EASL European Association for the Study of the Liver. Clinical Practice Guidelines: the diagnosis and management of patients with primary biliary cholangitis. *Hepatol.*2017;67(1):145–172. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/28427765)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=JHepatol&title=European+Association+for+the+Study+of+the+Liver.+Clinical+Practice+Guidelines:+the+diagnosis+and+management+of+patients+with+primary+biliary+cholangitis&volume=67&issue=1&publication_year=2017&pages=145-172&pmid=28427765&)]

37. Lung T., Sakem B.…Nydegger U. The complement system in liver diseases: evidence-based approach and therapeutic options. *J Trans Autoimmun.*2019 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7388403/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32743505)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J+Transl+Autoimmun&title=The+complement+system+in+liver+diseases:+evidence-based+approach+and+therapeutic+options&author=T.+Lung&author=B.+Sakem&author=U.+Nydegger&publication_year=2019&)]

38. Nydegger U., Lung T. T. B: inflammation thread runs across medical laboratory specialities. *Mediat.Inflamm.*2016;2016:4121837. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4963559/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/27493451)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Mediat.+Inflamm.&title=T.+B:+inflammation+thread+runs+across+medical+laboratory+specialities&author=U.+Nydegger&author=T.+Lung&volume=2016&publication_year=2016&pages=4121837&)]

39. Song S., De S., Barners B. Inhibition of IRF5 hyperactivation protects from lupus onset and severity. *J. Clin. Invest.*2020;130(12):6700–6717. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7685739/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32897883)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Clin.+Invest.&title=Inhibition+of+IRF5+hyperactivation+protects+from+lupus+onset+and+severity&author=S.+Song&author=S.+De&author=B.+Barners&volume=130&issue=12&publication_year=2020&pages=6700-6717&pmid=32897883&)]

40. Bettacchioly E, Legaffric C, ., Renaudineau Y: An elevated polyclonal free light chain level reflects a strong interferon signature in patients with systemic autoimmune diseases. J Trans Autoimmun 2021, 4(100090). [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8010703/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33817614)].

41. Annett S., Moore G., Robson T. FK506 binding proteins and inflammation related signalling pathways; basic biology, current status and future prospects for pharmacological intervention. *Pharmacol.Ther.*2020 [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32622856)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Pharmacol.+Ther.&title=FK506+binding+proteins+and+inflammation+related+signalling+pathways;+basic+biology,+current+status+and+future+prospects+for+pharmacological+intervention&author=S.+Annett&author=G.+Moore&author=T.+Robson&publication_year=2020&)]

42. Stojan G., Petri M. Anti-C1q in systemic lupus erythematosus. *Lupus.*2016;25(8):873–877. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7523495/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/27252264)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Lupus&title=Anti-C1q+in+systemic+lupus+erythematosus&author=G.+Stojan&author=M.+Petri&volume=25&issue=8&publication_year=2016&pages=873-877&pmid=27252264&)]

43. Bogdanos D.P., Sakkas L.I. From microbiome to infectome in autoimmunity. *Curr.Opin.Rheumatol.*2017;29(4):369–373. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/28394824)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Curr.+Opin.+Rheumatol.&title=From+microbiome+to+infectome+in+autoimmunity&author=D.P.+Bogdanos&author=L.I.+Sakkas&volume=29&issue=4&publication_year=2017&pages=369-373&pmid=28394824&)]

44. Nydegger U.E., Lambert P.H., Gerber H., Miescher P.A. Circulating immune complexes in the serum in systemic lupus erythematosus and in carriers of hepatitis B antigen. Quantitation by binding to radiolabelled C1q. *J. Clin. Invest.*1974;54:297–309. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC301557/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/4847246)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Clin.+Invest.&title=Circulating+immune+complexes+in+the+serum+in+systemic+lupus+erythematosus+and+in+carriers+of+hepatitis+B+antigen.+Quantitation+by+binding+to+radiolabeled+C1q&author=U.E.+Nydegger&author=P.H.+Lambert&author=H.+Gerber&author=P.A.+Miescher&volume=54&publication_year=1974&pages=297-309&pmid=4847246&)]

45. Arulraj T., Binder S., Meyer-Hermann M. Rate of immune complex cycling in follicular dendritic cells determines the extent of protecting antigen integrity and availability to germinal centre B cells. *J. Immunol.*2021;206(7):1436–1442. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7980531/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33608455)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Immunol.&title=Rate+of+immune+complex+cyclingg+in+follicular+dendritic+cells+determines+the+extent+of+protecting+antigen+integrity+and+availability+to+germinal+center+B+cels&author=T.+Arulraj&author=S.+Binder&author=M.+Meyer-Hermann&volume=206&issue=7&publication_year=2021&pages=1436-1442&pmid=33608455&)]

46. Abu-Rumeileh S., Abdelhak A.…Otto M. Guillain-Barré syndrome spectrum associated with COVID-19: an up-to-date systematic review of 73 cases. *J. Neurol.*2020:1–30. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7445716/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32840686)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Neurol.&title=Guillain-Barr%C3%A9+syndrome+spectrum+associated+with+COVID-19:+an+up-to-date+systematic+review+of+73+cases&author=S.+Abu-Rumeileh&author=A.+Abdelhak&author=M.+Otto&publication_year=2020&pages=1-30&)]

47. Soltani E.Z., Rahmani F., Rezaei N. Autoimmunity and cytokines in Guillain-Barre syndrome revisited: review of pathomechanisms with an eye on therapeutic options. *Eur. Cytokine Newt.*2019;30(1):1–14. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31074417)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Eur.+Cytokine+Netw.&title=Autoimmunity+and+cytokines+in+Guillain-Barre+syndrome+revisited:+review+of+pathomechanisms+with+an+eye+on+therapeutic+options&author=E.Z.+Soltani&author=F.+Rahmani&author=N.+Rezaei&volume=30&issue=1&publication_year=2019&pages=1-14&pmid=31074417&)]

48. Levine M.E., Lu A.T., Horvath S. doi: a-: an epigenetic biomarker of aging for lifespan and health span. *Aging (Albany NY)*2018;10(4):573–591. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5940111/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/29676998)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Aging+(Albany+NY)&title=doi:+a-:+an+epigenetic+biomarker+of+aging+for+lifespan+and+healthspan&author=M.E.+Levine&author=A.T.+Lu&author=S.+Horvath&volume=10&issue=4&publication_year=2018&pages=573-591&pmid=29676998&)]

49. Lu A.T., Quach A., Wilson J.G., Reiner A.P., Aviv A., Raj K., Hou L., Baccarelli A.A., Li Y., Stewart J.D. DNA methylation GrimAge strongly predicts lifespan and health span. *Aging (Albany NY)*2019;11(2):303–327. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6366976/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30669119)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Aging+(Albany+NY)&title=DNA+methylation+GrimAge+strongly+predicts+lifespan+and+healthspan&author=A.T.+Lu&author=A.+Quach&author=J.G.+Wilson&author=A.P.+Reiner&author=A.+Aviv&volume=11&issue=2&publication_year=2019&pages=303-327&pmid=30669119&)]

50. Zhang Y., Wilson R.… H. B: DNA methylation signatures in peripheral blood strongly predict all-cause mortality. *Nat. Commun.*2017;17(8) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5357865/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/28303888)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nat.+Commun.&title=H.+B:+DNA+methylation+signatures+in+peripheral+blood+strongly+predict+all-cause+mortality&author=Y.+Zhang&author=R.+Wilson&volume=17&issue=8&publication_year=2017&)]

51. Alpert A., YishaiPickman Y., Shen-Orr S.S. A clinically meaningful metric of immune age derived from high-dimensional longitudinal monitoring. *Nat. Med.*2019;25(3):487–495. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6686855/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30842675)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nat.+Med.&title=A+clinically+meaningful+metric+of+immune+age+derived+from+high-dimensional+longitudinal+monitoring&author=A.+Alpert&author=Y.+Yishai+Pickman&author=S.S.+Shen-Orr&volume=25&issue=3&publication_year=2019&pages=487-495&pmid=30842675&)]

52. Mamoshina P., Zhavoronkov A. edn. vol. 10.Moskalev A; 2019. Deep integrated biomarkers of aging. (Biomarkers of Human Aging, Healthy Aging and Longevity). [[Google Scholar](https://scholar.google.com/scholar?q=Mamoshina+P.+Zhavoronkov+A.+Deep+integrated+biomarkers+of+aging+edn.+Biomarkers+of+Human+Aging,+Healthy+Aging+and+Longevity+vol.+10+2019+Moskalev+A+)]

53. Rockwood K., Mitnitski A. Frailty defined by deficit accumulation and geriatric medicine defined by frailty. *Clin.Geriatr. Med.*2011;27(1):17–26. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/21093719)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Clin.+Geriatr.+Med.&title=Frailty+defined+by+deficit+accumulation+and+geriatric+medicine+defined+by+frailty&author=K.+Rockwood&author=A.+Mitnitski&volume=27&issue=1&publication_year=2011&pages=17-26&pmid=21093719&)]

54. Lehallier B., Gate D., Schaum N., Nanasi T., Lee S.E., Yousef H., Moran Losada P., Berdnik D., Keller A., Verghese J. Undulating changes in human plasma proteome profiles across the lifespan. *Nat. Med.*2019;25(12):1843–1850. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7062043/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31806903)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nat.+Med.&title=Undulating+changes+in+human+plasma+proteome+profiles+across+the+lifespan&author=B.+Lehallier&author=D.+Gate&author=N.+Schaum&author=T.+Nanasi&author=S.E.+Lee&volume=25&issue=12&publication_year=2019&pages=1843-1850&pmid=31806903&)]

55. Vučković F., Krištić J.… G. L: association of systemic lupus erythematosus with decreased immunosuppressive potential of the IgGglycome. *Arthritis Rheum.*2015;67(11):2978–2989. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4626261/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/26200652)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Arthritis+Rheum.&title=G.+L:+association+of+systemic+lupus+erythematosus+with+decreased+immunosuppressive+potential+of+the+IgG+glycome&author=F.+Vu%C4%8Dkovi%C4%87&author=J.+Kri%C5%A1ti%C4%87&volume=67&issue=11&publication_year=2015&pages=2978-2989&)]

56. Mueller A.L., McNamara M.S., Sinclair D.A. Why does COVID-19 disproportionately affect older people? *Aging (Albany NY)*2020;12(10):9959–9981. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7288963/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32470948)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Aging+(Albany+NY)&title=Why+does+COVID-19+disproportionately+affect+older+people?&author=A.L.+Mueller&author=M.S.+McNamara&author=D.A.+Sinclair&volume=12&issue=10&publication_year=2020&pages=9959-9981&pmid=32470948&)]

57. Kohli J., Veenstra I., Demaria M. The struggle of a good friend getting old: cellular senescence in viral responses and therapy. *EMBO Rep.*2021 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8024996/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33734564)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=EMBO+Rep.&title=The+struggle+of+a+good+friend+getting+old:cellular+senescence+in+viral+responses+and+therapy&author=J.+Kohli&author=I.+Veenstra&author=M.+Demaria&publication_year=2021&)]

58. Challenor S., Tucker D. SARS-CoV-2-induced remission of Hodgkin lymphoma. *Br. J. Haematol.*2021;(3):192. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33386647)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Br.+J.+Haematol.&title=SARS-CoV-2-induced+remission+of+Hodgkin+lymphoma&author=S.+Challenor&author=D.+Tucker&issue=3&publication_year=2021&pages=192&)]

59. VkPh, Annika Kratzel A., Thiel V. Coronavirus biology and replication: implications for SARS-CoV-2. *Nat. Rev. Microbiol.*2021;19(3):155–170. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7592455/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33116300)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nat.+Rev.+Microbiol.&title=Coronavirus+biology+and+replication:+implications+for+SARS-CoV-2&author=Ph+Vk&author=A.+Annika+Kratzel&author=V.+Thiel&volume=19&issue=3&publication_year=2021&pages=155-170&pmid=33116300&)]

60. Henriksson J., Chen X., Teichmann S.A. Genome-wide CRISPR screens in T helper cells reveal pervasive crosstalk between activation and differentiation. *Cell.*2019;176(4):882–896. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6370901/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30639098)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Cell&title=Genome-wide+CRISPR+screens+in+T+helper+cells+reveal+pervasive+crosstalk+between+activation+and+differentiation&author=J.+Henriksson&author=X.+Chen&author=S.A.+Teichmann&volume=176&issue=4&publication_year=2019&pages=882-896&pmid=30639098&)]

61. Moreno AM, Aleman F, .., Mali P: Long-lasting analgesia via targeted in situ repression of Nav 1.7 in mice. Sci. Transl. Med. 2021, 13(584). [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8830379/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33692134)]

62. Liu Y., Sawalha A.H., Lu Q. COVID-19 and autoimmune diseases. *Curr.Opin.Rheumatol.*2021;33(2):155–162. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7880581/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33332890)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Curr.+Opin.+Rheumatol.&title=COVID-19+and+autoimmune+diseases&author=Y.+Liu&author=A.H.+Sawalha&author=Q.+Lu&volume=33&issue=2&publication_year=2021&pages=155-162&pmid=33332890&)]

63. Lung T., Kazatchkine M., Risch L., Risch M., Nydegger U. A consideration of convalescent plasma and plasma derivatives in the care of severely- ill patients with COVID 19. *Transfuse. Apher. Sci.*2020;59(5) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7833822/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32919880)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Transfus.+Apher.+Sci.&title=A+consideration+of+convalescent+plasma+and+plasmaderivatives+in+the+care+of+severely-+ill+patients+with+COVID+19&author=T.+Lung&author=M.+Kazatchkine&author=L.+Risch&author=M.+Risch&author=U.+Nydegger&volume=59&issue=5&publication_year=2020&)]

64. McMillan P., Dexhiemer T.…Uhal B.D. COVID-19 - a theory of autoimmunity against ACE-2 explained. *Front. Immunol.*2021 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8021777/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33833750)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Front.+Immunol.&title=COVID-19+-+a+theory+of+autoimmunity+against+ACE-2+explained&author=P.+McMillan&author=T.+Dexhiemer&author=B.D.+Uhal&publication_year=2021&)]

65. Patriquin C., Kuo K.H.M. Eculizumab and beyond: the past, present, and future of complement therapeutics. *Transfuse. Med. Rev.*2019;33(4):254–265. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31703946)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Transfus.+Med.+Rev.&title=Eculizumab+and+beyond:+the+past,+present,+and+future+of+complement+therapeutics&author=C.+Patriquin&author=K.H.M.+Kuo&volume=33&issue=4&publication_year=2019&pages=254-265&)]

66. Hillmen P., Szer J., de la Tour P. Pegcetacoplan versus eculizumab in paroxysmal nocturnal haemoglobinuria. *N. Engl. J. Med.*2021;384:1028–1037. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33730455)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=N.+Engl.+J.+Med.&title=Pegcetacoplan+versus+eculizumab+in+paroxysmal+nocturnal+hemoglobinuria&author=P.+Hillmen&author=J.+Szer&author=P.+de+la+Tour&volume=384&publication_year=2021&pages=1028-1037&pmid=33730455&)]

67. Jayne D.R.W., Merkel P.A.…Bekker M.D. Avoca pan for the treatment of ANCA-associated vasculitis. *N. Engl. J. Med.*2021;384(feb 18):599–609. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33596356)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=N.+Engl.+J.+Med.&title=Avocapan+for+the+treatment+of+ANCA-associated+vasculitis&author=D.R.W.+Jayne&author=P.A.+Merkel&author=M.D.+Bekker&volume=384&issue=feb+18&publication_year=2021&pages=599-609&pmid=33596356&)]

68. Roth A., Barcellini W.…Berentsen S. Sutimlimab in cold agglutinin disease. *N. Engl. J. Med.*2021;384:1323–1334. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33826820)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=N.+Engl.+J.+Med.&title=Sutimlimab+in+cold+agglutinin+disease&author=A.+R%C3%B6th&author=W.+Barcellini&author=S.+Berentsen&volume=384&publication_year=2021&pages=1323-1334&pmid=33826820&)]

69. Berentsen S. How I treat cold agglutinin disease. *Blood.*2021;137(10):1295–1303. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33512410)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Blood&title=How+I+treat+cold+agglutinin+disease&author=S.+Berentsen&volume=137&issue=10&publication_year=2021&pages=1295-1303&pmid=33512410&)]

70. Zelek W., Xie L., Morgan B.P., Harris C.L. Compendium of curreent complement therapeutics. *Ml Immunol.*2019;114:341–352. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31446305)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Ml+Immunol&title=Compendium+of+curreent+complement+therapeutics&author=W.+Zelek&author=L.+Xie&author=B.P.+Morgan&author=C.L.+Harris&volume=114&publication_year=2019&pages=341-352&)]

71. Valenti L., Griffini S., Cugno M. Chromosome 3 cluster rs11385942 variant links complement activation with severe COVID-19. *J. Autoimmun.*2021 al e. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7796659/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33453462)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Autoimmun.&title=Chromosome+3+cluster+rs11385942+variant+links+complement+activation+with+severe+COVID-19&author=L.+Valenti&author=S.+Griffini&author=M.+Cugno&publication_year=2021&)]

72. Prochaska Z.…Frazer-Abel A. Complement analysis in the era of targeted therapeutics. *Mol. Immunol.*2018;102:84–88. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/29933889)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Mol.+Immunol.&title=Complement+analysis+in+the+era+of+targeted+therapeutics&author=Z.+Prohaszka&author=A.+Frazer-Abel&volume=102&publication_year=2018&pages=84-88&pmid=29933889&)]

73. Flemming A. mRNA vaccine shows promise in autoimmunity. *YNat Rev Immunol.*2021, Jan 12 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7802056/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33437044)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=YNat+Rev+Immunol&title=mRNA+vaccine+shows+promise+in+autoimmunity&author=A.+Flemming&publication_year=2021,+Jan+12&)]

74. Krienke C., Kolb L.… U. S: a noninflammatory mRNA vaccine for treatment of experimental autoimmune encephalomyelitis. *Science.*2021, Jan 8;(371):145–153. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33414215)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Science&title=U.+S:+a+noninflammatory+mRNA+vaccine+for+treatment+of+experimental+autoimmune+encephalomyelitis&author=C.+Krienke&author=L.+Kolb&issue=371&publication_year=2021,+Jan+8&pages=145-153&pmid=33414215&)]

75. Caso F., Costa L.…Scarpa R. Could SARS-CV-2 trigger autoimmune and/or autoinflammatory mechanisms in genetically predisposed subjects? *Autoimmun. Rev.*2020 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7271072/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/32220633)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Autoimmun.+Rev.&title=Could+SARS-CV-2+trigger+autoimmune+and/or+autoinflammatory+mechanisms+in+genetically+predisposed+subjects?&author=F.+Caso&author=L.+Costa&author=R.+Scarpa&publication_year=2020&)]

76. Robinson E.K., Covarrubias S., Carpenter S. The how and why of lncRNA function: an innate immune perspective. *Biochim Biopsy’s Acta Gene Regul Mech.*2020;(4):1863. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7185634/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31487549)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Biochim+Biophys+Acta+Gene+Regul+Mech&title=The+how+and+why+of+lncRNA+function:an+innate+immune+perspective&author=E.K.+Robinson&author=S.+Covarrubias&author=S.+Carpenter&issue=4&publication_year=2020&pages=1863&)]

77. Cohen Tervaert J.W. Autoinflammatory/autoimmunity syndrome induced by adjuvants (ASIA; Shoenfeld's syndrome): a new flame. *Autoimmun. Rev.*2018;17(12):1259–1264. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30316995)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Autoimmun.+Rev.&title=Autoinflammatory/autoimmunity+syndrome+induced+by+adjuvants+(ASIA;+Shoenfeld%27s+syndrome):+a+new+flame&author=J.W.+Cohen+Tervaert&volume=17&issue=12&publication_year=2018&pages=1259-1264&pmid=30316995&)]

78. Imbach P., Barandun S.…Wagner H.P. High-dose intravenous gammaglobulin for idiopathic thrombocytopenic purpura in childhood. *Lancet.*1981;i:1228–1231. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/6112565)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Lancet&title=High-dose+intravenous+gammaglobulin+for+idiopathic+thrombocytopenic+purpra+in+childhood&author=P.+Imbach&author=S.+Barandun&author=H.P.+Wagner&volume=i&publication_year=1981&pages=1228-1231&)]

79. Sultan Y., Kazatchkine M.D., Maisonneuve P., Nydegger U.E. Anti-idiotypic suppression of autoantibodies to factor VIII (antihemophilic factor) by high-dose intravenous gammaglobulin. *Lancet.*1984;8406:765–768. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/6148519)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Lancet&title=Anti-idiotypic+suppression+of+autoantibodies+to+factor+VIII+(antihaemophilic+factor)+by+high-dose+intravenous+gammaglobulin&author=Y.+Sultan&author=M.D.+Kazatchkine&author=P.+Maisonneuve&author=U.E.+Nydegger&volume=8406&publication_year=1984&pages=765-768&)]

80. Tian M., Cheng H.L.…Alt F.W. An in vivo method for diversifying the functions of therapeutic antibodies. *Proc. Natl. Acad. Sci. Unit. States Am.*2021;118(10) [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7958431/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33658386)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Proc.+Natl.+Acad.+Sci.+Unit.+States+Am.&title=An+in+vivo+method+for+diversifying+the+functions+of+therapeutic+antibodies&author=M.+Tian&author=H.L.+Cheng&author=F.W.+Alt&volume=118&issue=10&publication_year=2021&)]

81. Signore A., Erba P.A. Molecular imaging of inflammation/infection: the future of disease management. *Curr.Pharmaceut.Des.*2018;24(7):741–742. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/29773052)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Curr.+Pharmaceut.+Des.&title=Molecular+imaging+of+inflammation/infection:+the+future+of+disease+management&author=A.+Signore&author=P.A.+Erba&volume=24&issue=7&publication_year=2018&pages=741-742&)]

82. Ding X., Zhang B. DeepBAR: a fast and exact method for binding free energy computation. *J. Phys. Chem. Lett.*2021;12:2509–2515. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8030779/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33719449)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=J.+Phys.+Chem.+Lett.&title=DeepBAR:+a+fast+and+exact+method+for+binding+free+energy+computation&author=X.+Ding&author=B.+Zhang&volume=12&publication_year=2021&pages=2509-2515&pmid=33719449&)]

83. Vulto A.G., Jaquez O.A. The process defines the product: what really matters in biosimilar design and production? *Rheumatology.*2017;56:iv14–iv29. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5850795/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/28903544)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Rheumatology&title=The+process+defines+the+product:+what+really+matters+in+biosimilar+design+and+production?&author=A.G.+Vulto&author=O.A.+Jaquez&volume=56&publication_year=2017&pages=iv14-iv29&pmid=28903544&)]

84. Rajewsky K. The advent and rise of monoclonal antibodies. *Nature.*2019:47–49. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31686050)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Nature&title=The+advent+and+rise+of+monoclonal+antibodies&author=K.+Rajewsky&publication_year=2019&pages=47-49&)]

85. Weinreich D.M., Sivapalasingam S., Norton T. Investigators T: REGN-COV2, a neutralizing antibody cocktail. *N. Engl. J. Med.*2020;17dec [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7781102/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/33332778)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=N.+Engl.+J.+Med.&title=Investigators+T:+REGN-COV2,+a+neutralizing+antibody+cocktail&author=D.M.+Weinreich&author=S.+Sivapalasingam&author=T.+Norton&volume=17+dec&publication_year=2020&)]

86. Kohler H., AnastasPashov A., Kieber-Emmons T. The promise of anti-idiotype revisited. *Front. Immunol.*2019 [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6474207/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31031777)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Front.+Immunol.&title=The+promise+of+anti-idiotype+revisited&author=H.+Kohler&author=A.+Anastas+Pashov&author=T.+Kieber-Emmons&publication_year=2019&)]

87. Adamson H., Nicholl A.T.… DC. T: affimers as anti-idiotypic affinity reagents for pharmacokinetic analysis of biotherapeutics. *Biotechniques.*2019:261–269. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31823668)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Biotechniques&title=DC.+T:+affimers+as+anti-idiotypic+affinity+reagents+for+pharmacokinetic+analysis+of+biotherapeutics&author=H.+Adamson&author=A.T.+Nicholl&publication_year=2019&pages=261-269&)]

88. Terwiel M., Grutters J.C., van Morsel C.H.M. Clustering of immune-mediated diseases in sarcoidosis. *Curr.Opin.Plum. Med.*2019;25(5):539–553. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31365389)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Curr.+Opin.+Pulm.+Med.&title=Clustering+of+immune-mediated+diseases+in+sarcoidosis&author=M.+Terwiel&author=J.C.+Grutters&author=C.H.M.+van+Moorsel&volume=25&issue=5&publication_year=2019&pages=539-553&pmid=31365389&)]

89. Llanos O., Hamzeh N. Sarcoidosis. *Med. Clin.*2019;103(3):523–534. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30955519)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Med.+Clin.&title=Sarcoidosis&author=O.+Llanos&author=N.+Hamzeh&volume=103&issue=3&publication_year=2019&pages=523-534&)]

90. Starshinova A., Zinchenko Y., Yablonskiy P. Specific features of immune complexes in patients with sarcoidosis and pulmonary tuberculosis. *Immunol. Res.*2018;66(6):737–743. [[PubMed](https://pubmed.ncbi.nlm.nih.gov/30552618)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Immunol.+Res.&title=Specific+features+of+immune+complexes+in+patients+with+sarcoidosis+and+pulmonary+tuberculosis&author=A.+Starshinova&author=Y.+Zinchenko&author=P.+Yablonskiy&volume=66&issue=6&publication_year=2018&pages=737-743&pmid=30552618&)].

91. Starshinova A.A., Malkova A.M.…Yablonskiy P.K. Sarcoidosis as an autoimmune disease. *Front. Immunol.*2020:10. [[PMC free article](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6960207/)] [[PubMed](https://pubmed.ncbi.nlm.nih.gov/31969879)] [[Google Scholar](https://scholar.google.com/scholar_lookup?journal=Front.+Immunol.&title=Sarcoidosis+as+an+autoimmune+disease&author=A.A.+Starshinova&author=A.M.+Malkova&author=P.K.+Yablonskiy&publication_year=2020&pages=10&pmid=32117219&)].

92. Bhattacharya, K., Dey, R., Sen, D., Paul, N., Basak, A. K., Purkait, M. P., Shukla, N., Chaudhuri, G. R., Bhattacharya, A., Maiti, R., Adhikary, K., Chatterjee, P., Karak, P., & Syamal, A. K. (2024). Polycystic ovary syndrome and its management: In view of oxidative stress. *Biomolecular concepts*, *15*(1), 10.1515/bmc-2022-0038. <https://doi.org/10.1515/bmc-2022-0038>

93. Adhikary, K., Banerjee, P., Barman, S., Bandyopadhyay, B., & Bagchi, D. (2024). Nutritional Aspects, Chemistry Profile, Extraction Techniques of Lemongrass Essential Oil and It's Physiological Benefits. *Journal of the American Nutrition Association*, *43*(2), 183–200. <https://doi.org/10.1080/27697061.2023.2245435>

94. Adhikary, K., Mohanty, S., Bandyopadhyay, B., Maiti, R., Bhattacharya, K., & Karak, P. (2024). β-Amyloid peptide modulates peripheral immune responses and neuroinflammation in rats. *Biomolecular concepts*, *15*(1), 10.1515/bmc-2022-0042. <https://doi.org/10.1515/bmc-2022-0042>

95. Adhikary, K., Chatterjee, A., Banerjee, P.(2021). An updated review on nanomaterials for biomedical advancements: Concepts and applications. *Bioscience Biotechnology Research Communications*, *14*(4), 1428–1434. https://doi.org/10.21786/bbrc/14.4.9

96. Banerjee, P., Adhikary, K., Chatterjee, A., Sarkar, R., Bagchi, D., Ghosh, N., Das, A.(2021) Digestion and gut microbiome. In Bagchi, D(Ed.), Ohia, S(Ed.). Nutrition and Functional Foods in Boosting Digestion, Metabolism and Immune Health (123-138).United Kingdom: Academic Press.

97. Adhikary, K., Chatterjee, A., Chakraborty, S., Bhattacherjee, A., Banerjee, P.(2022).Malaria: Epidemiology, pathogenesis and therapeutics. In Bagchi, D (Ed.), Das, A (Ed.), Downs, B., W. Viral, Parasitic, Bacterial, and Fungal Infections (314-358).United Kingdom: Academic Press.

98. Banerjee, P., Adhikary, K., Sarkar, R., Chakraborty, S., Jana, S. (2022).Prion diseases: A raregroup of neurodegenerative disorders. In Bagchi, D (Ed.), Das, A (Ed.), Downs, B., W. Viral, Parasitic, Bacterial, and Fungal Infections (314-358).United Kingdom: Academic Press.

99. Chowdhury, M., & Chowdhury, S., Bhattacherjee, A., Roy, C., Sarkar, R., Adhikary, K., Maiti, R., & Karak, P. (2023). Natural antioxidants and nutraceuticals to fight against common human diseases: an overview. *European Chemical Bulletin*, 12(9), 1505-1521 <https://doi.org/10.48047/ecb>

100. Gupte, C., A., Kama, S., Gupta, A., Adhikary, K., & Ali, S. (2023). Predicting Protein Structure Using Deep Learning and Molecular Dynamics Simulations. *European Chemical Bulletin*, *12*(Special issue 4), 19882–19904. <https://doi.org/10.48047/ecb/2023.12.si4.1767>