# THE CARDIOVASCULAR EFFECTS OF THE 2019 SPREAD OF SARS-COV-2 (COVID-19) IN LATIN COMMUNITIES

By

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**Abstract**

The Coronavirus disease 2019 (COVID-19), caused by the worldwide spread of the SARS-CoV-2 virus continues to cause significant morbidity and mortality. The prevalence of cardiac injury and vascular dysfunction in positive COVID-19 patients is concerning due to the potential long-term cardiovascular effects of the infection. This is especially concerning in the Latino community due to higher infection and death rates of COVID-19. Understanding the pathophysiology of the infection, how the immune system attacks the virus as well as the cardiovascular system involvement, it is clear that comorbidities intensify the severity of the disease and may lead to hypertension, myocarditis, heart failure, and arrythmias, among others. For Latin communities, pre-existing medical conditions and the lack of access to appropriate healthcare are the main reasons for the disproportionate disparities since Latinos have a high incidence of cardiovascular disease as well as many not having health insurance. Due to the fact that the pandemic is ongoing, it's impossible to really understand the long-term effects of COVID-19, on any population. However, understanding the mechanisms (as we know them currently) can help with education and health choices, for the general population, and Latin communities in particular.

## Introduction

The novel coronavirus disease that emerged in 2019 commonly known as COVID-19 is an infectious viral disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1] It first emerged in Wuhan, China in December of 2019 and spread across the world creating one of the most chaotic pandemics in recent history. Due to it being a new strain, humans have no known immunity to the virus, so it started spreading within a couple of weeks and is currently associated with over 147 million cases and about 3 million deaths.[2] Although COVID-19 is known to infect the respiratory-tract and haves major effects on the lungs, it has also been shown to cause significant damage to the cardiovascular system.[1] More recently, as individuals who have had COVID-19 have started to recover, it has been observed that there are long term complications associated with this disease.[3] Latino populations seem to be vulnerable to the virus. This paper will investigate the higher rates of cardiovascular effects of the 2019-2021 Spread of SARS-CoV-2 in Latin Communities. To understand how the disease attacks the body and how the body reacts to the virus it is helpful to understand the physiology of the immune and cardiovascular systems.

## The Immune System

The immune system is a complex network of cells and organs that defend the human body from pathogens. The main purpose of this biological defense system is to combat infections by identifying pathogens, viruses, fungi, parasites, bacteria, and most importantly being able to identify self from foreign cells in the body.[4] The immune system is composed of white blood cells, antibodies, the lymphatic system, spleen, bone marrow and the thymus.[4] There are two branches of the immune system: the

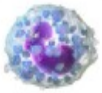
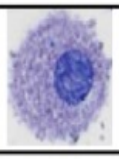
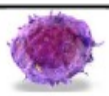
innate and the adaptive systems. The innate immune system is the first line of defense that provides a rapid but non-specific response to pathogens.[4] The adaptive immune system acts as the second line of defense to provide a specific response to the pathogen presented.[4] This system takes longer to get started than the innate system, but it may lead to long lasting immunity to a pathogen meaning that they develop memory.

## Innate immune system

The main goal of the innate immune system is to immediately prevent the spread of pathogens in the human body by recruiting immune cells to the site of infection and cause inflammation. This system arranges to inactivate, destroy, and remove intruders from our body.[5] It involves a variety of different cells found in the blood that have different functions and characteristics. Blood cells include erythrocytes (which are red blood cells); leukocytes, which are nucleated cells known are white blood cells and include mononuclear cells including monocytes, macrophages, and lymphocytes; polymorphonuclear cells which have a lobulated nucleus including the eosinophils, basophils, and neutrophils.[6] Macrophages levels vary in adults and they function as phagocytes that engulf pathogens and kills them through different pathways. Macrophages are also antigen presenting cells, meaning they are able to help cells of the adaptive immune system (discussed next). Neutrophils constitute about 40-75% of white blood cells and have the same phagocytosis function as macrophages.[7] Eosinophils, about 1-6% of cells, function to kill large antibody-coated parasites by degranulation, and release of histamine, enzymes and cytokines. [7] Basophils (less than 1%) are circulatory cells that are important for inflammatory responses. [7] Another type of

cell, mast cells, also produce cytokines. Eosinophils, basophils, and mast cells all play an important role in asthma and allergy responses. Natural killer cells are also very important for the innate system and levels usually varies in humans. They focus on rejecting tumors, killing of infected cells, and are also an important source of cytokines. [7] Figure 1 lists the cells of the immune system.

**Figure 1:** Summarizes the characteristics, levels, and functions of the cells that are involved in the innate immune system. [7]

| Cell | Image | % in adults | Nucleus | Functions | Lifetime | Main targets |
|---|---|---|---|---|---|---|
| Macrophage* |  | Varies | Varies | • Phagocytosis<br>• Antigen presentation to T cells | Months – years | • Various |
| Neutrophil |  | 40-75% | Multi-lobed | • Phagocytosis<br>• Degranulation (discharge of contents of a cell) | 6 hours – few days | • Bacteria<br>• Fungi |
| Eosinophil |  | 1-6% | Bi-lobed | • Degranulation<br>• Release of enzymes, growth factors, cytokines | 8-12 days (circulate for 4-5 hours) | • Parasites<br>• Various allergic tissues |
| Basophil |  | < 1% | Bi- or tri-lobed | • Degranulation<br>• Release of histamine, enzymes, cytokines | Lifetime uncertain; likely a few hours – few days | • Various allergic tissues |
| Mast cell |  | Common in tissues | Central, single-lobed | • Degranulation<br>• Release of histamine, enzymes, cytokines | Months to years | • Parasites<br>• Various allergic tissues |
| Lymphocytes (T cells) |  | 20-40% | Deeply staining, eccentric | T helper (Th) cells (CD4+): immune response mediators<br><br>Cytotoxic T cells (CD8+): cell destruction | Weeks to years | • Th cells: intracellular bacteria<br>• Cytotoxic T cells: virus infected and tumour cells<br>• Natural killer cells: virus-infected and tumour cells |
| Monocyte |  | 2-6% | Kidney shaped | Differentiate into macrophages and dendritic cells to elicit an immune response | Hours – days | • Various |
| Natural killer (NK) cell |  | 15% (varies) of circulating lymphocytes and tissues | Single-lobed | • Tumour rejection<br>• Destruction of infected cells<br>• Release of perforin and granzymes which induce apoptosis | 7-10 days | • Viruses<br>• Tumour cells |

It's possible to break the innate immune system into four types of defensive barriers with non-specific host-defense mechanisms for each (Figure 2). These mechanisms make the innate system effective in fighting off invaders. The innate system focusses on recruiting cells to the site of infection through the production of cytokines and chemokines, such as tumor necrosis factor (TNF), interleukin 1 (IL-1) and interleukin 6 (IL-6). [7] Cytokines are crucial for recruitment, they mobilize defense mechanisms, activate cellular responses, elicit inflammation, and lead to temperature elevation resulting in fever. [7]

**Figure 2:** Describes the mechanisms for specific barriers of the innate immune system. [7]

| Barrier | Specific | Mechanism |
|---|---|---|
| **Anatomic** | Skin | Serves as the mechanical barrier that fights the entry of microbes. |
| | Mucous membrane | Normal flora, mucous, and cilia help compete, entrap and propel microbes. |
| **Physiological** | Temperature | Temperature adjustment inhibits growth of pathogens. |
| | Low pH | Kills most undigested microbes. |
| | Chemical mediators | • Lysozyme – cleaves bacterial cell wall.<br>• Interferon – antiviral defenses in uninfected cells.<br>• Complement – lyses microbes or facilitates phagocytosis. |
| **Phagocytic/endocytic** | N/A | • Endocytosis – breakdown of foreign molecules.<br>• Phagocytosis – kill and digest organisms |
| **Inflammatory** | N/A | Damage elicits antibacterial activity and influx of phagocytic cells into the area. |

The innate system relies on pattern recognition receptors (PRRs) to recognize pathogen-associated molecular patterns (PAMP), damage-associated molecular patterns (DAMP) and the absence of certain normal cell surface molecules. [5] PRRs are highly expressed in antigen presenting cells, such as macrophages and dendritic cells

(DC), but are present on most cells in the body, and are divided into four groups: Toll-Like Receptors (TLR), Nucleotide-Binding Oligomerization Domain-Like Receptors (NLR), C-type Lectin Receptors (CLR), RIG-1 Like Receptors (RLR).[8] TLRs bind to DAMPs that are released by stressed cells in the body causing a cascade of reactions that lead to inflammation.[5] Dendritic cells (DCs), which are phagocytic cells in the skin, lungs, and mucous membranes, get activated and ingest the antigen in the periphery, carry it to a lymph-node, and presents the antigen to lymphocytes. This is where the adaptive immune response comes in.

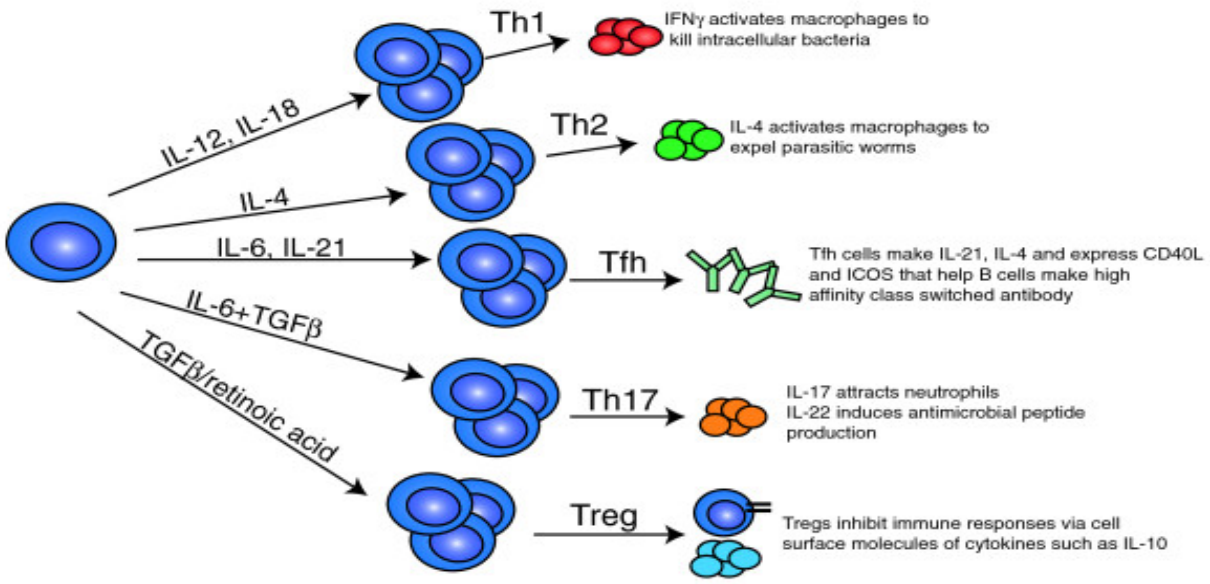
### Adaptive Immune System

The adaptive immune system as the second line of defense aids the human immune system when the innate immunity is unable to completely fix the problem. Its functions are to recognize antigens, distinguish foreign antigens from “self”, generate the pathways to eliminate the pathogens, and to create immunologic memory for future use as immunization against infectious diseases.[7] It involves two kinds of cells: lymphocytes and phagocytes. Lymphocytes are specialized to recognize foreign or damaged cells and phagocytes, as mentioned before, serve to kill pathogens by eating and digestion.[5] For lymphocytes to recognize an antigen, the antigen’s epitope (a small part of the larger antigen) must fit into the lymphocyte’s receptors. Lymphocytes are not created after exposure to the antigen (we already have millions of lymphocytes with different receptors), rather, the antigen bumps into lymphocytes until they find a receptor that they fit into.[5] There are two kinds of lymphocytes: T cells and B cells.

T cells are proliferating cells that contain antigen-binding receptors called T-cell receptor (TCR) and are produced in the bone marrow and mature in the thymus.[7] They

require antigen-presenting cells (APCs) to “show” the T cell what they’ve have either eaten or made. APCs express the major histocompatibility complex (MHC) which is has two classes: MCH class I or MCH class II. MHC class I are found on all nucleated cells and they present intracellular molecules.[7] MHC class II is found on macrophages, dendritic cells and B cells, and its job is to present extracellular peptides.[7] Different types of T cells can recognize MHC class 1 (these are called Cytotoxic T cells (CTLs) and have a marker called CD8+) and others can recognize MHC class II and are called Helper T cells (Th) and have a marker called CD4+). This all takes place in a lymph node. When T cells encounter APCs that have digested an antigen and is able to recognize it in conjunction with MHC, the T cell gets activated, it proliferates and some of these cells travel around the body to find the source of the antigen.[5] Figure 3 shows the differentiation of different forms of Th cells. Each type has a slightly different role in the body.

**Figure 3:** T-helper cell types and function[9]



B cells are immune cells that can recognize antigen directly.[5] They are produced in the bone marrow and once mature they express a unique antigen-receptor and move to a lymph node. Their main function is to create antibody to fight infections in the human body. Once activated, they go through proliferation and some will differentiate into memory B cells. Activated B cells, called plasma cells, can eliminate an antigen before infection by quickly producing antibodies that protect the body. The five major types of antibodies are: IgA, IgD, IgE, IgG and IgM. IgA levels in the body are 200mg/dL and has a mucosal response meaning that it protects mucosal surfaces from pathogens by binding of the antigen and not allowing it to enter the body.[5,7] There is only about 5mg/dL of IgD which functions as the B cells receptor, although plasma IgD has no known function.[5,7] An adult has about 0.02 mg/dL of IgE in their serum which in involved in hypersensitivity and allergic reactions.[5,7] An adult has about 1000mg/dL of IgG in their serum and is the main antibody in secondary response.[5,7] It is the only antibody that can cross the placenta, neutralizes pathogens, and can activate complement.[5,7] Lastly, IgM levels are of 100mg/dL and works by activating complement and coating antigen for destruction.[5,7]

## Infection of Cells

A virus attacks the immune system by invading the hosts cells and replicating in order to survive. When referring to viruses specifically there are many different immune responses that the body can generate to attack the pathogen, there is a cell-mediated response and an antibody-mediated response.

Starting with cell-mediated responses, one way is via Cytotoxic T cells (CTL) which can kill infected cells directly. An infected cell can show what it has been making

(either normal cell protein, or virus) on MHC class I. The TCRs in the cytotoxic cell binds to the combination of MHC class I and antigen. After the virally infected cell is identified, the cytotoxic cell will release perforin which will make tiny pores in the cell membrane, allowing granzyme to enter leading to apoptosis (a very organized form of cell death).[7] There are some instances where viruses inhibit MHC class I, so other cells such as Natural Killer cells (NK) must take over.[7] The body also attacks viruses via interferons which are proteins released by infected cells. These proteins serve to prevent replication and to warn nearby cells of the infected cell to prepare for an immune response.

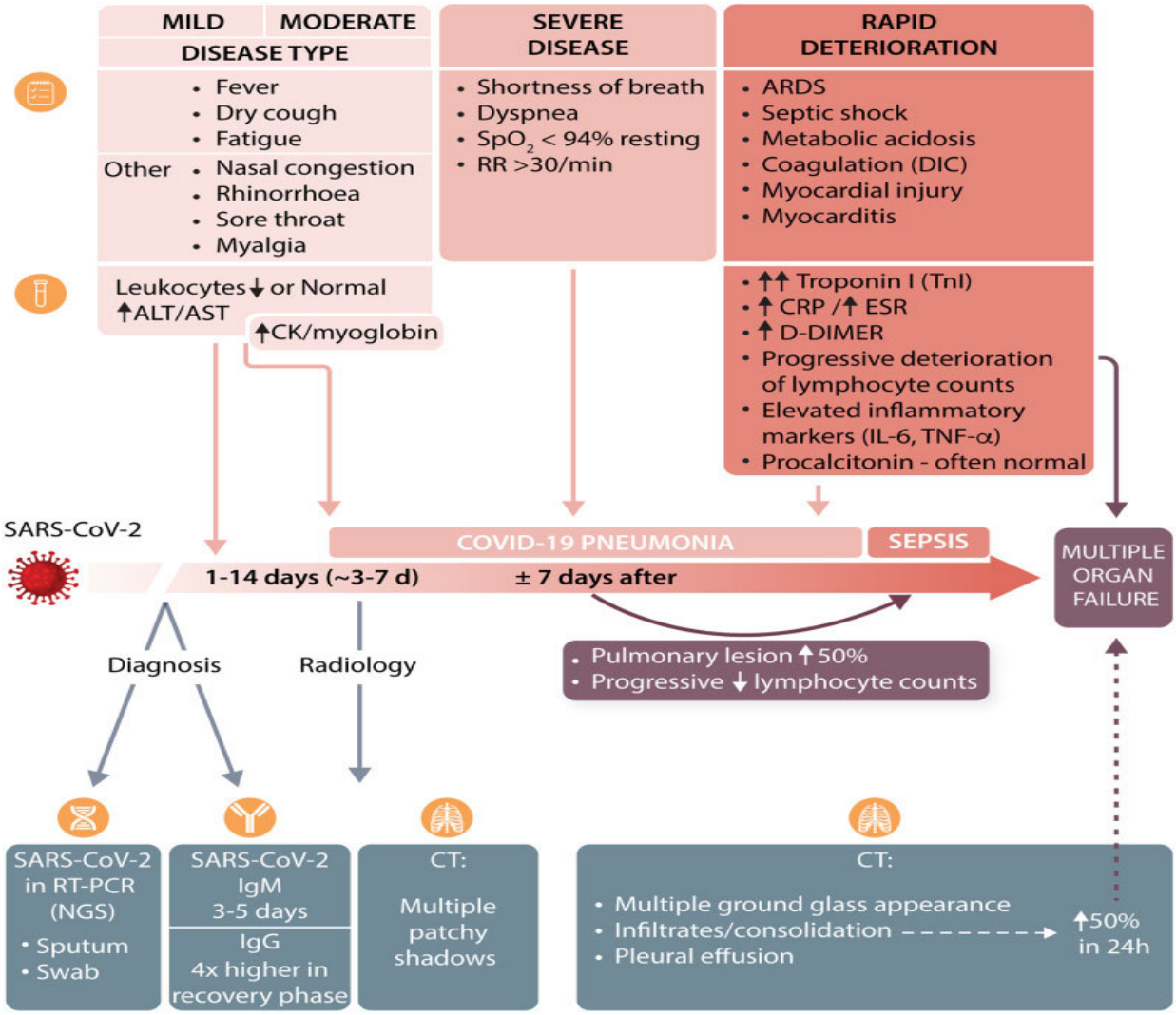
The antibody-mediated response protects the body from viruses by preventing the virus from entering the cell in the first place. Antibodies will bind to pathogens before they infect cells and neutralize them meaning that they can no longer invade other cells.[7] After, antibodies will use agglutination, phagocytosis, or the complement system to eradicate the virus.[5]

**Immune response to COVID-19**

The virus is mainly transmitted person-to-person by droplets expelled by coughing, sneezing, or talking and are absorbed by mucous membranes in the body mostly by inhaling into the nasal system. Once the virus has entered an individual, in infects cells throughout the body by binding to a surface marker called ACE-2.[10] Infections of SARS-CoV-2 can be divided into three stages classified by its severity. Stage I is the asymptomatic incubation of the virus involving the innate system.[10] Stage II is a non-severe symptomatic period which is where the adaptive immune systems gets involved in attempt to neutralize the virus before it progresses.[10] Stage III is when

severe respiratory symptoms are experience and there is a high viral load.[10] If it is not neutralized in stage II, then the infection will spread, leading to inflammation which can cause massive damage to multiple organs and multi-organ failure.[10] Figure 4 displays the course of COVID-19 infection and the complications that it gives rise to.

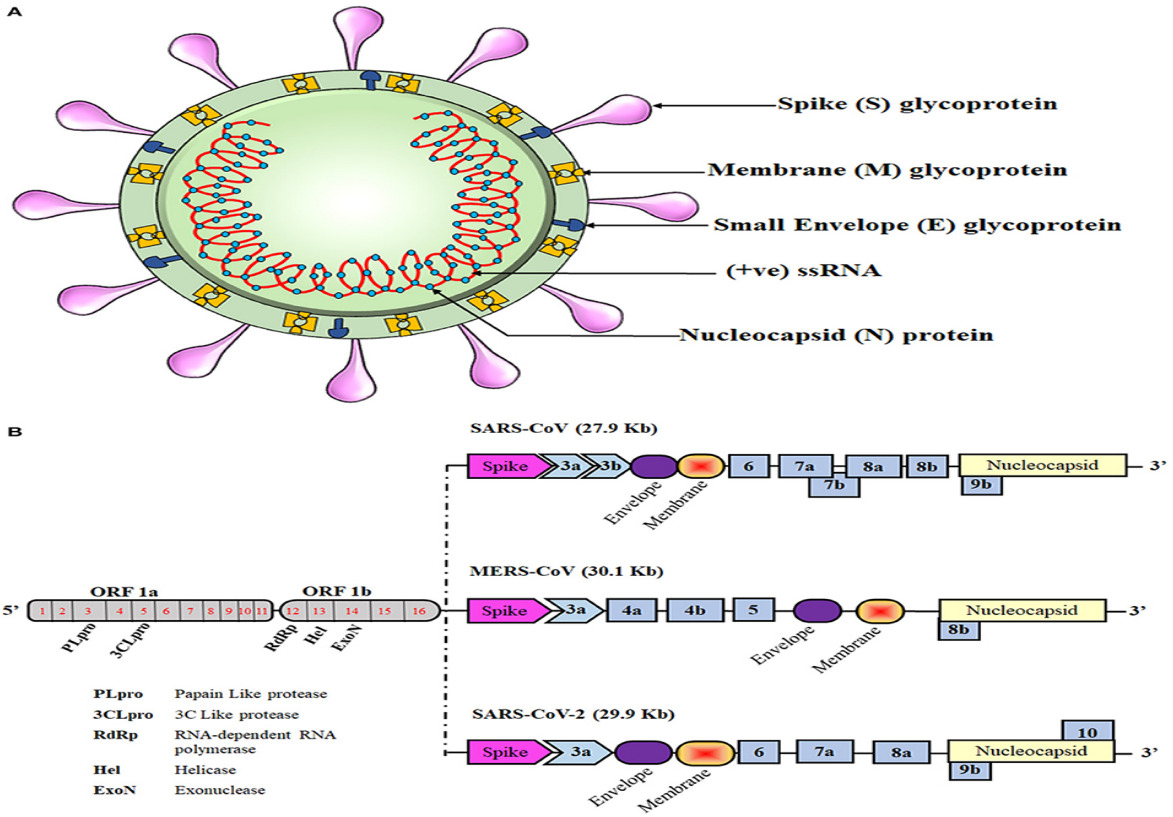
**Figure 4:** Course of infection from the SARS-CoV-2 virus resulting in COVID-19 pneumonia and the complications that arise from the virus leading to multiple organ failure.[1]



## Replication

COVID-19 has a crown-like structure with spike glycoproteins (S), membrane proteins (M), envelope proteins €, nucleoproteins (N), and genomic RNA as shown in in Figure 5.[10] It is a single-stranded RNA virus with a diameter of 60-140nm and spikes ranging from 9-12 nm long.[11] The S protein is essential for the entrance of the virus as it has two domains S1 and S2 which attach and enter the host cell.[12] The domains contain a receptor-bonding domain (RBD) that is recognized by the main receptors angiotensin-converting enzyme 2 (ACE2) or transmembrane protease serine 2 (TMPRSS2).[12] The virus gets inside the cell via membrane fusion, once inside, it releases +ve ssRNA genome into the cytoplasm where translation of ORF-1a and ORF-1b begins.[12] This translation-replication process of the virus will eventually lead to creating more viral RNA polymerase and accessory proteins which will merge with host cell.[12] These new virions will be able to infect other cells and can be released via extremely contagious respiratory droplets into the environment.

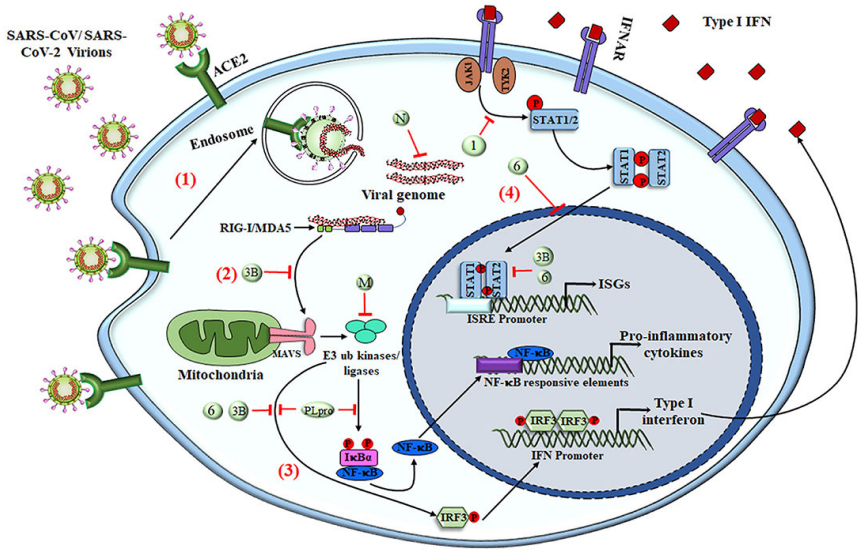
**Figure 5:** Structure of the SARS-CoV-2 virus compared to MERS-CoV and SARS-CoV-2.[1]



## Innate Response

As explained above, the innate system is the first to respond to the viral infection, however it is not yet fully understood but it is thought to be very similar to that of SARS-CoV, which was first described in 2003. The innate response is triggered by epithelial cells in the lungs, neutrophils, and alveolar macrophages when the PRRs recognize AMPs that are released by the host cell that underwent pyroptosis.[13] When the virus binds to ACE2, it acts as a PAMP and is recognized by TLR7, which is expressed on monocytes, macrophages, and DCs, and by RIG-I-like receptor (RIG-1).[14] TLR7 will activate the Janus kinase transducers (JAK/STAT) by expressing Type 1 INF in monocytes and DCs via IFN alpha-beta receptor (IFNAR).[14] A few complexes are formed by JAK1 and TYK2 phosphorylating STAT1 and STAT2 and joining it with IRF-9, to stimulate the transcription of IFN-stimulated genes and the expression of antiviral proteins. [14] Nuclear factor kB (NF-κB), activator protein 1 (AP-1), interferon response factor 3 (IRF3), and IRF7 are also activated by TLR7 and it will translocate to the nucleus. [15] The cascades will increase the secretion of pro-inflammatory cytokines, such as IL-1, IL-6, TNF, which will lead both T and NK cells to further increase production of cytokines, including IL-2, GM-CSF and IFN-γ. [14] The accumulation of the high amounts of pro-inflammatory cytokines results in mobilization of neutrophils, macrophages and T cells into the infected tissue. [14] Although this strong inflammatory response is required to help rid the body of the virus, it's also possible that it may lead to a cytokine storm (overproduction of cytokines), damaging the lungs as well as other organs.[12] The response is illustrated in Figure 6 below.

**Figure 6:** Innate system response to SARS-CoV/SARS-CoV-2 infection.[12]




## Adaptive Response

The adaptive immune response is affected by the innate response since COVID-19 can cause a cytokine storm that promotes the necrosis or apoptosis of T cells, resulting in viral survival and a prolonged infection due to loss of immune cells.[14] However, the adaptive system is still very important in the fight against SARS-CoV-2. The adaptive immune response gets activated by T and B lymphocytes through antigen presentation. APCs present the antigen to CD+4 and CD+8 cells which secrete even more pro-inflammatory cytokines.[14] Antigen-specific T cytotoxic cells will act to kill the cells infected by the virus. CD8+ cells also play a critical role in protecting the body from a lethal infection by the production of cytokines and cytolytic molecules and it will also create memory T cells to further protection form the virus.[14] Moreover, Th17 cells, neutrophils, and granulocytes will secrete IL-17 and stimulate the production of IL-1, IL-6, IL-8, MCP-1, Gro-a, G-CSF, GM-CSF, TNF-α, and PGE2 which will also mobilize immune cells to the site of infection to try to eradicate the virus from the system.[14]

Additionally, T cells, CD4+, and CD8 + T cells will induce T-dependent B cells to secrete antigen-specific antibodies to kill the viral infected cells.[14] The antibodies present are the typical pattern of IgM and IgG. Since IgG is produced at higher quantities and will remain in the blood stream longer than IgM, this indicates that it plays a protective role.[14]

In conclusion, a healthy immune response will use macrophages to rapidly clear infected cells by recognizing and phagocytizing infected cells and by neutralize the virus.[13] T cells will mediate an efficient immune response by eliminating infected cells with minimal inflammation and damage to the body organ systems.[13] On the other hand, a defective immune response will lead to excessive monocytes, macrophages and T cells, resulting in a systemic cytokine storm.[13] Consequently, the disease can cause pulmonary edema and pneumonia and may eventually lead to widespread inflammation and multi-organ damage.[13]

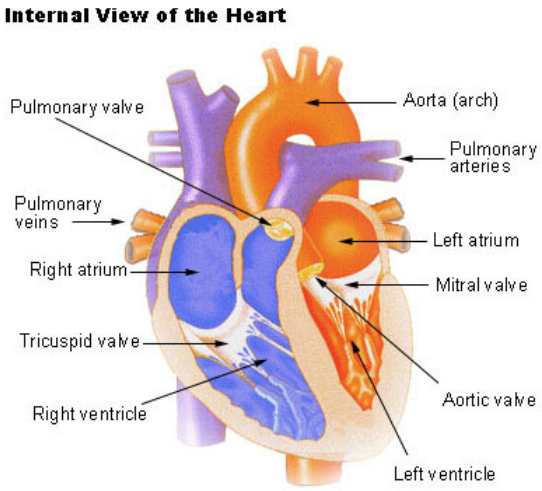
**The Cardiovascular System**

The cardiovascular system is composed of the heart, blood, blood vessels. The heart, which is a muscular pumping device, pumps blood through the circulatory system around the body to maintain homeostasis. Its main job is to deliver oxygen, hormones, nutrients, and other essential substances to cells and organs around the body and serves to remove waste.[16]

A normal adult heart pumps about 5 liters of blood through the body every minute.[16] The heart has 3 layers: the epicardium (outer), the myocardium (middle), and the endocardium (inner). [17] It is composed of four chambers: right atrium, right ventricle,

left atrium, and left ventricle. The two atrial chambers receive blood from the veins, whereas the two ventricles pump the blood out of the heart.[17] In order to keep the fluid flowing in one direction, the heart has both atrioventricular and semilunar valves. The atrioventricular valves, which sit between the atria and ventricles, close to prevent blood from flowing back into the atria when the ventricles contract, and when the heart relaxes, the semilunar valves close to prevent blood from going back into the ventricles.[17] The blood flows from the right atrium to the right ventricle, and then is transported to the lungs to receive oxygen before returning to the left atrium, then to the left ventricle.[17] After, the blood is pumped to the systemic circulation. Figure 7 is a diagram of the heart. In order to pump blood, the heart must have a functional electrical conduction system to coordinate the cardiac cycle. The conduction system is composed of the sinoatrial node, the atrioventricular node, atrioventricular bundle, bundle branches, and conduction myofibers.[18] The sinoatrial node is known as the "pacemaker" of the heart because it allows the cardiac cycle contractions and relaxations to occur in sequence. [18]

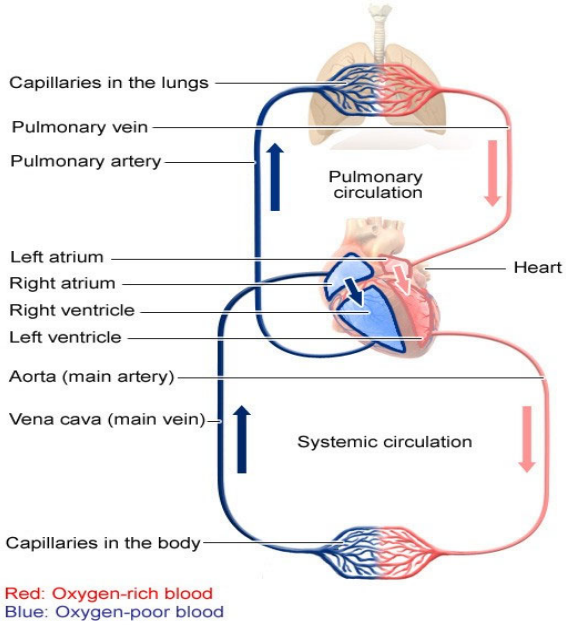
**Figure 7:** The internal view of the heart with its labeled components.[17]



When the blood is pumped out of the right and left ventricles respectively, it is transported through the pulmonary and systemic circulations through the blood vessels, which include the arteries, arterioles, veins and capillaries.[19] Arteries carry blood, that is usually oxygenated, away from the heart with the exception for the pulmonary artery which carries deoxygenate blood to the lungs.[19] This blood then flows into arterioles, which help control flow. The smallest vessels are the capillaries. Capillaries are vessels that function to exchange gases, nutrients and waste products by diffusion, filtration, and osmosis.[19] They are also the connection between arteries and veins. After passing through the capillaries, blood enter the venules and goes on into the veins, which carry blood back toward the heart.[19] This blood is usually deoxygenated since it was used for the metabolic activities needed for homeostasis, the only exception are the pulmonary veins since they carry oxygenated blood from the lungs to the heart for transport to systemic circulation.[19]

There are two main circulatory circuits: the pulmonary circuit and the systemic circuit. The pulmonary circulation transports deoxygenated blood from the right ventricle of the heart to the lungs, where blood gets oxygenated, it then transports the oxygenated blood to the left atrium.[20] The systemic circulation carries oxygen and nutrients to the body tissues and picks up carbon dioxide and waste products.[20] Systemic circulation transports oxygenated blood from the left ventricle of the heart, through the arteries, to the capillaries in the tissues of the body and then the deoxygenated blood returns to the heart through the veins.[20] (See Figure 7)

**Figure 7:** Pulmonary and systemic circulation of the cardiovascular system.[21]



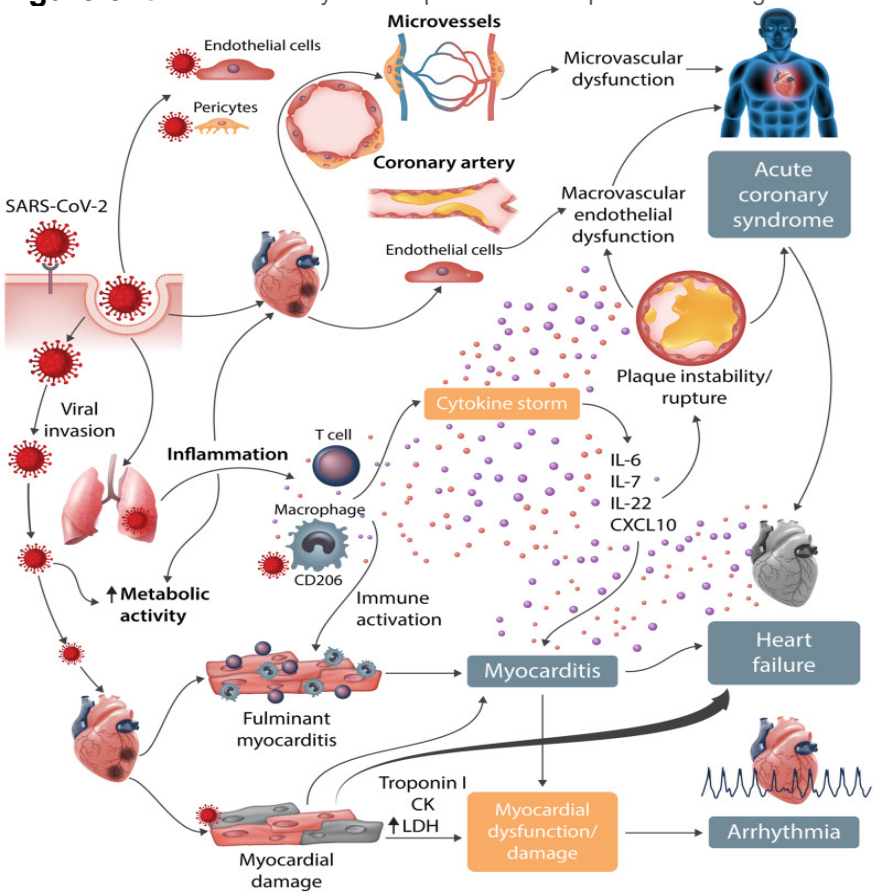

## Cardiovascular System and COVID-19

While COVID-19 is a respiratory disease that primarily affects the lungs and the respiratory system, it can also affect other organs depending on the severity of the infection, especially the cardiovascular system. It is known that people with comorbidities, such as hypertension, were at a higher risk of infection and associated with worsening outcomes, therefore leading to a higher mortality rate.[1] According to a study done in China, cardiovascular disease (CVD) was reported in 4.2% of the population but in 22.7% of those who died.[1] They also found that the fatality rate for those with CVD was 10.5%, for those with hypertension it was 6%, compared 0.9% mortality rate for people with no comorbidities.[1] Moreover, a study found that out of 100 recovered patients there was cardiac involvement in 78% of them and ongoing myocardial inflammation in 60% of the patients.[3]

## Mechanisms

Mechanistically, cardiovascular complications are believed to arise from the binding of SARS-CoV-2 to ACE2 since it is linked to the cardiovascular system.[1] ACE2 is highly expressed in type II alveolar epithelial cells, macrophages, endothelial cells, cardiomyocytes and in pericytes, which line the surface of vascular tubes, and may lead to cardiovascular damage such as microvascular and macrovascular dysfunction.[1, 22] ACE2 is also up regulated during heart failure and may lead to a higher infectivity and mortality rate of the virus.[1, 22] Some links between COVID-19 and the cardiovascular systems are hypertension, myocarditis, myocardial injury, heart failure, arrythmias, ischemic heart disease, coagulation abnormalities, among others as seen in Figure 8.[1]

**Figure 8:** Cardiovascular system response and complications arising from SARS-C0V-2 infection.[1]



In the case of the pathophysiology of hypertension, the renin–angiotensin–aldosterone system (RAAS) increases water and electrolyte reabsorption leading to augmenting sympathetic flow.[1] Therefore, hypertension is treated with ACE inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs) which inhibit RAAS but leads to an increase in ACE2 facilitating a most severe infection of SARS-CoV-2, however this is still not fully understood and contradicted by other sources.[1] Another possible mechanism that links COVID-19 and hypertension is that poorly controlled blood pressure is associated with CD8+ T cell dysfunction and decreases the ability of the immune system to fight off infections.[1]

Other COVID-19 related mechanisms are believed to cause myocarditis and myocardial damage leading to heart failure or arrythmias. As explained above, SARS-CoV-2 infection is characterized by the progression of inflammation and overactivation of cells leading to a cytokine storm.[1] This results in elevated IL-6, IL-7, IL-22, and is believed that activated T cells and macrophages may infiltrate infected myocardium resulting in myocarditis, myocardial dysfunction and cardiac damage.[1] These complications may lead to arrythmias, which is the second most common complication in covid-19 infected individuals after ARDS.[1]

**Higher prevalence of cardiovascular effects in Latin Communities**

Latinos across the united states face disproportionate health impacts from COVID-19 compared to other races and ethnicities. According to the Centers of Disease Control and Prevention (CDC), Hispanic/Latino population comprise 18.5% of the entire Unites States population but they encompass 29.3% of all COVID-19 cases.[23] Latinos also comprise 18.9% of all deaths, but the number increased to 34.5% when they

weighted the populations distributions.[23, 24] The exact reason for the disproportionate prevalence is unknown but may be caused by many different reasons, such as coexisting medical conditions, access to healthcare, and financial burden, among others.[25] All these reasons contribute to the cardiovascular effects of COVID-19 infections among the Latino community.

As discussed above, comorbidities lead to a most severe COVID-19 infection which result in a worse outcome. According to a research done by Georgetown University, 56% of the Hispanic population have at least one chronic illness and 39% have multiple conditions.[26] Among Hispanic adults over the age of 20, 48.3% of men and 32.4% of women have a cardiovascular disease.[27] And according to the CDC, Hispanic adults over the age of 20, 46.0% of men and 35.4% of women have hypertension and 44.8% of men and 46.8% of women are diagnosed with obesity.[28] These extremely concerning rates help explain the elevated mortality rate of COVID-19 among the Latino population and being at a high risk for exacerbating their pre-existing cardiovascular conditions.

Another reason for the disparities that Latinos face is their lack of access to medical care which is caused directly by their lack of medical insurance, financial burden, immigration status, and language barriers. The percentage of uninsured Hispanics is 30.2% within adults ages 18 to 64.[28] Due to being uninsured and not being able to afford healthcare, these individuals tend to not seek medical care or get tested, due to fear of cost or stigma.

Moreover, many seek other forms of treatment, such as traveling to Mexico for medication or self-medicating with over the counter or homeopathic treatments.

According to a discussion I had with Mexican doctor Sergio Rivera, he explained that he had seen an increase in the number of office visits from people that lived in the United States, looking for quick and aggressive treatment for their COVID-19 infections as well as purchasing antibiotics that were not fully approved as COVID-19 treatments. Some medications that these individuals were self-medicating with included: ivermectin, dexamethasone, hydroxychloroquine and azithromycin, all of which are easily accessible to the public but associate with several cardiovascular side effects. Some of these medications had been found to be helpful in treating the infection, but people were taking them at higher doses, without need for them, or without being aware of their side effects. For example, ivermectin was found to have no therapeutic effect against covid and some people using it required medical treatment due to its cardiovascular side effects of hypertension, tachycardia, and edema.[29, 30] Moreover, hydroxychloroquine has been found to damage the conduction system of the heart by elongating the QT interval can lead to arrythmias and even death.[31]

## Conclusion

Due to the ongoing pandemic, it is difficult to make any conclusions as data is still being collected and investigated across the world. However, with the data and research studies available, it is permissible to conclude that the immune system attack on SARS-CoV-1 virus leads to a cytokine storm resulting in inflammation. This inflammation may lead to multi-organ failure and, in the worst-case scenario, to death. The infection can be intensified by the cardiovascular system if comorbidities are present in the individual. Higher rates of infection and death are seen among the Hispanic/Latino population leading to believe, based on data, that pre-existing medical

conditions and lack of access to appropriate healthcare are the main reasons for the disproportionate disparities, especially the effects in cardiovascular disease. Pre-existing medical conditions, which have a high prevalence among Hispanics, were found to intensify the severity of the infection mainly caused my heart failure or cardiovascular diseases. The lack of appropriate medical care also led to an increase in cardiovascular problems related to inappropriate treatment of the infection or no treatment at all. Long-term cardiovascular effects of COVID-19 infection are still being investigated but it has come to the attention of scientists that many people are experiencing from post-COVID health issues and more data will be reported as it is available to them.

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## Appendix: Reason Behind My Research

As a Latino member of my community, I wanted to have a better understanding of the SARS-CoV-2 virus and how it affected our bodies and communities. Being from a border town with Mexico, I got to experience COVID-19 from two very different settings. Mexico has a very different healthcare system than the United States. Most people don't have health insurance and if they do, it is still best to go with a private physician as they offer better care. In Mexico, doctors were treating the infection aggressively and prescribing many different medications that were not approved as treatment for COVI-19. Consequently, the country suffered from a shortage of medication due to people panicking and self-medicating. Although the United States was not prepared for the pandemic, the government and healthcare system did the best they could to handle it. On the other hand, Mexico, classified as a developing country, had a very difficult time through the pandemic. The healthcare system in Mexico collapsed and could not handle the burden that it was presented with. I, personally, had a difficult experience with COVID-19 when my dad got infected, and he did not want to go to the hospital due to his lack of health insurance. Instead, he went to a private physician in Mexico who prescribed 9 different medications to treat his symptoms. Fortunately, he was able to get through it. However, till this date, he is still experiencing some post-COVID-19 infection symptoms such as worsening hypertension, fatigue, and difficulty thinking and concentrating. I did this research to investigate what is the reason he is still experiencing symptoms specially his worsening hypertension.