Ortho Clinical Diagnostics

Donor Screening Infectious Diseases Learning Cards

North America

The information on this document has been created by the Global Transfusion Medicine Business Unit at Ortho Clinical Diagnostics and is intended for healthcare professionals for education purposes.

Donors and patients should consult a healthcare professional regarding specific medical conditions and treatments.

Assays availability may vary based on local regulations. Please consult your local representative for more information.

www.orthoclinicaldiagnostics.com

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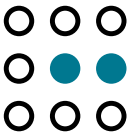
ANALYTICAL SENSITIVITY

Represents the smallest amount of substance in a sample that can accurately be measured by an assay. [1]

CLINICAL SENSITIVITY

It is the percentage of persons who have a given disorder who are identified by the assay as positive for the disorder. [1]



ANALYTICAL SPECIFICITY

Refers to the ability of an assay to measure on particular organism or substance, rather than others, in a sample. [1]

CLINICAL SPECIFICITY

It is the percentage of persons who do not have a given condition who are identified by the assay as negative for the condition. [1]

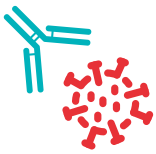
WINDOW PERIOD

In medicine, the window period for a test designed to detect a specific disease (particularly infectious disease) is the time between first infection and when the test can reliably detect that infection. In antibody-based testing, the window period is dependent on the time taken for seroconversion. [2]

Key Terms and Definitions

CELLULAR IMMUNE RESPONSE

It is a protective immune process that involves the activation of phagocytes, antigen-sensitized cytotoxic T cells and the release of cytokines and chemokines in response to antigen. Cellular immunity is most effective against cells infected with viruses, intracellular bacteria, fungi and protozoans, and cancerous cells. [3]



HUMORAL IMMUNE RESPONSE

The humoral immune response is mediated by antibody molecules that are secreted by plasma cells. [4]

INCIDENCE

It is a measure of the number of new cases of a characteristic that develop in a population in a specified time period. [5]

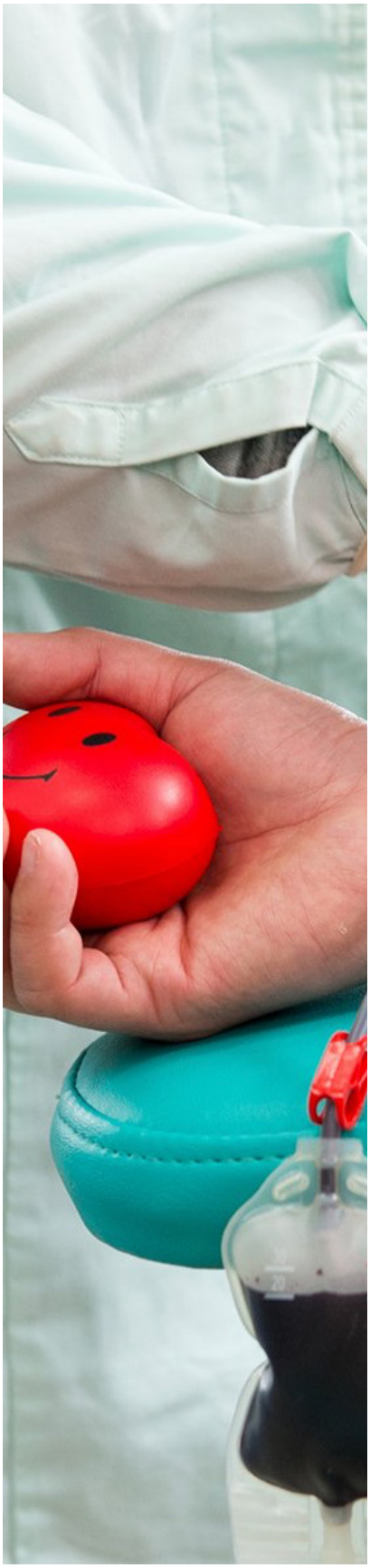
PREVALENCE

It is the proportion of a population who have a specific characteristic in a given time period, regardless of when they first developed the characteristic. [5]

1. Saah et al., "Sensitivity" and "specificity" reconsidered: the meaning of these terms in analytical and diagnostic settings. Ann Intern Med. 1997 Jan 1;126(1):91-4.
2. https://en.wikipedia.org/wiki/Window_period#:~:text=In%20medicine%2C%20the%20window%20period,the%20time%20taken%20for%20seroconversion.
3. https://www.nature.com/subjects/cellular-immunity
4. Immunobiology: The Immune System in Health and Disease. 5th edition
5. https://www.nimh.nih.gov/health/statistics/what-is-prevalence#:~:text=Incidence%20is%20a%20measure%20of,they%20first%20developed%20the%20characteristic.

Blood Donation Types

Blood donation is a voluntary procedure that can help save lives. There are several types of blood donation. Each type helps meet different medical needs.



WHOLE BLOOD DONATION

Whole blood donation is the most common type of blood donation. During this donation, the donor donates about a pint (about half a liter) of whole blood. The blood is then separated into its components; red cells, plasma and sometimes platelets.

APHERESIS

During apheresis, the donor is hooked up to a machine that collects and separates different parts of their blood. These blood components include red cells, plasma and platelets. The machine then returns the remaining parts of the blood back to the donor.

PLATELET DONATION (PLATELETPHERESIS)

Collects only platelets. Platelets are the cells that help stop bleeding by clumping and forming plugs in blood vessels (clotting).

- Donated platelets are commonly given to people with clotting problems or cancer and people who will have organ transplants or major surgeries.

DOUBLE RED CELL DONATION

Allows you to donate a concentrated amount of red blood cells. Red blood cells deliver oxygen to your organs and tissues.

- Donated red blood cells are typically given to people with severe blood loss, such as after an injury or accident, and people with sickle cell anemia.

PLASMA DONATION (PLASMAPHERESIS)

Collects the liquid portion of the blood (plasma). Plasma helps blood clot and contains antibodies that help fight off infections.

- Plasma is commonly given to people in emergency and trauma situations to help stop bleeding.

https://www.mayoclinic.org/tests-procedures/blood-donation/about/pac-20385144

Importance of Donating Blood

Ensuring a safe blood supply is critical so that blood products transfusions can help save lives

- Every two seconds, someone needs blood.
- A premature baby. A cancer patient. A burn victim. A new mother threatened with obstetric hemorrhaging. A patient suffering traumatic injuries. The impact of blood product donations is immense.
- 118.5 million total global blood donations/year. [1]
- 3 lives can potentially be saved with each blood donation. [1]
- 1 in 7 hospital patients needs blood. [2]
- There is no substitute for human blood — all transfusions use blood from a donor. [3]

1. World Health Organization. Blood safety and availability. Accessed August 20, 2021. https://www.who.int/news-room/fact-sheets/detail/blood-safety-and-availability
2. Community Blood Center. Blood donation facts. Accessed October 6, 2021. https://givingblood.org/about-blood/blood-facts.aspx
3. https://www.mayoclinic.org/tests-procedures/blood-donation/about/pac-20385144

Importance of Donating Plasma

Plasma is the liquid portion of blood. About 55% of our blood is plasma, and the remaining 45% are red blood cells, white blood cells and platelets that are suspended in the plasma. [1]

Plasma is about 92% water. It also contains 7% vital proteins such as albumin, gamma globulin and anti-hemophilic factor, and 1% mineral salts, sugars, fats, hormones and vitamins. [1]

The United States supplies 70% of the world's plasma - Plasma is essential in treating both common and rare infirmities from cancer, to trauma and burn victims, to hemophilia, and immune disorders. The therapies made possible by plasma proteins save lives every day and are essential to improving the lives of hundreds of thousand people suffering from immune deficiency and hemophilia. It takes 1,200 plasma donations to treat one hemophilia patient, and 130 plasma donations to treat one patient with a primary immune deficiency. [2]

- In a plasma-only donation, the liquid portion of the donor's blood is separated from the cells. [1]
- Blood is drawn from one arm and sent through a high-tech machine that collects the plasma. [1]
- The donor's red blood cells and platelets are then returned to the donor along with some saline. [1]
- The process is safe and only takes a few minutes longer than donating whole blood. [1]

Donated plasma is frozen within 24 hours of being donated to preserve its valuable clotting factors. It can be stored for up to one year and thawed for transfusion to a patient when needed. [1]

1. https://www.redcrossblood.org/donate-blood/dlp/plasma-information.html
2. www.PPTA.org
3. https://www.octapharmaplasma.com/what-is-plasma/
4. https://www.mayoclinic.org/tests-procedures/convalescent-plasma-therapy/about/pac-20486440

Importance of Donating Plasma

MEDICAL USES OF PLASMA [3]

Therapies: hemophilia, autoimmune disorder.

- Treatments: cancer, leukemia, lymphatic primary immune deficiency disorder.
- Emergencies: traumas, burns, shock.

OTHER USES

Blood donated by people who've recovered from COVID-19 has antibodies to the virus that causes it. The donated blood is processed to remove blood cells, leaving behind liquid (plasma) and antibodies. These can be given to people with COVID-19 to boost their ability to fight the virus. [4]

1. https://www.redcrossblood.org/donate-blood/dlp/plasma-information.html
2. www.PPTA.org
3. https://www.octapharmaplasma.com/what-is-plasma/
4. https://www.mayoclinic.org/tests-procedures/convalescent-plasma-therapy/about/pac-20486440

Donor Screening Process

It is important to determine if the donor has an infection that could be transmitted to recipients through the transplanted organs and/or tissues.

Blood/plasma donations are tested for multiple disease markers provide definitive evidence.

DONOR SCREENING PROCESS

The donor screening process **protects the safety of the transfusion recipient** by identifying eligible donors who meet specific criteria to help ensure blood safety.

- Blood tested to determine blood type and Rh factor. Blood type is classified as A, B, AB or O. The Rh factor refers to the presence or absence of a specific antigen — a substance capable of stimulating an immune response — in the blood. The donor will be classified as Rh positive or Rh negative. This information is important because the blood type and Rh factor must be compatible with the blood type and Rh factor of the person receiving the blood/plasma.
- Donor testing for transfusion-transmitted infectious diseases such as hepatitis and HIV. If these tests are negative, the blood is distributed for use in hospitals and clinics. If any of these tests are positive, the donor center notifies the donor and the blood product is discarded.

  Management of all donation information including adverse donation events and post donation information.
- Blood/plasma Donor Educational Material.
- Donor History Questionnaires (DHQ) and Related Materials designed to assess both the safety of the donor and the blood collection,
- A focused health exam including hemoglobin screening.

https://www.aabb.org/regulatory-and-advocacy/regulatory-affairs/regulatory-for-blood/donor-safety-screening-and-testing#:~:text=Key%20elements%20of%20the%20blood,4)%20donor%20testing%20for%20transfusion%2Dhttps://www.cdc.gov/transplantsafety/protecting-patient/screening-testing.html#:~:text=OPTN%20policy%20requires%20OPOs%20and,)%2C%20and%20toxoplasmosis%20(deceased%20donors

Transfusion Transmitted Infections

Blood products screening is vital to prevent infection through transfusion and organ transplantation all over the world. [1]

Transfusion-transmitted infections (TTIs) are infections resulting from the introduction of a pathogen into a person through blood products transfusion. [2]

A wide variety of organisms, including bacteria, viruses, prions, and parasites can be transmitted through blood transfusions. [2]

The use of a standard donor screening questionnaire as well as laboratory tests help to reduce the risk of an infectious organism being transmitted by blood transfusion. [2]

Per FDA requirements, all Human Cells, Tissues, and Cellular and Tissue-Based Products (HCT/P's) donors must be tested for the following infectious diseases: HIV, types 1 and 2; HBV; HCV; and Treponema pallidum. [3]

> Other infectious diseases are regulated by local guidelines, this may vary country by country Additional guidelines for emerging pathogens can be found in https://www.aabb.org/regulatory-and-advocacy/regulatory-affairs/regulatory-for-cellular-therapies/hctps/donor-eligibility-screening-and-testing.

1. https://www.who.int/news-room/fact-sheets
2. https://www.cdc.gov/bloodsafety/bbp/diseases-organisms.html#:~:text=Transfusion%2Dtransmitted%20infections%20(TTIs),be%20transmitted%20through%20blood%20transfusions.
3. https://www.aabb.org/regulatory-and-advocacy/regulatory-affairs/regulatory-for-cellular-therapies/hctps/donor-eligibility-screening-and-testing

Human Immunodeficiency Virus (HIV)

Overview

HUMAN IMMUNODEFICIENCY VIRUS

In September 1982, the CDC had labeled the condition acquired immunodeficiency syndrome (AIDS) and by 1984, researchers had identified the cause as a virus they named human immunodeficiency virus (HIV). [4]

Human immunodeficiency virus (HIV) is a retrovirus (genus Lentivirus) with a single-stranded, positive-sense RNA genome. [3]

Upon entry of the target cell, the viral RNA genome is converted to double-stranded DNA by a virally encoded reverse transcriptase that is present in the virus particle. [3]

This viral DNA is then integrated into the cellular DNA by a virally encoded integrase allowing the genome to be transcribed. Once the virus has infected the cell, two pathways are possible: either the virus becomes latent and the infected cell continues to function, or the virus becomes active and replicates, and a large number of virus particles are liberated to infect other cells. [3]

HIV invades various immune cells (e.g., CD4+ T cells and monocytes) resulting in a decline in CD4+ T cell numbers below the critical level, and loss of cell-mediated immunity. [1]

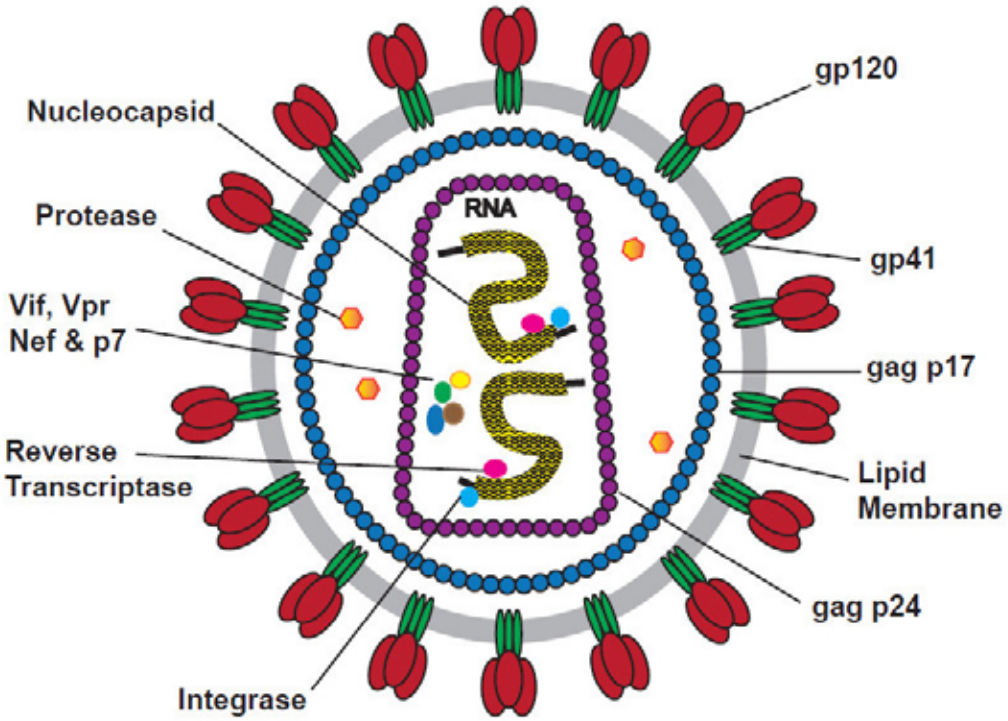
The virus devastates the immune system because its main target is the T4 lymphocyte, which is the key component for generating and regulating the immune response. [2]

1. https://www.immunology.org/public-information/bitesized-immunology/pathogens-and-disease/human-immunodeficiency-virus-hiv
2. Immunological features of human immunodeficiency virus disease. PubMed. Clin Haematol. 1990 Jan;3(1):37-63
3. https://thenativeantigencompany.com/products/human-immunodeficiency-virus-p24-protein-hiv-1-clade-c/
4. https://www.gileadhiv.com/landscape/history-of-hiv/?utm_id=iw_sa_15442187160_127739509142&utm_medium=cpc&utm_term=origin+of+hiv&gclid=Cj0KCQjwuaiXBhCCARIsAKZLt3mTn302mOPtb02pDGzblea7c0gUpLuaQuu0k4lXKccSyhtiWGJtDplaAjRwEALw_wcB&gclsrc=aw.ds

Overview

HIV can destroy CD4 cells by direct virus cytotoxicity and indirectly through the host response against HIV-infected cells or gp120-targeted cells. [2]

The gag gene of HIV-1 encodes a precursor protein known as Pr55Gag. The viral protease PR cleaves this precursor to generate p17, p24, p7, and p6 proteins which are required for virus particle assembly. p24 is a major viral core structural protein. Its measurement is commonly used as an indicator of HIV-1 infection and viral load. P24 is present at high copy number in HIV-1 virions. The onset of symptoms of AIDS correlates with increased levels of virus and p24 in the blood and a reduction in the number of CD4 T-cells which can be detected as early as 2 weeks after HIV infection using immunoassays. [3]



Structure of HIV [5]

1. https://www.immunology.org/public-information/bitesized-immunology/pathogens-and-disease/human-immunodeficiency-virus-hiv
2. Immunological features of human immunodeficiency virus disease. PubMed. Clin Haematol. 1990 Jan;3(1):37-63
3. https://thenativeantigencompany.com/products/human-immunodeficiency-virus-p24-protein-hiv-1-clade-c/
4. https://www.gileadhiv.com/landscape/history-of-hiv/?utm_id=iw_sa_15442187160_127739509142&utm_medium=cpc&utm_term=origin+of+hiv&gclid=Cj0KCQjwuaiXBhCCARIsAKZLt3mTn302mOPtb02pDGzblea7c0gUpLuaQuu0k4lXKccSyhtiWGJtDplaAjRwEALw_wcB&gclsrc=aw.ds
5. https://www.eenzyme.com/HIV-research-tools.aspx

INNATE IMMUNE RESPONSE TO HIV

Innate immune cells are the first line of defense which HIV encounters upon entry to the body:

- Macrophages
- Dendritic cells (DCs)
- Natural killer (NK) cells

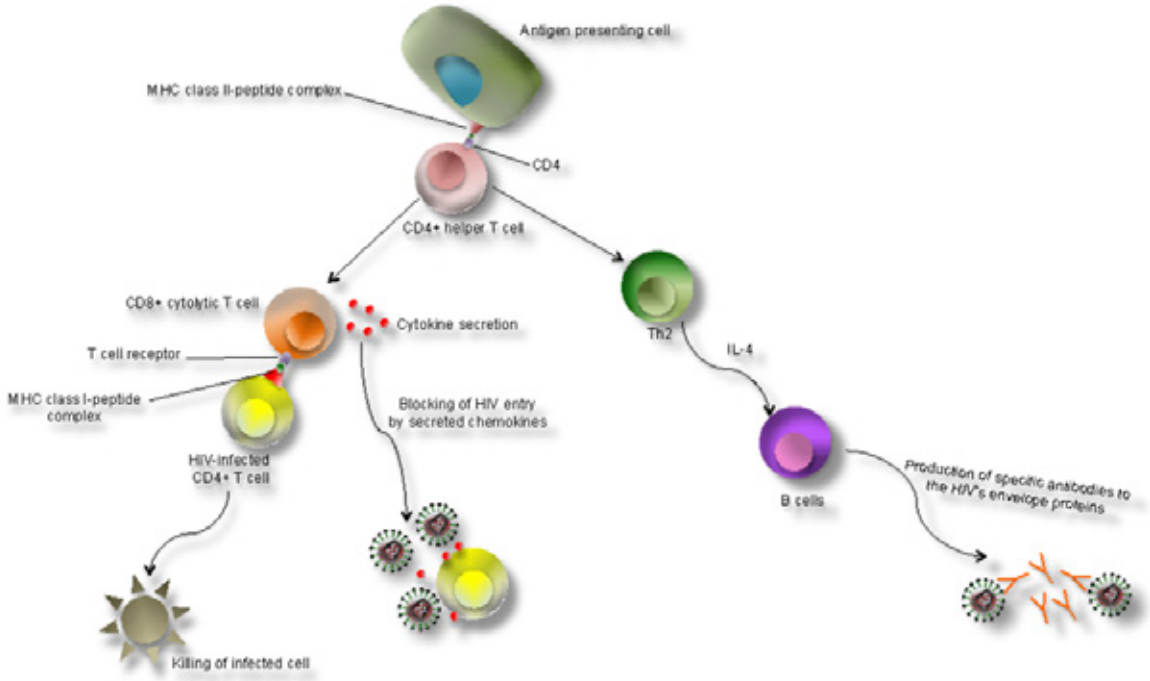
ADAPTIVE IMMUNE RESPONSE TO HIV

- Cellular immune response:

It is induced upon the entry of HIV into the target cells (e.g., T cells) and synthesis of viral proteins.

HUMORAL RESPONSE

It occurs later in infection; therefore, the level of antibodies during the acute infection is very low. Non-neutralizing antibodies to structural proteins (i.e. P17 and P24) are first to appear and generally do not persist.



Cellular & humoral immune responses to HIV

https://www.immunology.org/public-information/bitesized-immunology/pathogens-and-disease/human-immunodeficiency-virus-hiv

Possible Causes, Clinical Signs & Symptons

Acquired immunodeficiency syndrome (AIDS) is a chronic, potentially life-threatening condition caused by the human immunodeficiency virus (HIV).
By damaging the immune system, HIV interferes with the body's ability to fight infection and disease.

- HIV is a sexually transmitted infection (STI).
- It can also be spread by contact with infected blood and from illicit injection drug use or sharing needles.
- It can also be spread from mother to child during pregnancy, childbirth or breastfeeding.

Primary infection (Acute HIV)

- Some people infected by HIV develop a flu-like illness within 2 to 4 weeks after the virus enters the body.
- Possible signs and symptoms include:
- Fever
- Headache
- Muscle aches and joint pain
- Rash
- Sore throat and painful mouth sores
- Swollen lymph glands, mainly on the neck
- Diarrhea
- Weight loss
- Cough
- Night sweats

Symptomatic HIV infection

- As the virus continues to multiply and destroy the immune cells, the patient may develop mild infections or chronic signs and symptoms such as:
- Fever
- Fatigue
- Swollen lymph nodes — often one of the first signs of HIV infection
- Diarrhea
- Weight loss
- Oral yeast infection (thrush)
- Shingles (herpes zoster)
- Pneumonia

Chronic Latent Infection (Chronic HIV)

- In this stage of infection, HIV is still present in the body and in white blood cells. However, many people may not have any symptoms or infections during this time.
- This stage can last for many years if the person is receiving antiretroviral therapy (ART). Some people develop more severe disease much sooner.

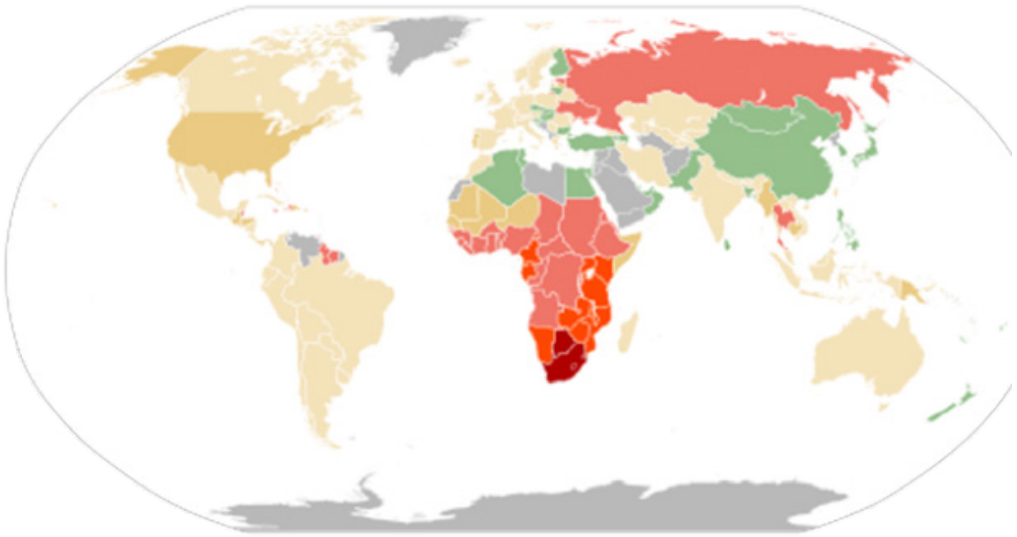
Chronic Latent Infection (Chronic HIV)

- Untreated, HIV typically turns into AIDS in about 8 to 10 years.
- When AIDS occurs, the immune system has been severely damaged. Opportunistic infections may start appearing more frequently.
- The signs and symptoms of some of these infections may include:
- Sweats
- Chills
- Recurring fever
- Chronic diarrhea
- Swollen lymph glands
- Persistent white spots or unusual lesions on your tongue or in your mouth
- Persistent, unexplained fatigue
- Weakness
- Weight loss
- Skin rashes or bumps

https://www.mayoclinic.org/diseases-conditions/hiv-aids/symptoms-causes/syc-20373524

Incidence & Prevalence

- HIV continues to be a serious health issue for parts of the world.
- Worldwide, there were about 1.5 million new cases of HIV in 2020.
- About 37.7 million people were living with HIV around the world in 2020.
- An estimated 680,000 people died from AIDS-related illnesses in 2020.
- An estimated 36.3 million people have died from AIDS-related illnesses since the start of the epidemic.
- Eastern and Southern Africa is the region most affected by HIV worldwide, and accounts for about 45% of all new HIV infections.
- Other regions significantly affected by HIV include Asia and the Pacific, Western and Central Africa, Western and Central Europe and North America, and Latin America.



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1. https://www.cdc.gov/hiv/statistics/overview/index.html
2. https://en.wikipedia.org/wiki/Epidemiology_of_HIV/AIDS

Hepatitis B

Overview

HEPATITIS B

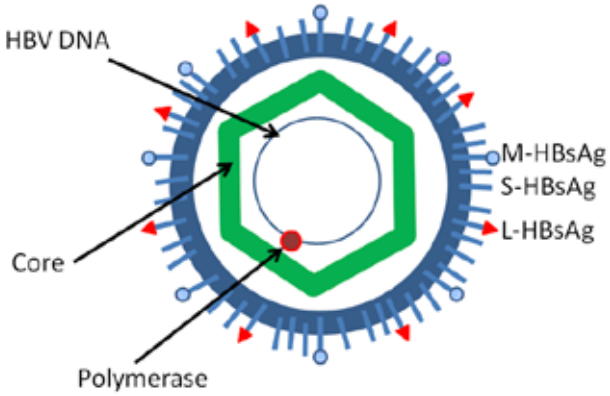
The hepatitis B virus was discovered in 1965 by Dr. Baruch Blumberg who won the Nobel Prize for his discovery. [3]

Hepatitis B virus (HBV) is a partially double-stranded DNA virus a species of the genus Orthohepadnavirus and a member of the Hepadnaviridae family of viruses. This virus causes the disease hepatitis B. [1]

The virus is one of the smallest enveloped animal viruses. The 42 nm virions, which are capable of infecting liver cells known as hepatocytes, are referred to as "Dane particles". [1]

Hepatitis B virus (HBV) is a non-cytopathic, hepatotropic virus with the potential to cause a persistent infection, ultimately leading to cirrhosis and hepatocellular carcinoma. [2]

There is an outer shell (or envelope) composed of lipid and protein that is termed "surface antigen" or "HBsAg". [4]
Inner protein shell that is referred to as the core particle or "HBcAg", contains the viral DNA and enzymes used in viral replication (called "DNA polymerase"). [4]
HBeAg (hepatitis B e antigen) is the antigenic determinant that is closely associated with the nucleocapsid of HBV. It also circulates as a soluble protein in serum. [4]



Structure of Hepatitis B Virus [5]

1. https://en.wikipedia.org/wiki/Hepatitis_B
2. Iannacone et al., Immunobiology and pathogenesis of hepatitis B virus infection. Nature Reviews Immunology. 17 May 2021.
3. https://www.hepb.org/prevention-and-diagnosis/vaccination/history-of-hepatitis-b-vaccine/#:~:text=The%20hepatitis%20B%20virus%20was,of%20an%20American%20hemophilia%20patient.
4. https://microbiologyinfo.com/hepatitis-b-virus-structure-epidemiology-symptoms-pathogenesis-diagnosis-treatment-and-vaccines/
5. https://commons.wikimedia.org/wiki/User:Graham_Beards

Immunology

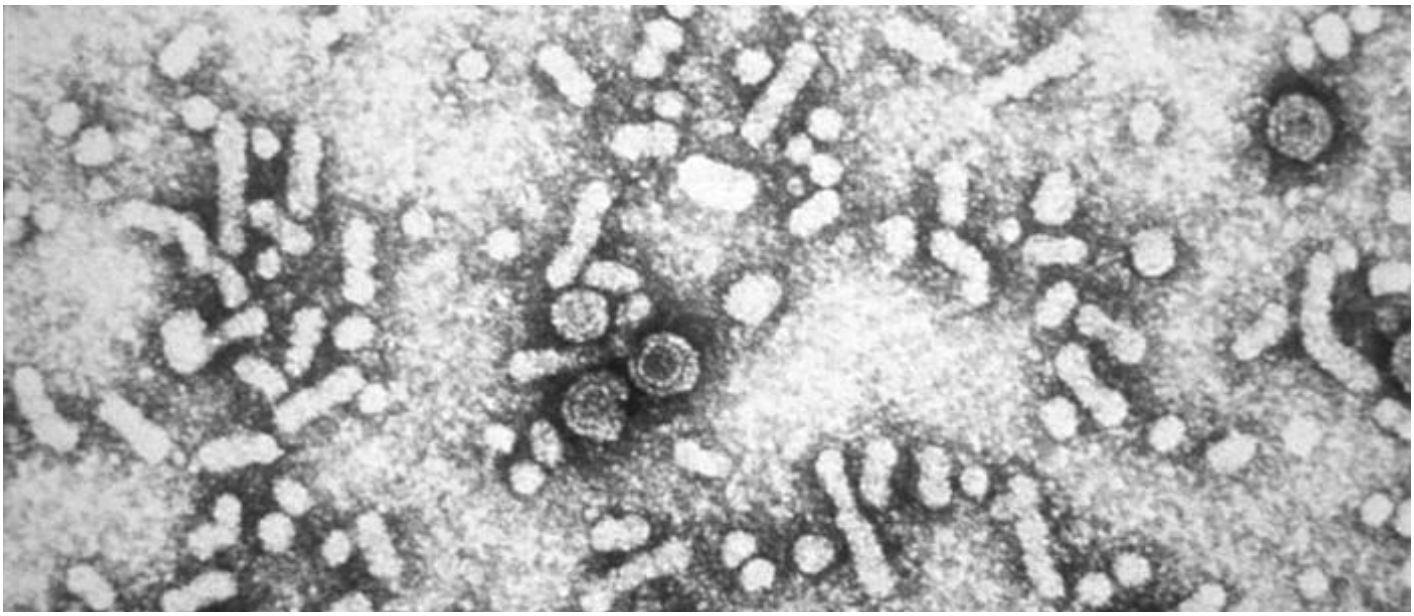
Hepatitis B virus primarily interferes with the functions of the liver by replicating in hepatocytes. A functional receptor is NTCP.

There is evidence that the receptor in the closely related duck hepatitis B virus is carboxypeptidase D.

The virions bind to the host cell via the preS domain of the viral surface antigen and are subsequently internalized by endocytosis. HBV-preS-specific receptors are expressed primarily on hepatocytes; however, viral DNA and proteins have also been detected in extrahepatic sites, suggesting that cellular receptors for HBV may also exist on extrahepatic cells.

During HBV infection, the host immune response causes both hepatocellular damage and viral clearance. Although the innate immune response does not play a significant role in these processes, the adaptive immune response, in particular virus-specific cytotoxic T lymphocytes (CTLs), contributes to most of the liver injury associated with HBV infection.

CTLs eliminate HBV infection by killing infected cells and producing antiviral cytokines, which are then used to purge HBV from viable hepatocytes.



Electron Micrograph of Hepatitis B Virus

https://en.wikipedia.org/wiki/Hepatitis_B

Possible Causes, Clinical Signs & Symptoms

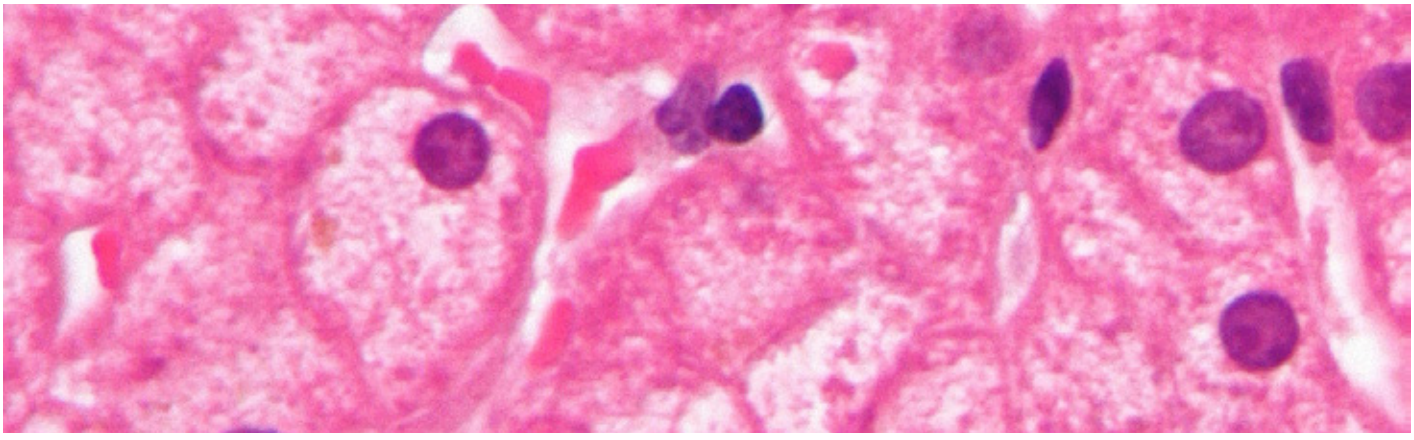
Hepatitis B is an infectious disease caused by the hepatitis B virus (HBV) that affects the liver; it is a type of viral hepatitis. It can cause both acute and chronic infection.

The virus is transmitted by exposure to infectious blood or body fluids. In areas where the disease is common, infection around the time of birth or from contact with other people's blood during childhood are the most frequent methods by which hepatitis B is acquired. In areas where the disease is rare, intravenous drug use and sexual intercourse are the most frequent routes of infection. Other risk factors include working in healthcare, blood transfusions, dialysis, living with an infected person, travel in countries with high infection rates, and living in an institution.

Many people have no symptoms during an initial infection.

In acute infection, some may develop a rapid onset of sickness with vomiting, yellowish skin, tiredness, dark urine, and abdominal pain. These symptoms often last a few weeks and initial infection rarely results in death. Once infected, it may take 30 to 180 days for symptoms to appear.

Among those infected around the time of birth, **90%** develop chronic hepatitis B, while less than **10%** of those infected after the age of five develop chronic cases. Most of those with chronic disease have no symptoms; however, cirrhosis and liver cancer eventually develop in about **25%** of those with chronic HBV.



Ground Glass Hepatocytes as seen in a Chronic Hepatitis B Liver Biopsy. H&E Stain

https://en.wikipedia.org/wiki/Hepatitis_B

Incidence & Prevalence

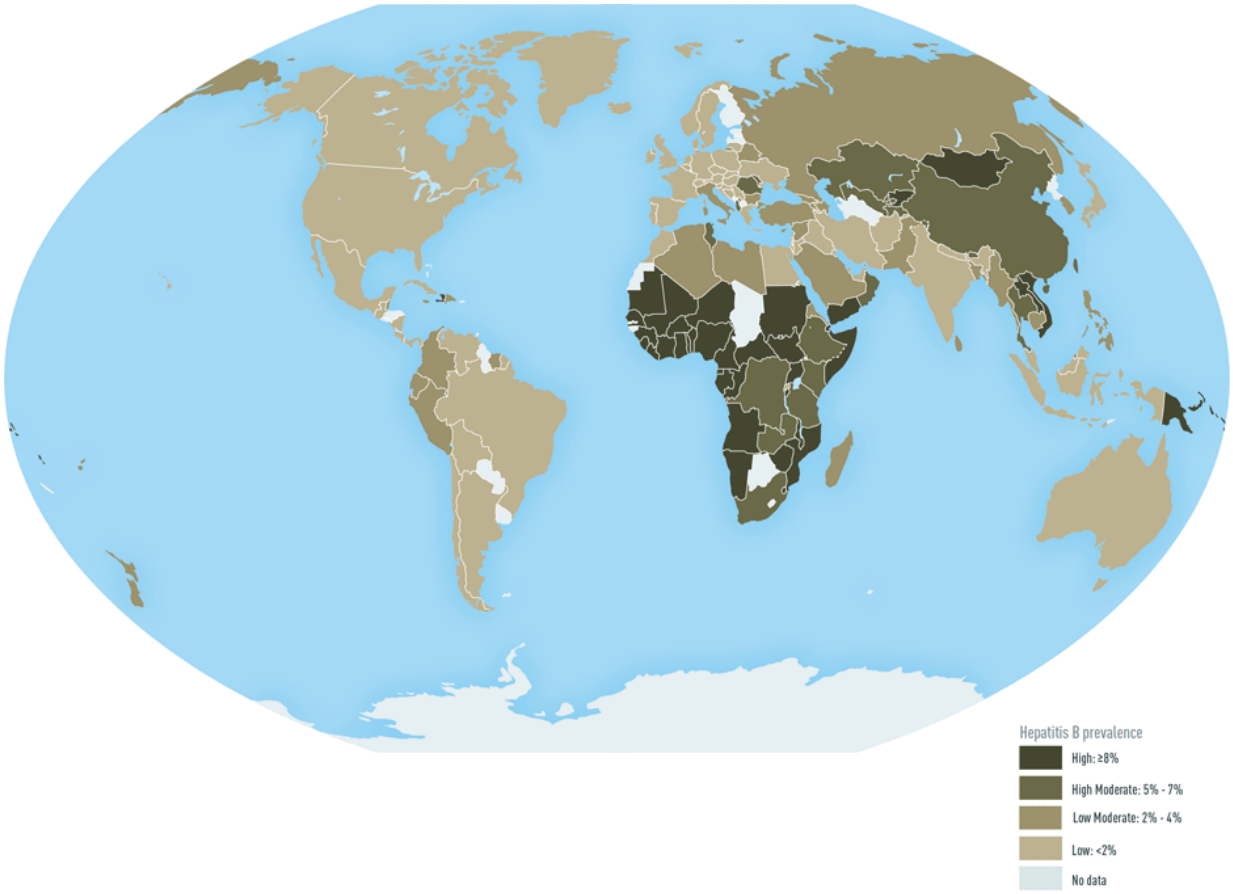
- In 2018, a total of 3,322 cases of acute hepatitis B were reported to CDC, for an overall incidence rate of 1.0 cases per 100,000 population. After adjusting for under-ascertainment and under-reporting, an estimated 21,600 acute hepatitis B cases occurred in 2018.

- The rate of reported acute HBV infections declined approximately 90% since recommendations for HepB vaccination were first issued, from 9.6 cases per 100,000 population in 1982 to 1.0 cases per 100,000 population in 2018.

- WHO estimates that 296 million people were living with chronic hepatitis B infection in 2019, with 1.5 million new infections each year.

- An estimated 850,000 to 2.2 million persons in the United States are chronically infected with HBV.

- In 2019, hepatitis B resulted in an estimated 820 000 deaths, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer).



https://www.cdc.gov/vaccines/pubs/pinkbook/hepb.html#:~:text=In%202018%2C%20a%20total%20of,B%20cases%20occurred%20in%202018.
https://www.who.int/news-room/fact-sheets/detail/hepatitis-b

Hepatitis C

Overview

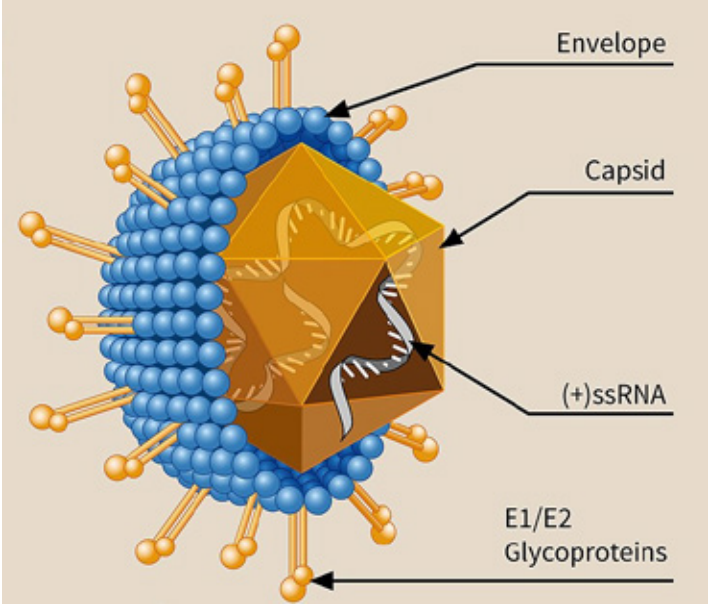
HEPATITIS C

A trio of scientists identified and characterized the virus responsible for many cases of hepatitis and liver disease — hepatitis C — they are the recipients of the 2020 Nobel Prize in Physiology or Medicine, Dr. Harvey Alter, Dr. Michael Houghton and Dr. Charles Rice; with the first discovery by Dr. Alter in 1972 that hepatitis still occurred in patients who had received blood transfusions, and whatever blood-borne agent was causing the disease was not being screened out by tests that had been developed for hepatitis A or B. [1]

The hepatitis C virus (HCV) is a small (55–65 nm in size), enveloped, positive-sense single-stranded RNA virus of the family Flaviviridae. [2]

The hepatitis C virus is the cause of hepatitis C and some cancers such as liver cancer (hepatocellular carcinoma, abbreviated HCC) and lymphomas in humans. [2]

The hepatitis C virus particle consists of a lipid membrane envelope. Two viral envelope glycoproteins, E1 and E2, are embedded in the lipid envelope. They take part in viral attachment and entry into the cell. Within the envelope is an icosahedral core. Inside the core is the RNA material of the virus. [2]



Structure of Hepatitis C Virus

1. https://www.nature.com/articles/d41586-020-02763-x
2.. https://en.wikipedia.org/wiki/Hepatitis_C_virus

Immunology

Replication of HCV involves several steps. The virus replicates mainly in the hepatocytes of the liver, where it is estimated that daily each infected cell produces approximately fifty virions (virus particles) with a calculated total of one trillion virions generated. [1]

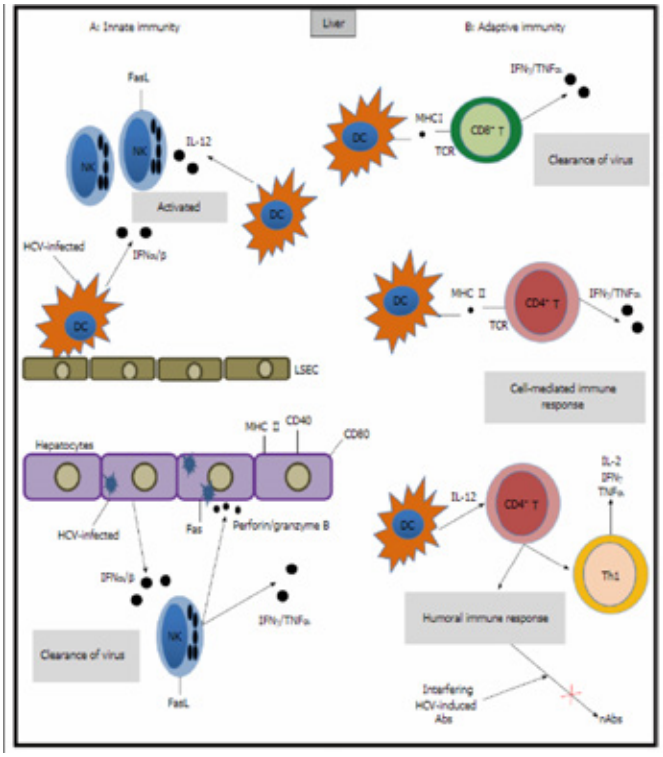
The virus may also replicate in peripheral blood mononuclear cells, potentially accounting for the high levels of immunological disorders found in chronically infected HCV patients. [1]

In the liver, the HCV particles are brought into the hepatic sinusoids by blood flow. These sinusoids neighbor hepatocyte cells. [1]

HCV is able to pass through the endothelium of the sinusoids and make its way to the basolateral surface of the hepatocyte cells. [1]

The mechanisms that regulate disease progression during hepatitis C virus (HCV) infection and the response to treatment are not clearly identified. [2]

Successful clearance of HCV infection requires the coordinated action of innate immunity and acquired immunity. After infection, there is activation of natural killer (NK) cells, as well as processing of viral antigens by immature dendritic cells (iDCs). [2]



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1. https://en.wikipedia.org/wiki/Hepatitis_C_virus
2.Koziel., Cellular Immune Responses against Hepatitis C Virus. Clinical Infectious Diseases, Volume 41, Issue Supplement_1, July 2005
3. Fierro et al., Immunologic, metabolic and genetic factors in hepatitis C virus infection. World J Gastroenterol 2014 April 7; 20(13): 3443-3456
4. Mechanisms of Immune response to hepatitis C virus. Multiple innate (A) and adaptive immune (B) components are involved in hepatitis C virus (HCV) infection. Dendritic cells (DC), natural killer (NK) cells, CD4+, CD8+ T cells and B-cell mediated humoral immune response are crucial for disease outcome. IFN- : interferon-gamma; TNF-a: tumor necrosis factor-a; IL: Interleukin; nAbs: Neutralizing anti-HCV antibodies. 3

Possible Causes, Clinical Signs & Symptons

The hepatitis C virus is usually spread when someone comes into contact with blood from an infected person. This can happen through:

- SHARING DRUG-INJECTION EQUIPMENT: Most people become infected with hepatitis C by sharing needles, syringes, or any other equipment used to prepare and inject drugs.
- BIRTH: Approximately 6% of infants born to infected mothers will get hepatitis C.
- HEALTH CARE EXPOSURES: Although uncommon, people can become infected when health care professionals do not follow the proper steps needed to prevent the spread of bloodborne infections.
- SEX WITH AN INFECTED PERSON: While uncommon, hepatitis C can spread during sex, though it has been reported more often among men who have sex with men.
- UNREGULATED TATTOOS OR BODY PIERCINGS: Hepatitis C can spread when getting tattoos or body piercings in unlicensed facilities, informal settings, or with non-sterile instruments.
- SHARING PERSONAL ITEMS: People can get infected from sharing glucose monitors, razors, nail clippers, toothbrushes, and other items that may have come into contact with infected blood, even in amounts too small to see.
- BLOOD TRANSFUSIONS AND ORGAN TRANSPLANTS

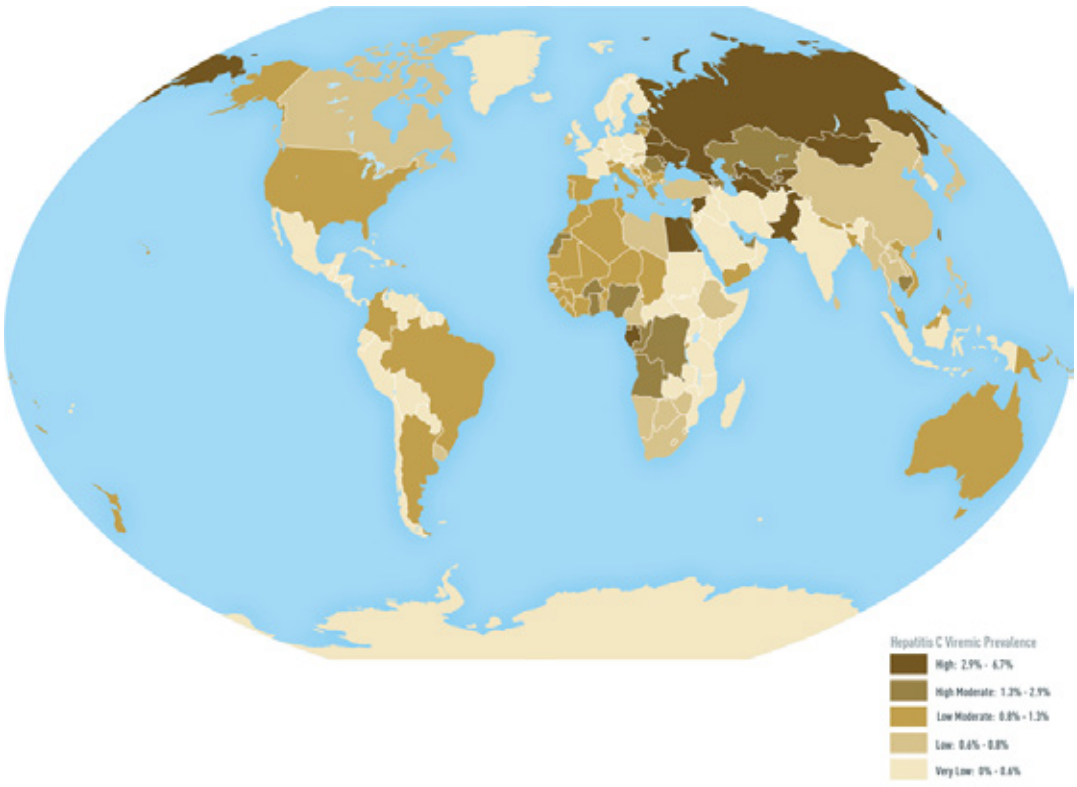
Many people with hepatitis C do not have symptoms and do not know they are infected.
If symptoms occur, they can include: yellow skin or eyes, not wanting to eat, upset stomach, throwing up, stomach pain, fever, dark urine, light-colored stool, joint pain, and feeling tired.
If symptoms occur with a new infection, they usually appear within 2 to 12 weeks, but can take up to 6 months to develop.

People with chronic hepatitis C can live for years without symptoms or feeling sick. When symptoms appear with chronic hepatitis C, they often are a sign of advanced liver disease. Acute HCV infections are usually asymptomatic and most do not lead to a life-threatening disease. Around 30% (15–45%) of infected persons spontaneously clear the virus within 6 months of infection without any treatment. The remaining 70% (55–85%) of persons will develop chronic HCV infection. Of those with chronic HCV infection, the risk of cirrhosis ranges from 15% to 30% within 20 years. [2]

1. https://en.wikipedia.org/wiki/Hepatitis_C_virus
2. Koziel., Cellular Immune Responses against Hepatitis C Virus. Clinical Infectious Diseases, Volume 41, Issue Supplement_1, July 2005
3. Fierro et al., Immunologic, metabolic and genetic factors in hepatitis C virus infection. World J Gastroenterol 2014 April 7; 20(13): 3443-3456

Incidence and Prevalence

- Globally, an estimated 58 million people have chronic hepatitis C virus infection, with about 1.5 million new infections occurring per year.
- There are an estimated 3.2 million adolescents and children with chronic hepatitis C infection.
- WHO estimated that in 2019, approximately 290 000 people died from hepatitis C, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer).
- The highest burden of disease is in the Eastern Mediterranean Region and European Region, with 12 million people chronically infected in each region.
- In the South-East Asia Region and the Western Pacific Region, an estimated 10 million people in each region are chronically infected.
- Nine million people are chronically infected in the African Region and 5 million the Region of the Americas.



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1. https://www.who.int/news-room/fact-sheets/detail/hepatitis-c#:~:text=Globally%2C%20an%20estimated%2058%20million,with%20chronic%20hepatitis%20C%20infection.
2. https://wwwnc.cdc.gov/travel/yellowbook/2020/travel-related-infectious-diseases/hepatitis-b

Syphilis

Overview

SYPHILIS

Syphilis is a sexually transmitted disease caused by Treponema Pallidum, a gram-negative bacteria classified under Spirochaets phylum, Spirochaetales order, Spirochaetaceae family. [1]

In 1905, Fritz Richard Schaudinn, a German zoologist, and Erich Hoffman, a dermatologist, discovered the cause of syphilis: the bacterium called Treponema pallidum. [2]

Treponema pallidum has one of the smallest bacterial genomes at 1.14 million base pairs, and has limited metabolic capabilities, reflecting its adaptation through genome reduction to the rich environment of mammalian tissue. [3]

The shape of T. pallidum is flat and wavy. In order to avoid antibodies attacking, the cell has few proteins exposed on the outer membrane sheath. Its chromosome of about 1000 kilo base pairs is circular with a 52.8% G + C average. Sequencing has revealed a bundle of twelve proteins and some putative hemolysins are potential virulence factors of T. pallidum. [3]

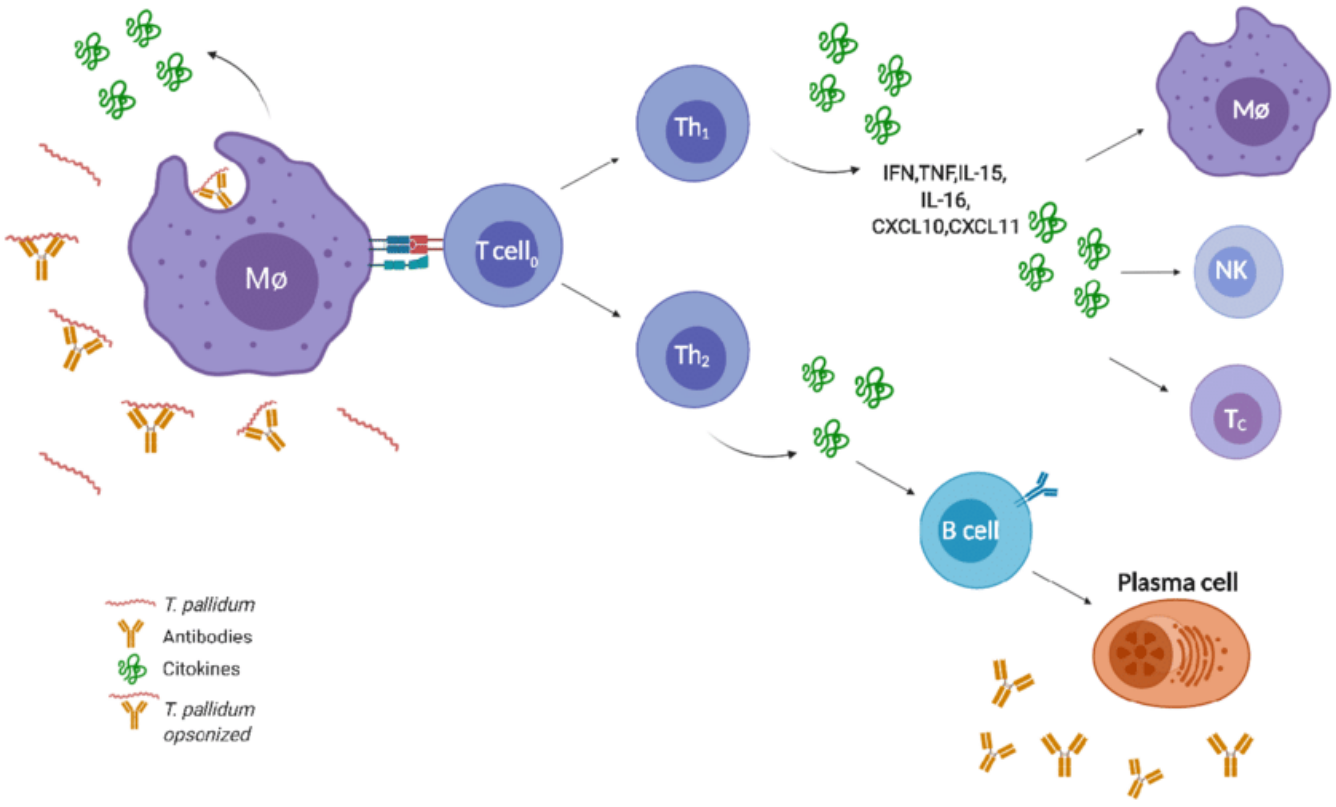
Treponema Pallidum Bacteria [4]

1. Brief History of Syphilis. Tampa et al., J Med Life. 2014 Mar 15; 7(1): 4–10
2. https://www.everydayhealth.com/syphilis/painful-history-odd-bug/
3. https://en.wikipedia.org/wiki/Treponema_pallidum
4. https://www.istockphoto.com/photo/treponema-pallidum-bacterium-gm486710522-73520673?phrase=treponema%20pallidum%20bacteria

Variation in the expression of the different Tpr proteins in the syphilis spirochete, Treponema pallidum subsp. pallidum, may have important implications in its ability to evade host immune detection and cause persistent infection.

During the early stages of infection, the host mounts a vigorous immune response that is able to clear the majority of the treponemes from early lesions but is unable to completely eradicate the infection. Phagocytosis of opsonized treponemes by macrophages is the primary mechanism by which the host immune system clears treponemes from early lesions.

While T. pallidum has been shown to be susceptible to bactericidal and opsonic antibodies, the inability to culture the organism, combined with the fragile nature of its outer membrane, have made the positive identification of outer surface molecules that may be antibody targets difficult and often inconclusive.



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1. Antibody Responses Elicited against the Treponema pallidum Repeat Proteins Differ during Infection with Different Isolates of Treponema pallidum subsp. Pallidum. Leader et al., ASM Journals Infection and Immunity Vol. 71, No. 10
2. New perspectives in the study of the congenital syphilis: A narrative review. September 2020
DOI:10.34119/bjhrv3n4-255

Possible Causes, Clinical Signs & Symptoms

Syphilis is a bacterial infection usually spread by sexual contact.

The disease starts as a painless sore — typically on the genitals, rectum or mouth. Syphilis spreads from person to person via skin or mucous membrane contact with these sores.

After the initial infection, the syphilis bacteria can remain inactive in the body for decades before becoming active again. Early syphilis can be cured, sometimes with a single shot (injection) of penicillin.

Without treatment, syphilis can severely damage the heart, brain or other organs, and can be life-threatening. Syphilis can also be passed from mothers to unborn children. Approximately two thirds of infected people who are not treated, will suffer no consequences of the infection.

Primary Syphilis

The first sign of syphilis is a small sore, called a chancre. The sore appears at the spot where the bacteria entered your body.

The chancre usually develops about three weeks after exposure.

Secondary Syphilis

Within a few weeks a rash usually appears, it may be not itchy and may be accompanied by wartlike sores in mouth or genital area

Latent Syphilis

If the person is not treated for syphilis, the disease moves from the secondary stage to the hidden (latent) stage. The latent stage can last for years. Signs and symptoms may never return, or the disease may progress to the third (tertiary) stage.

Tertiary Syphilis

About 15% to 30% of people infected with syphilis who don't get treatment will develop complications known as tertiary syphilis. In the late stage, the disease may damage the brain, nerves, eyes, heart, blood vessels, liver, bones and joints.

Neurosyphilis

At any stage, syphilis can spread and, among other damage, cause damage to the brain and nervous system and the eye.

Congenital Syphilis

Babies born to women who have syphilis can become infected through the placenta or during birth.

https://www.mayoclinic.org/diseases-conditions/syphilis/symptoms-causes/syc-20351756

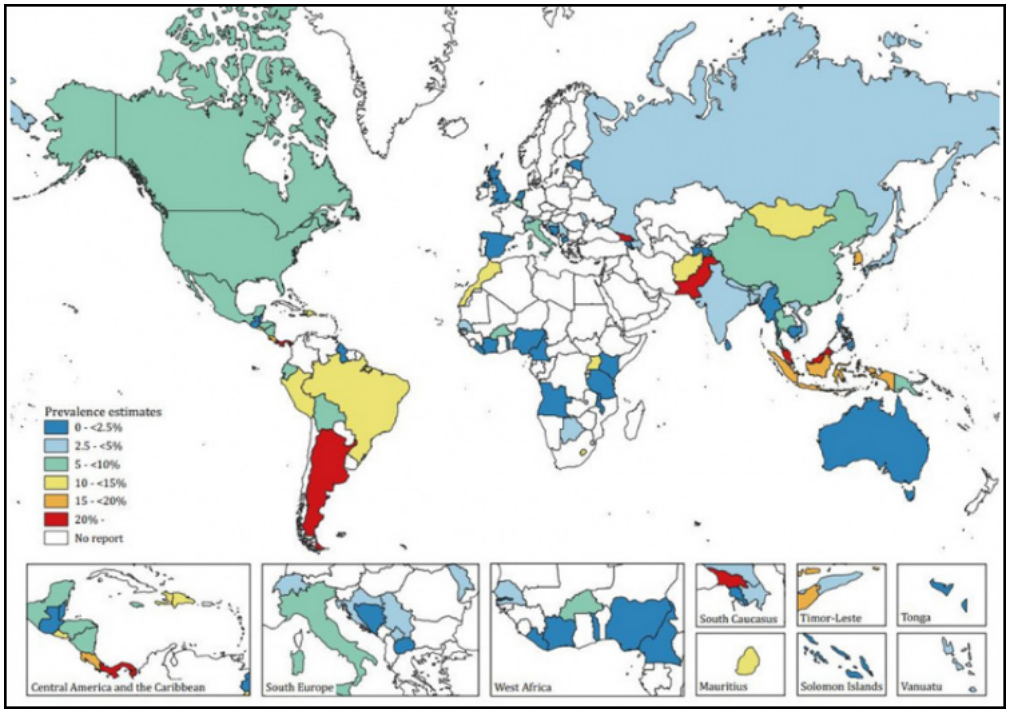
Incidence and Prevalence

- Globally, there were an estimated 7 million new syphilis infections in 2020.

- In the US, In 2020, 133,945 cases of all stages of syphilis were reported, including 41,655 cases of primary and secondary (P&S) syphilis, the most infectious stages of the disease. Since reaching a historic low in 2000 and 2001, the rate of P&S syphilis has increased almost every year, increasing 6.8% during 2019–2020.

- The 2013 rate of congenital syphilis (9.2 cases per 100,000 live births) marked the first increase in congenital syphilis since 2008. Since 2013, the rate of congenital syphilis has increased each year. In 2020, 2,148 cases of congenital syphilis were reported, including 149 congenital syphilis-related stillbirths and infant deaths.

- The national congenital syphilis rate of 57.3 cases per 100,000 live births in 2020 represents a 15% increase relative to 2019 and 254% increase relative to 2016.



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1. https://www.cdc.gov/std/statistics/2020/overview.htm#Syphilis
2. https://www.lshtm.ac.uk/newsevents/news/2021/globally

Chagas Disease

Overview

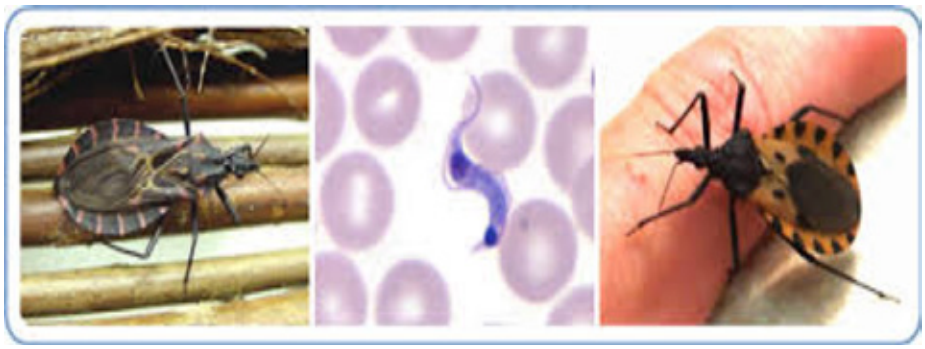
CHAGAS DISEASE

Chagas disease is named after Carlos Ribeiro Justiniano Chagas, a Brazilian physician and researcher who discovered the disease in 1909. [1]

Trypansosoma cruzi, a blood-borne protozoan parasite, causes Chagas disease – first enters the human blood stream by blood sucking triatomine bugs. [1]

The parasite reaches the secondary lymphoid organs, the heart, skeletal muscles, neurons in the intestine and esophagus among other tissues. The disease is characterized by mega syndromes, which may affect the esophagus, the colon and the heart, in about 30% of infected people. [1]

The disease is found mainly in endemic areas of 21 continental Latin American countries, where it has been mostly transmitted to humans and other mammals by contact with faeces or urine of triatomine bugs (vector-borne), known as kissing bugs. [2]



Trypanosoma Cruzi [3]

1. Immunity and immune modulation in Trypanosoma cruzi infection. Cardillo et al., Pathog Dis. 2015 Oct 5
2. https://www.who.int/news-room/fact-sheets/detail/chagas-disease-(american-trypanosomiasis)
3. https://asm.org/Articles/2021/April/Chagas-Disease-in-the-U-S-What-We-Know-About-the-K

Immunology

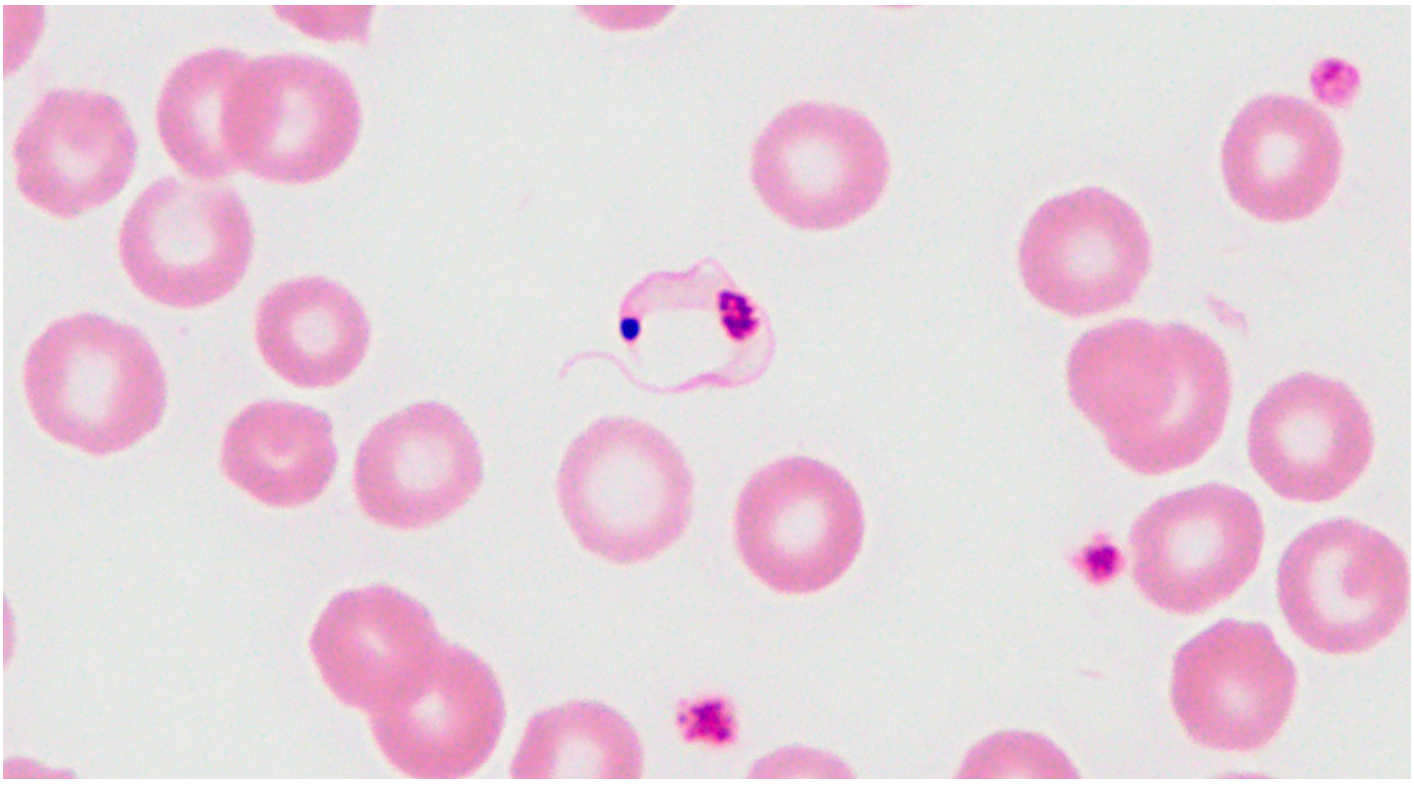
The infection is characterized by an acute phase resulting in parasitemia that resolves upon the appearance of an effective immune response (Cardillo et al. 2002).

The acute infection is characterized by high parasitemia that increases after 1–8 weeks following infection, depending on the T. cruzi strain (Cardillo et al. 1996).

However, the immune response induced during the acute infection is not sufficient to completely eradicate the pathogen, thus resulting in chronic infection (Albareda et al. 2006).

The chronic infection may be accompanied by additional autoimmune mechanisms triggered by the parasite and its persistence (dos Santos et al. 1992; Mengel and Rossi 1992; Bonney and Engman 2008, 2015; Cunha-Neto et al. 2011).

Initial IFN- production prior to the generation of T-cellmediated adaptive immunity is known to occur during the course of many infections and may be important in the development of resistance to many intracellular infections (Locksley and Scott 1991; Ramarathinam, Niesel and Klimpel 1993; Sher et al. 1993; Cardillo et al. 1996).



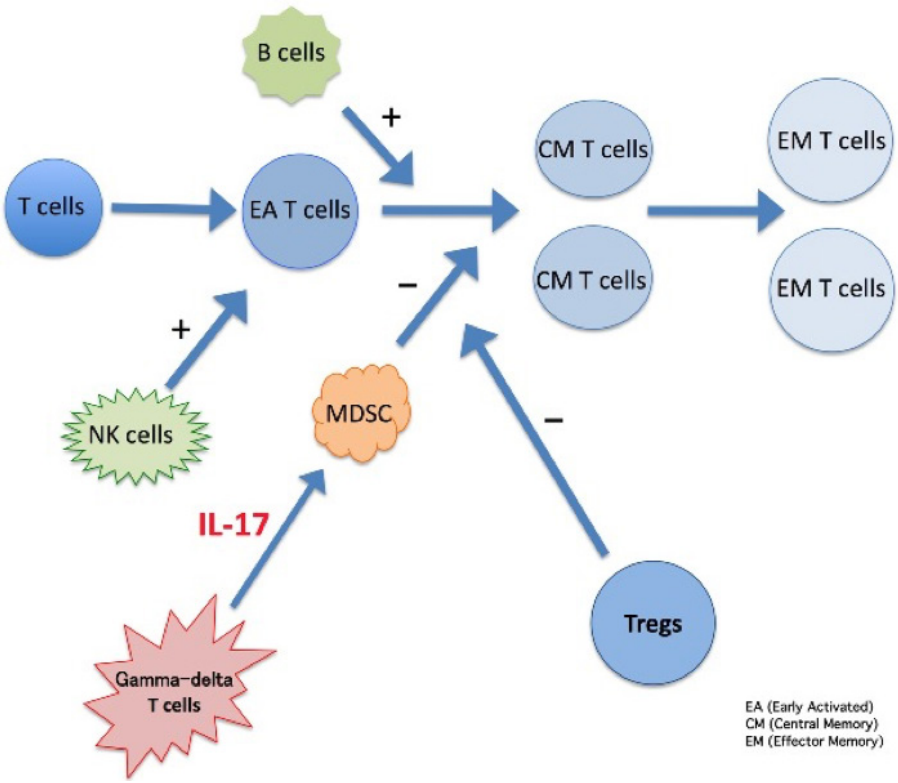
Microscopic Chagas Disease [2]

1. Immunity and immune modulation in Trypanosoma cruzi infection. Cardillo et al., Pathog Dis. 2015 Oct 5
2. https://www.cdc.gov/dotw/chagas/images/main_928px.png

Natural killer cells may be the major cell type responsible for IFN- production in the early stages of T. cruzi infection and their activation requires the presence of live parasites (Cardillo et al. 1996).

In addition, innate or adaptive immune cells, such as dendritic cells, macrophages, NKT lymphocytes, T cells and B cells, may contribute to host resistance (Locksley and Scott 1991; Sher et al. 1993; Cardillo et al. 1996; Galli et al. 2003, 2007; Takahashi and Strober 2008).

B lymphocytes are also required to mount an effective immune response to T. cruzi, helping in the control of the infection (Cardillo et al. 2007; Sullivan et al. 2015)



Immunity and immune modulation in Trypanosoma cruzi infection. Cardillo et al., Pathog Dis. 2015 Oct 5

Possible Causes and Clinical Signs & Symptoms

Chagas disease is transmitted through various routes, but primarily via triatomine insects, also known as kissing bugs.

The T. cruzi parasite is found in the hindguts of a variety of species of triatomines in North, Central and South America. These insects feed at night on the blood of animals, as well as humans, and have a relatively painless bite. If an infected triatomine defecates while feeding, it can transmit the parasite through its feces.

When the person or animal who has been bitten (usually still sleeping) scratches the bite or rubs the area, they may unintentionally introduce triatomine feces contaminated with T. cruzi into their bloodstream.

The disease can also be transmitted several other ways, such as orally through consumption of food or drink contaminated with triatomine feces, from infected mothers to their infants during pregnancy, blood transfusions and organ transplantations, laboratory accidents and sharing of syringes.

Acute Phase

The acute phase, which begins several days after infection, is usually asymptomatic or with mild flu-like symptoms and lasts about 8-10 weeks.

Chronic Phase

After the acute phase, the person enters into the chronic phase of the disease.

There are 2 possible chronic disease phases, the indeterminant (or asymptomatic) phase and the determinant (or symptomatic) phase.

Asymptomatic

About 70-80% of people will remain asymptomatic for life and never develop Chagas-related symptoms.

Late Symptomatic Phase

However, an estimated 20–30% of infected people will develop health problems years to decades later that are often fatal.

The most common symptoms in this late symptomatic stage are cardiac and include conduction abnormalities (arrhythmias), heart failure and sudden death.

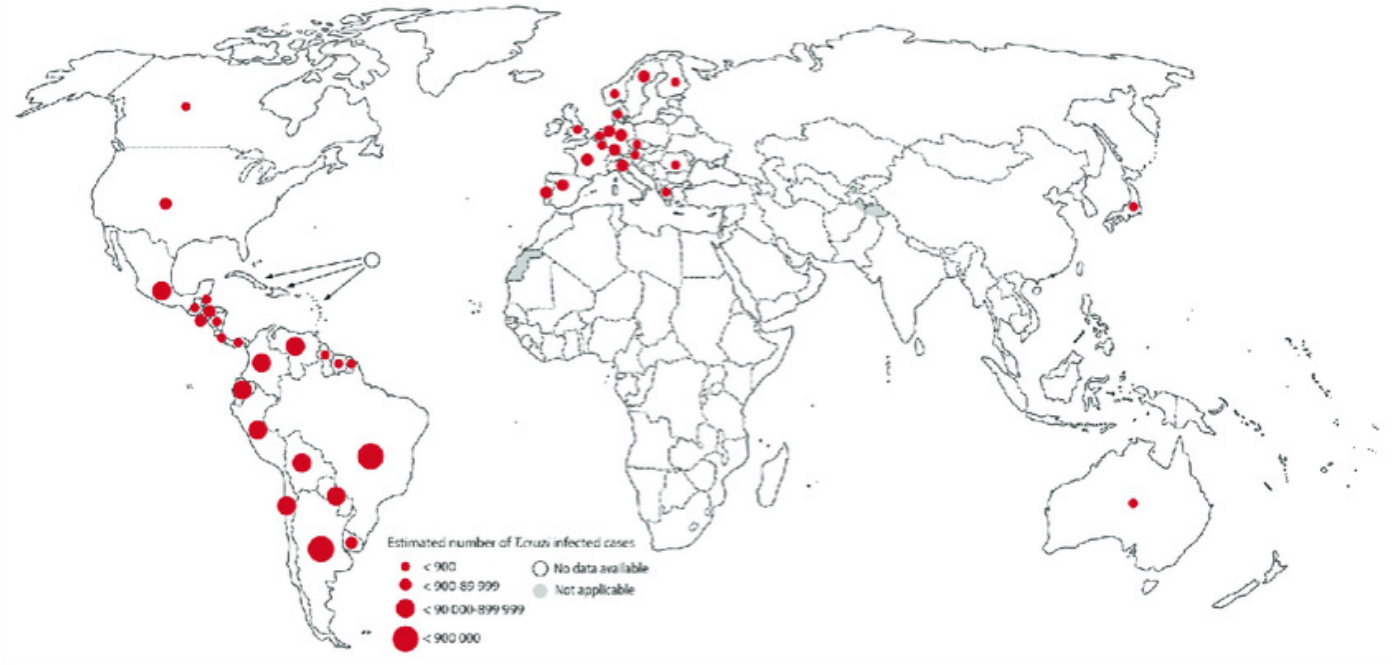
The chronic form of the disease mainly affects the peripheral autonomous nervous system in the gastrointestinal tract and heart and the heart muscle in approximately 30% of the infected patients (Koberle 1968; Andrade, Gollob and Dutra 2014).

Serological Analysis of New York City COVID19 Convalescent Plasma Donors. Larry L. Luchsinger PhD. New York Blood Center

Incidence & Prevalence

- T. cruzi is endemic in vectors and wildlife reservoirs throughout the Americas from the southern half of the United States down to Argentina. Chagas disease cases have been reported from South and Central American countries, particularly in rural, impoverished areas. There have been a small number of autochthonous cases of Chagas disease in the United States. [1]

- Today, there are an estimated 8 million people living with Chagas disease globally, and more than 10,000 deaths per year can be attributed to this significant public health problem. [2]



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1. https://www.cdc.gov/dpdx/trypanosomiasisamerican/index.html#:~:text=Trypanosoma%20cruzi%20is%20transmitted%20by,wound%20by%20the%20host%20scratching.
2. https://asm.org/Articles/2021/April/Chagas-Disease-in-the-U-S-What-We-Know-About-the-K
3. https://www.researchgate.net/figure/Global-distribution-of-Chagas-disease-cases-based-on-official-estimates-2006-2015-9_fig3_340283917

HTLV

Overview

HTLV

The Human T-Lymphotropic virus type 1 (HTLV-1) was the first oncogenic human retrovirus to be discovered. It was first studied in 1977. The virus can cause adult T-cell leukaemia/lymphoma (ATL) and progressive nervous system condition known as HTLV-1-associated myelopathy or tropical spastic paraparesis (HAM/TSP). [1]

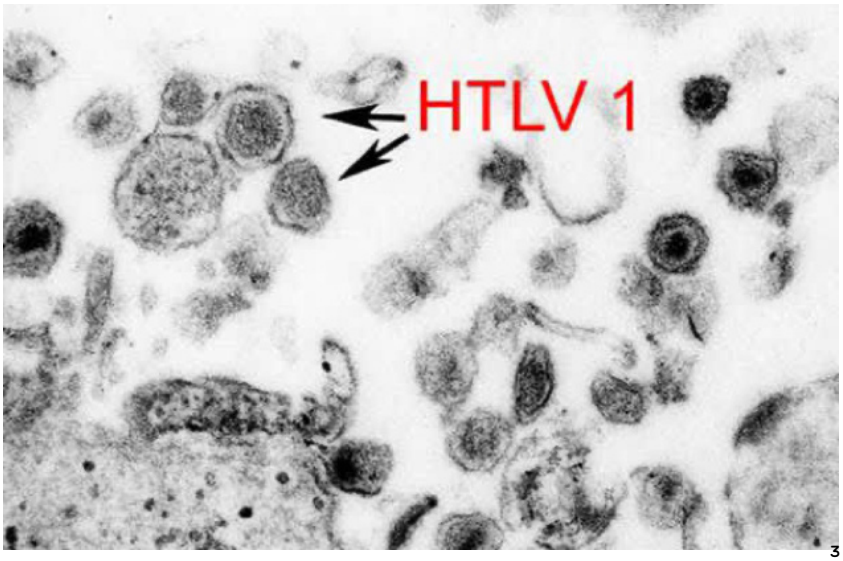
It is classified as a complex type C retrovirus belonging to the genus Deltaretrovirus, family Retroviridae, and subfamily Orthoretrovirinae. [2]

Originally acquired through interspecies transmission from infected monkeys in the Old World.
To date, four types of HTLVs have been discovered. HTLV I is the most prevalent. [1]

HTLV-1 possesses a remarkable genetic stability, an unusual feature for a retrovirus. [1]

HTLV-1 is the etiological agent of two severe diseases, which
are relatively frequent in the main HTLV-1-endemic areas: a malignant T CD4+ cell lymphoproliferation, of very poor prognosis, known as adult T-cell leukaemia/lymphoma (ATLL) and a severe chronic neuro-myelopathy named Tropical Spastic Paraparesis/HTLV-1-associated myelopathy (TSP/HAM). [1]

HTLV-1 is present throughout the world with clusters of high endemicity in southern Japan, the Caribbean region, areas of South America and tropical Africa and foci in the Middle East, Australia and Melanesia. [1]



[3]

1. https://www.ecdc.europa.eu/sites/default/files/media/en/publications/Publications/geographical-distribution-areas-high-prevalence-HTLV1
2. HTLV-1, Immune Response and Autoimmunity. Quaresma et al., Viruses. 2016 Jan; 8(1): 5.
3. https://www.thebodypro.com/article/hivs-largely-ignored-relative-htlv-1-has-prevalenc

Immunology

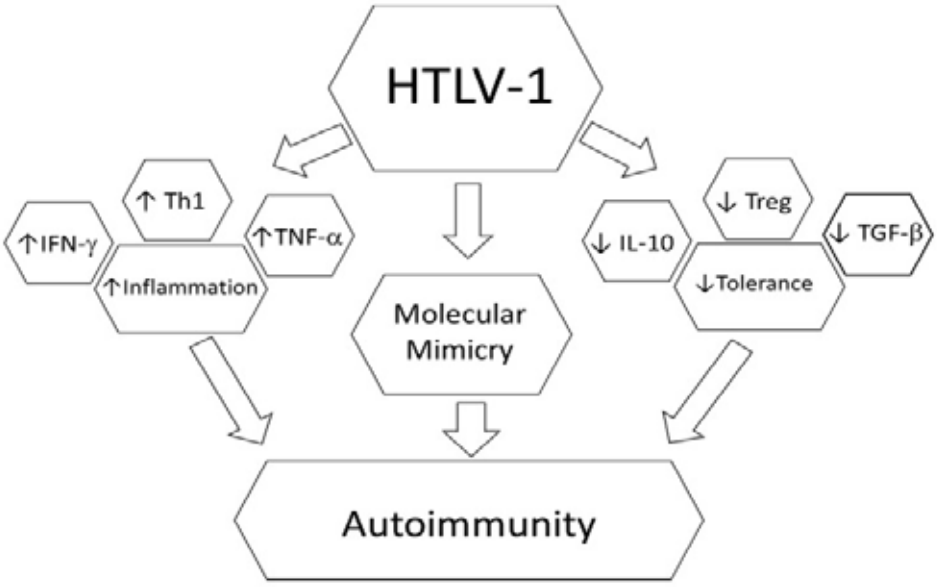
The HTLV-1 virus infects CD4+ T lymphocytes, and can modify the cell function. CD4+ T lymphocytes are the central acquired immune response regulators. Changes in their behavior can trigger inflammatory reactions that can break immune system tolerance, leading to autoimmunity.

HTLV-1-infected CD4+ T lymphocytes exhibit altered signaling cascades and transcription factor activation, leading to changes in cell behavior.

HTLV-1 infection leads to changes in the systemic immune response even in asymptomatic patients.

During the development of autoimmunity, there is a loss of tolerance to self-antigens, causing an inflammatory response that attacks organs and tissues of the individual.

The association between autoimmunity and HTLV-1 infection has been previously described; however, the mechanisms underlying this association are not yet fully understood. Many studies have indicated that molecular mimicry could be the trigger for the development of certain diseases. However, as previously described, HTLV-1 can result in several immune response anomalies since it infects CD4+ T lymphocytes and alters their behavior.



Possible mechanisms involved in HTLV-1 association with autoimmunity.

HTLV-1, Immune Response and Autoimmunity. Quaresma et al., Viruses. 2016 Jan; 8(1): 5.

Possible Causes and Clinical Signs & Symptoms

HTLV-1 has three modes of transmission:

- Mother-to-child, mainly linked to prolonged breast-feeding.
- Sexual, mainly occurring from male to female,
- Via transplantation of organs, tissues and leucocyte-rich blood components.

Several serious diseases are thought to be caused by or strongly associated with the virus. The lifetime risk of developing adult T-cell leukaemia/lymphoma (ATL) among people with an HTLV-1 infection is about 5% (although this may be conservative due to unreported cases).

ATL presents as four clinical subtypes: acute, lymphomatous, chronic and smouldering, with the more aggressive subtypes (acute and lymphomatous) representing the majority of cases.

Clinical presentation depends on the subtype. People may present with lymphadenopathy, hepatosplenomegaly, hypercalcaemia through involvement of the skin, lung, bones and other organs.

Another disease is HTLV-1-associated myelopathy or tropical spastic paraparesis (HAM/TSP). This is a chronic inflammatory disease of the central nervous system, characterized by progressive spastic weakness of the lower limbs, lower back pain and bowel and bladder dysfunction.

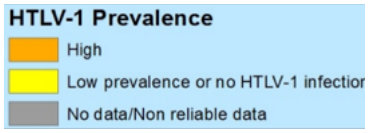
Clinical findings can include muscle weakness, hyperreflexia and clonus in the lower limbs, along with extensor plantar responsive and a spastic gait. Estimates of the lifetime risk of HAM/TSP among people with HTLV-1 infection have ranged from 0.18% to 1.8%.

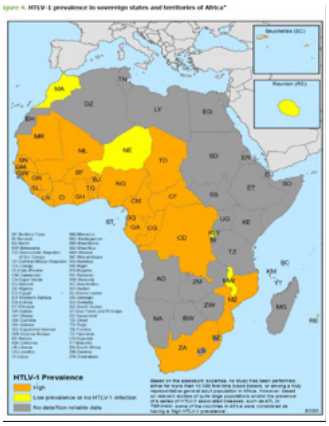
Other diseases connected to HTLV-1 infection include HTLV-1-associated uveitis (HAU), infective dermatitis, bronchiectasis, bronchitis and bronchiolitis, seborrheic dermatitis, Sjögren's syndrome, rheumatoid arthritis, fibromyalgia and ulcerative colitis. There is little evidence that HTLV-1 infections cause other forms of cancer.

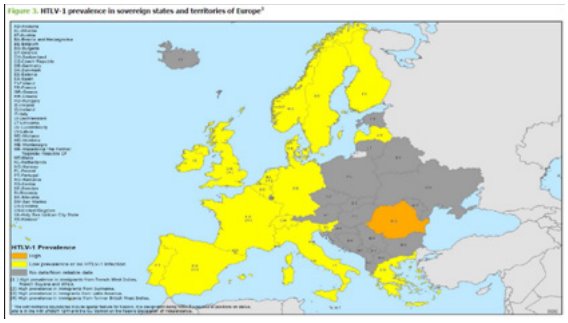
https://www.who.int/news-room/fact-sheets/detail/human-t-lymphotropic-virus-type-1

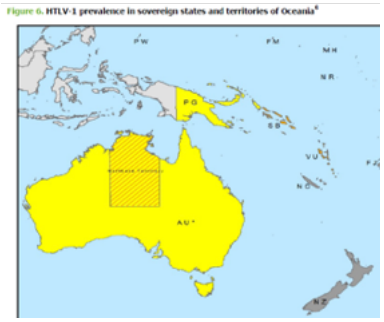
Incidence & Prevalence

- High prevalence areas are in South America, Africa and parts of the Middle East.
- The current estimates for the total number of people living with HTLV-1 infection range from 5 million to 10 million as of 2012.
- The prevalence of the infection is particularly high in parts of Japan, Australia and the Pacific islands.







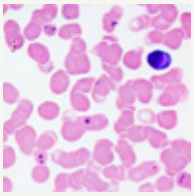


Geographical distribution of areas with high prevalence HTLV-infection

Other Emerging Diseases

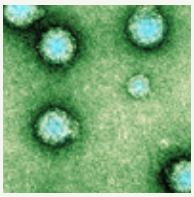
Malaria, Chikungunya, Zika

OTHER EMERGING DISEASES



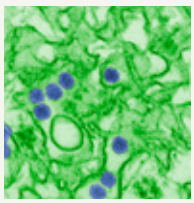
MALARIA

Malaria is a serious and sometimes fatal disease caused by a parasite that commonly infects a certain type of mosquito which feeds on humans. People who get malaria are typically very sick with high fevers, shaking chills, and flu-like illness. About 1,700 cases of malaria are diagnosed in the United States each year. The vast majority of cases in the United States are in travelers and immigrants returning from countries where malaria transmission occurs, many from sub-Saharan Africa and South Asia.



CHIKUNGUNYA VIRUS

Chikungunya Virus (ChikV) is an arbovirus spread to humans from mosquitos. ChikV outbreaks have occurred in Africa, Asia, Europe, the Indian and Pacific Oceans, and the Caribbean. No ChikV outbreaks have been reported in the United States. Symptoms include fever and joint pain, and there is no vaccine or medicine to prevent or treat ChikV



ZIKA VIRUS

Zika Virus (ZIKV) is a mosquito-borne arbovirus spread by the Aedes species mosquito. ZIKV can be passed from a pregnant woman to her fetus, and infection during pregnancy can lead to serious birth defects. Symptoms of Zika include fever, rash, headache, joint pain, red eyes, and muscle pain. Transfusion transmitted cases of ZIKV have been reported

https://www.cdc.gov/bloodsafety/bbp/diseases-organisms.html#:~:text=Transfusion%2Dtransmitted%20infections%20(TTIs),be%20transmitted%20through%20blood%20transfusions.

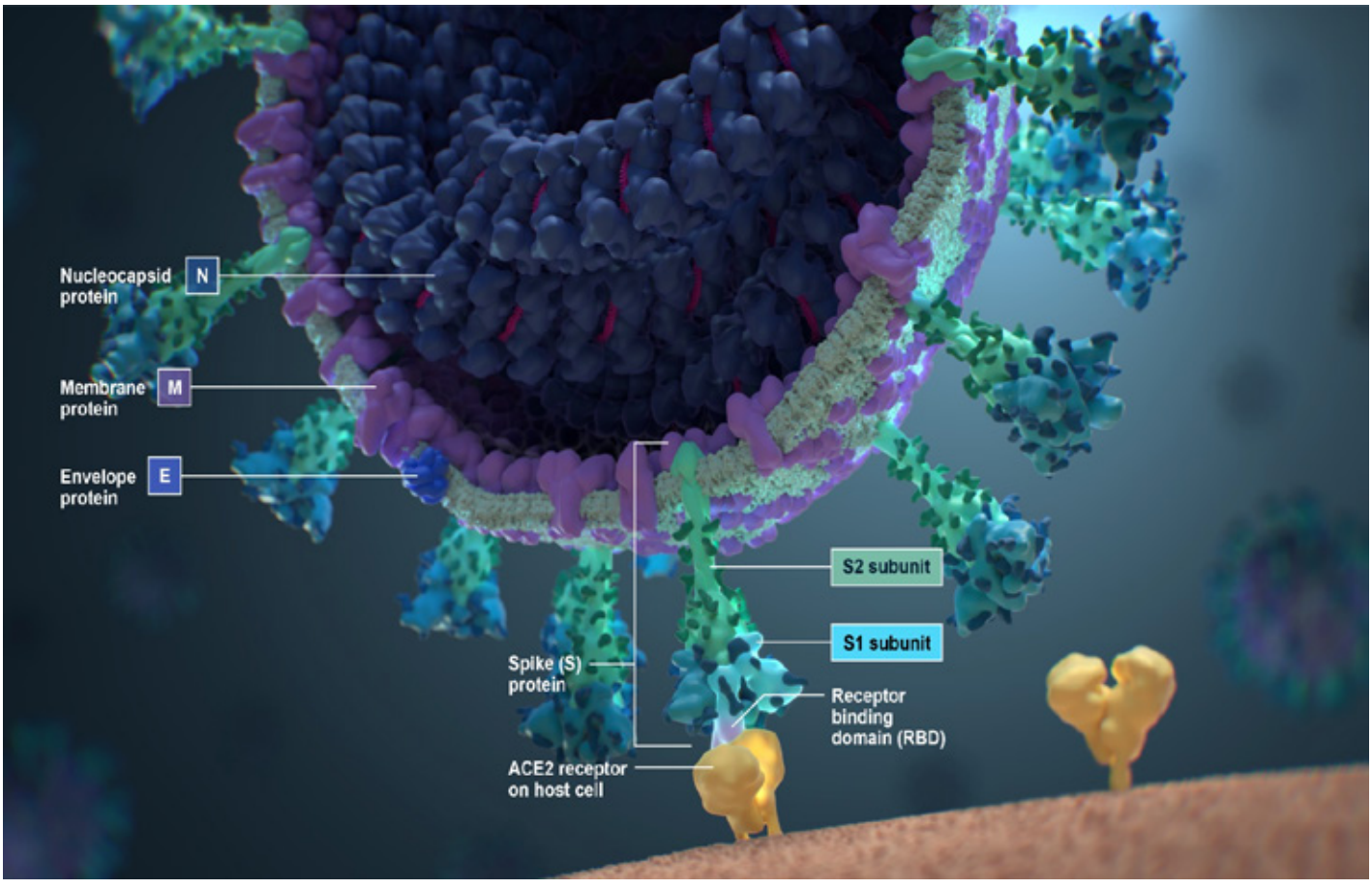
COVID-19 Overview

COVID-19 was identified in December 2019. It is caused by the virus severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a new virus in humans causing respiratory illness which can be spread from person-to-person. [1]

Coronaviruses are spherical and enveloped with club-shaped spikes on the surface. The viral genome contains four major structural proteins: the spike (S), membrane (M), envelope (E) and the nucleocapsid (N) protein. [1]

> Globally, as of 5 August 2022, there have been **579,092,623 confirmed cases** of COVID-19, including **6,407,556 deaths**, reported to WHO.

NOTE: The FDA has said "The potential for transmission of SARS-CoV-2 by blood and blood components is unknown at this time. However, respiratory viruses, in general, are not known to be transmitted by blood transfusion, and there have been no reported cases of transfusion-transmitted coronavirus.". [2]



1. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8919811
2. Is SARS-COV-2 Transfusion Transmitted. Katz. 2022

COVID-19 Testing Solutions

ANTIGEN TEST

Provides accurate and reliable results in 10 minutes, allowing for COVID-19 testing of symptomatic patients within the first five days of symptoms and asymptomatic patients when tested serially.

ANTI-SARS-COV-2 IGG QUANTITATIVE TEST [1]

A standardized quantitative antibody assay calibrated to the WHO standard,* can play a crucial role in understanding the future with COVID-19.

Antibodies to the spike protein have been shown to be highly specific to SARS-CoV-2 and the highest concordance to neutralizing antibodies. [2] [3]
Provide numerical values calibrated to the WHO standard* making it a key test to help clinicians determine the level of antibody response to the SARS-CoV-2 virus and its persistence over time

1. https://www.orthoclinicaldiagnostics.com/global/covid19/antibody-test
2. SARS-CoV-2 specific antibody responses in COVID-19 patients Nisreen et all
3. In Vitro Diagnostic Assays for COVID-19: Recent Advances and Emerging Trends, Vashist, MDPI Editorial, 5 April 2020
* WHO International Standard Anti-SARS-CoV-2 Immunoglobulin (Human) NIBSC 20136

Vitros® COVID-19 Antibody Tests

To help identify recovering patients who could potentially be serum and plasma donors for CCP

VITROS®
Anti-SARS-CoV-2 Total Test
Qualitatively Measures
Total Antibodies
to the SARS-CoV-2 virus.

VITROS®
Anti-SARS-CoV-2 IgG Test
Qualitatively Measures
IgG Antibodies
to the SARS-CoV-2 virus.

Key Performance Characteristics to optimize positive predictive value [1] [2] [3]

- 100% Specificity providing 100% PPV at ANY disease prevalence
- Targets S1 "Spike" Protein – antibodies measured by the test include neutralizing antibodies
- No cross-reactivity shown with samples negative to SARS-CoV-2
- No Biotin interference
- Workflow advantages of random-access testing, continuous sample loading and STAT sample option.

KEY SYSTEM TECHNOLOGIES


Micro Well


Intellicheck® Technology


Versa Tip


Micro Sensor

1. VITROS® Anti-SARS-CoV-2 Total Reagent Pack and Calibrators Instructions For Use (code 6199922 – vs 1.0)
2. VITROS® Anti-SARS-CoV-2 IgG Reagent Pack and Calibrators Instructions For Use (code 6199919 – vs 1.0)
3. SARS-CoV-2 specific antibody responses in COVID-19 patients Nisreen et all

The Donor Screening Lab

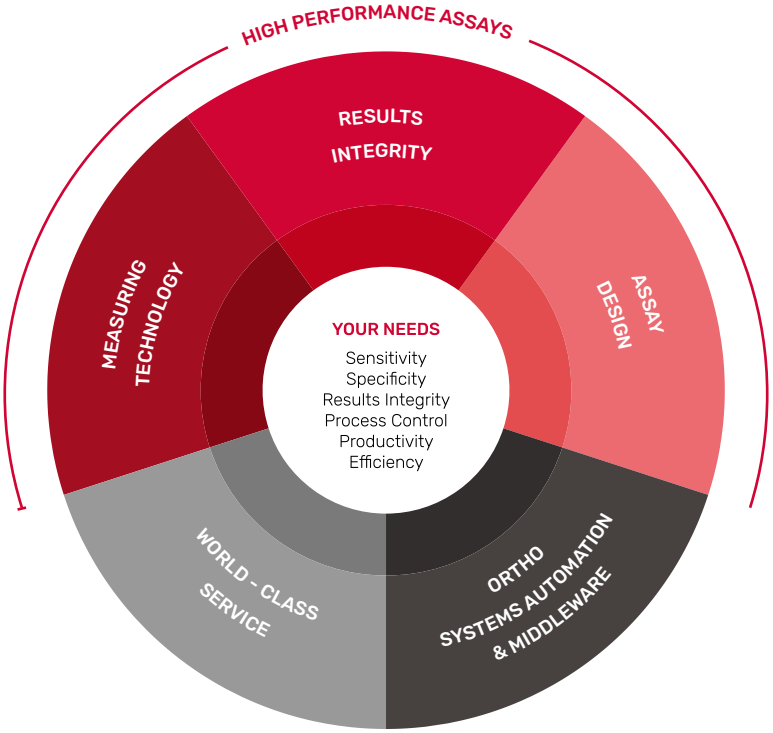
The Donor Screening Lab Needs

Enabling access to safe blood products for patients, every day.

- Minimize the rejection of blood and blood products.

The Challenge

Fluctuating activity levels throughout the day create operational complexity. And declining donor numbers amplify the importance of minimizing the rejection of blood and blood products. With sample quality and integrity determining the usability of results, and the increasing complexity of sample management and algorithms for confirmatory testing of new threats, Lab instruments must work with both consistency and absolute precision to overcome these challenges.



Ortho Clinical Diagnostics

Because **Every** Test is a **Life**™

To learn more about our business and our commitments, please visit:
www.orthoclinicaldiagnostics.com

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