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Association of ABO Blood Group with COVID-19: A Review on Effectiveness and Severity.

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Abstract

COVID-19 is related to the blood type ABO system and vulnerability to COVID-19 infection. The ABO system comprises the B, A, and O alleles, which impact the antigen expression on red blood cells, and the presence of naturally occurring antibodies in plasma. People with type A blood may now be more susceptible to contracting COVID-19. On the other hand, type O may indicate a decreased risk of serious viral consequences. One theory aims to explain the relationship between blood type and risk of infection. In individuals with blood types B and O, anti-A antibodies may prevent the virus's spike protein from adhering to human cells. This could reduce the likelihood that the virus will infiltrate when issues are not only protected by these protections but also by similar ones. IgG versions of anti-A antibodies, which are frequently produced by people with type O blood, seem to be more efficient at preventing the virus than the IgM forms, which are typically produced by people with type B blood. When things go wrong in the body during bad cases of COVID, those changes are tied to what happens early on. Not far behind comes a detail about TMPRSS2 - this protein opens the door for the virus, yet blood type may dial its activity up or down. Studies confirm this: your blood group can change your chances of getting an infection and the severity of the disease. This evidence explains the variation in outcomes across people; one person recover it easily, while another fights a much worse strain. Genetic factors may play a role in determining a person's fate on this scale

Keywords: Covid-19, SARS-CoV-2, ABO blood groups, angiotensin, cytopathic

الملخص

يرتبط فيروس كوفيد-19 بنظام فصائل الدم **ABO** وبمدى التعرض للإصابة بالفيروس. يتألف نظام **ABO** من الأليلات **B** و **A** و**O** ، التي تؤثر على تعبير المستضدات على خلايا الدم الحمراء، وعلى وجود الأجسام المضادة الطبيعية في البلازما. وقد يكون الأشخاص من فصيلة الدم **A** أكثر عرضة للإصابة بفيروس كوفيد-19. من ناحية أخرى، قد تشير فصيلة الدم **O** إلى انخفاض خطر التعرض لمضاعفات فيروسية خطيرة. تهدف إحدى النظريات إلى تفسير العلاقة بين فصيلة الدم وخطر الإصابة. في الأفراد من فصائل الدم **B** و**O** ، قد تمنع الأجسام المضادة لـ **A** بروتين سبايك الفيروس من الالتصاق بالخلايا البشرية. وهذا قد يقلل من احتمالية تسلل الفيروس عندما لا تكون الخلايا محمية بهذه الحماية فحسب، بل أيضًا بحماية مماثلة. يبدو أن نسخ **IgG** من الأجسام المضادة لـ**A** ، التي ينتجها غالبًا الأشخاص ذوو فصيلة الدم**O** ، أكثر فعالية في منع الفيروس من أشكال**IgM** ، التي ينتجها عادةً الأشخاص ذوو فصيلة الدم **B.** عندما تسوء الأمور في الجسم خلال الحالات الشديدة من كوفيد، ترتبط تلك التغييرات بما يحدث في المراحل المبكرة. ويأتي بعد ذلك مباشرةً تفصيل عن **TMPRSS2** - هذا البروتين يفتح الباب للفيروس، لكن فصيلة الدم قد تزيد من نشاطه أو تقلله. تؤكد الدراسات ذلك: فصيلة دمك يمكن أن تغير فرص إصابتك بالعدوى وشدة المرض. تفسر هذه الأدلة التباين في النتائج بين الأشخاص؛ حيث يتعافى أحدهم بسهولة، بينما يصارع آخر سلالة أسوأ بكثير. قد تلعب العوامل الوراثية دورًا في تحديد مصير الشخص على هذا النطاق.

الكلمات المفتاحية: Covid-19، SARS-CoV-2، فصائل الدم ABO، الأنجيوتنسين، التلف الخلوي.

1. Introduction

Three major coronavirus pandemics have occurred since 2002. Not long after 2002 began, a serious respiratory illness surfaced in China - called SARS-CoV., and moved through several countries before fading by 2013 (de Wit E. et al., 2016). Then came early reports near the end of 2019: another virus, named SARS-CoV-2, started spreading fast from the same region. Unlike earlier events, this one did not stop - it turned into something much wider, known now as COVID-19 (Shurin et al., 2020). This piece looks at what researchers currently understand about how different ABO blood types might

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connect to who gets hit harder - or more often - by the infection (Shokri et al., 2021).

2. Pathogenesis of COVID-19 infection:

It also explores potential pathways underlying the action between various ABO blood groups and the virus. SARS-CoV-2 belongs to the coronaviridae family, which is taxonomically categorized into four genera: Alpha (a), Beta (β), Gamma (γ), and Delta (δ). Among these, only the alpha and beta genera have been identified as capable of infecting humans (Shibeeb, 2022). The SARS-CoV-2 belongs to the β-coronaviruses, which are enveloped viruses characterized by a single-stranded (positive-sense) RNA genome (Guo Y-R et al., 2020). SARS-CoV-2, such as other coronaviruses, has four major structural proteins: the spike (S) glycoprotein, envelope (E) protein, membrane (M) protein, and nucleocapsid (N) protein (Fehr et al., 2015). The Virus gentic composition approximately ten (ORFs) open reading frames, with alone ORF1a/b occupying nearly two-thirds of the RNA viral, Transcription of the RNA viral from ORF1a/b two yields polyproteins, pp1a and pp1ab, which are subsequently cleaved into 16 non-structural proteins (nsps) that are essential for the formation of the viral replicase-transcriptase complex (Li et al., 2020). The spike (S) protein is composed of two functional subunits: S1, which contains the receptor-binding domain (RBD) responsible for attachment to the host cell receptor, and S2, which facilitates fusion of the viral envelope with the membrane of the infected cell (Walls et al., 2020). SARS-CoV-2 gains entry into infected cells by binding to the angiotensin-converting enzyme 2 (ACE2) receptor situated on tissue cells of humans (Zhou et al., 2020). Viral attachment to the host cell is mediated by the S1 subunit of the spike glycoprotein through its interaction with the ACE2 receptor, whereas the S2 subunit، facilitates membrane fusion and entry (Walls & Zhou, 2020). The SARS-CoV-2 pandemic is having an impact on people's health all around the world; some people are more vulnerable to the illness than others, and variations in the clinical outcomes of those infected with the virus are frequently observed. Most people who are infected with the virus show no signs, yet can pass it on. Some get a fever or cough. Others experience difficulty breathing, even developing serious lung issues. Scientists believe that how each person's immune system reacts plays a big role. These differing responses may explain why sickness levels vary so much. A study conducted in 2020 confirms this. Reducing viral replication and stopping the spread of infection to the lungs can be achieved through an early and effective immune response. In contrast, an exaggerated or dysregulated immune response may trigger a hyperinflammatory state (Shibeeb & Khan, 2021). Furthermore, epidemiological data indicate a higher prevalence of comorbidities such as hypertension, diabetes, and liver disease among patients with severe COVID-19, suggesting that underlying metabolic conditions may influence the host's immune response and contribute to infection (Costa et al., 2020).

3. Cytopathic effect:

SARS-CoV-2-associated coronaviruses gain insertion in the infected cell by the attachment S- protein to the virus, to special proteins that bind to the cell covering. The tendency of different coronaviruses to bind is determined via the S-protein. Homotrimer on the infection cell surface biomolecules, including C-type lectin domain family member 4 or CD299 (CLEC4M/DC-SIGNR) to SARS-CoV human angiotensin-converting enzyme 2 (hACE2), (APN) Aminopeptidase N for SARS-CoV, Dipeptidyl peptidase 4 (DPP4) to MERS-CoV, in addition to Neuropilin-1(NRP1) and carcinoembryonic antigen-associated cell binding molecule 1 (CEACAM1) for COVID-

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19, inspit the SARS-CoV-2 spike protein of main target is the ACE2 receptor, in addition to the TMPRSS2 serine protease, that to spike protein is used. The association establishes the potential of insertion of the virus enter the various cellular phenotypes within the host, as through the expression of some of these biomolecules on the surface. This relation, identical between coronaviruses connected to SARS-CoV, stimulated a transition of structural form from prefusion to post-fusion that includes processing by proteases such as TMPRSS2 produced in the surface of the infected cell or furin and cathepsins in the endosomal compartment. Then, a pore of fusion is formed, facilitating the fusion of both cell membranes and the viral membrane, therefore enabling the viral genome to reach the cytoplasm of the cell. Viral reproduction occurs primarily in virus-induced (DMV) double-membrane vesicles obtained from the endoplasmic reticulum(ER), where they come together to create complex networks of detailed membranes. The entering viral genome serves as a template for nsp12 to produce viral genomic RNA, and all viral proteins are introduced into the ER-Golgi intermediate compartment (ERGIC) for joint association with the virion genome.The freshly formed virions are released through the plasma membrane, just as other viruses linked to SARS-CoV. Furthermore, it has been shown that the symptoms of COVID-19 infection extend beyond the respiratory system, resulting in different extrapulmonary symptoms. Therefore, depending on cellular phenotype, virus reproduction in the human body may have different effects on tissue physiology and/or cellular function. Understanding the variety of clinical outcomes associated with COVID-19 may be facilitated by identifying putative pathogenic pathways that are stimulated by molecular reactions between the virus and the host in a tissue-specific manner (Costa et al., 2020).

3.1. Mitochondrial cytopathic effect:

The distribution, size, and structure of mitochondria are all impacted by SARS-CoV proteins. SARS-CoV's alteration of mitochondrial structure, spatial organization, and functional activity contributes to its pathogenesis. In HEK293 cells, the SARS-CoV orf9b localizes into the mitochondria and induces the mitochondria to elongate. Additionally, orf9b inhibits mitochondrial fission by activating the ubiquitination and proteasomal degradation of DRP1. Additionally, there was contact between the elongated mitochondria and autophagosomes. Mitophagy may be triggered by the interaction between mitochondria with the autophagosomes. Therefore, impaired antiviral response and signaling may result from a disruption of the mitochondrial structure. Not every protein encoded by SARS-CoV is transported to the mitochondria (Shama et al., 2023).

3.2. Cytopathic effect on the Golgi:

COVID-19 is a global pandemic resulting from infection with SARS-CoV-2, an enveloped coronavirus. Despite extensive research, the molecular mechanisms underlying its formation and secretion are still largely unknown. Therefore, they demonstrate how the host endomembrane system is completely altered by SARS-CoV-2 infection, including the Golgi apparatus. In the broken Golgi, SARS-CoV-2 virions are more prevalent. SARS-CoV-2 assembly and secretion are significantly inhibited by blocking endoplasmic reticulum (ER) to Golgi trafficking, although viral genome replication is not decreased. Notably, SARS-CoV-2 infection increases TGN46 protein levels while downregulating GRASP55. Remarkably, GRASP55 depletion has the opposite effect from GRASP55 expression, which reduces viral production and spike

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quantity on each virion without affecting viral entry. On the other hand, TGN46 depletion does not affect spike integration into virions; it simply prevents viral secretion. Therefore, we note that SARS-CoV-2 alters Golgi structure and function to modulate viral assembly and secretion, highlighting the Golgi as a potential therapeutic target for blocking SARS-CoV-2 infection (Zhang et al., 2025).

3.3. Cytopathic effect on the Endoplasmic Reticulum:

ORF3a is exceptional to SARS-CoV and SARS-CoV-2 among the seven known human coronaviruses, which suggests the clinical importance of ORF3a in inducing severe human diseases like SARS or COVID-19. Additionally, clinical studies found that COVID-19 patients have significant amounts of anti-ORF3a antibodies, and their sera showed increased IgG and IgA reactivity, particularly against ORF3a. ORF3a has also been connected to the severity and mortality of SARS-CoV-2 in numerous studies. It has been demonstrated in vitro that ORF3a triggers a proinflammatory immunological response, resulting in inflammasome activation, utilizing either cells expressing ORF3a alone or cells infected with COVID-2. The mortality rate associated with SARS-CoV-2 is attributed to NLRP3, a key component of cytokine storm. Furthermore, ORF3a expression in kidney and lung epithelial cells triggers innate oxidative stress and infectious cellular proinflammatory immunological responses, which in turn promote apoptotic cell death through the production of reactive oxygen species (ROS) mediated by cellular oxidative stress. Additionally, nuclear factor kappa B (NF-kB) mediated tumor necrosis factor-alpha TNFα and interleukin-6 cytokine formation, which are two robust and independent predictors of SARS-CoV-2-associated death (Pablo et al., 2023; Zhang et al.,2023).

3.4. Cytopathic effect on the Nucleus:

Pathogenic SARS-CoV-2 commonly interferes with the interferon (IFN) process but stimulates an anomalous immune response by releasing elevated cytokines and chemokines (cytokine storm). When host cells identify different pathogen-related molecular patterns, they create and release IFNs, a family of antiviral cytokines. IFNs come in three different varieties: Type I IFNs, such as α and β, have antiviral properties. The sole type II IFN (γ) combats viral and bacterial infections. Following viral or fungal infections, type III IFNs (λ) are generated. Coronaviruses target all three types of IFNs. The expression of type I and typeIII interferons produced by SARS-CoV-2 is influenced by several parameters, including age, disease severity, and tissue location. In addition, SARS-CoV-2 downregulates type II IFN (Chen et al., 2022).

4. Complications:

4.1. Respiratory Complications: Following COVID-19, a range of long-term respiratory problems have been reported, including persistent symptoms, radiographic abnormalities, reduced pulmonary function, vascular consequences, thrombosis, and pulmonary fibrosis (Al-Jahdhami et al., 2022).

4.2. Cardiovascular Complications: It is well established that SARS-CoV-2 infection increases cytokine activity, which in turn increases cardiac activity. Additionally, by attaching to ACE2 receptors in heart cells, the virus may cause direct harm to myocardial tissue. Myocarditis, myocardial damage, venous thromboembolic events, dysrhythmias, acute myocardial infarction, and heart failure are among the complications (Long MD et al, 2020).

4.3. Genitourinary Complications: Some impacts on the male genitourinary system, including significant changes in semen parameters, such as azoospermia,

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oligozoospermia, and cryptozoospermia, have been linked to SARS-CoV-2 infection. Furthermore, total testosterone levels are lower in COVID-19 patients, with reductions being more noticeable in those with severe illness. There have been many reports of lower urinary tract symptoms, including urgency, nocturia, and increased frequency of urination. Testicular inflammation, epididymitis, testicular edema, discomfort, and tenderness indicated possible testicular injury. Notably, five cases of priapism have also been documented, further increasing the impact of COVID-19 on the male reproductive system (Luca Schiliró et al., 2023).

4.4. Gastrointestinal Complications: SARS-CoV-2 is associated with gastrointestinal complications such as pancreatitis, perforation, and, less commonly, pneumatosis intestinalis, gastrointestinal hemorrhage, thrombotic and ischemic events (Ribeiro et al., 2021).

5. Blood Type System: The blood type (ABO)system, detected in 1901, involve three polymorphic forms—B, A, and O—encoded by the blood group gene, The assembly of the 3 polymorphic forms (RBCs) red blood cells outcomes in four phenotypes and 6 potential genotypes, producing antibodies in plasma with antigens on (RBCs) (Yamamoto, 2021). Since its discovery, the ABO blood group system has been extensively investigated for possible associations with various diseases and infections. Polymorphisms within the ABO system have been reported to influence both susceptibility to and clinical outcomes of numerous pathological conditions, such as coronary heart disease, tumors, *Helicobacter pylori* (Liu et al., 2020), SARS-CoV, and Hepatitis B Virus (W J. et al., 2020). ABO blood type and COVID-19 infection and susceptibility:

5.1. ABO and COVID-19 infection and susceptibility:

The effect of the blood type (ABO) system on SARS-CoV-2 susceptibility and certain COVID-19 patients at three hospitals in China (Zhao, 2021). One study found that people with blood type A get hit by SARS-CoV-2 more often compared to people who have type O. In groups where about 31% carry type O and 34% have type A, the pattern stands out clearly. Cases confirmed as COVID-19 included 37% of individuals with blood group A. Meanwhile, just 26% were from group O in the same data set. This difference indicates an increased susceptibility to infection among A types. In contrast, type O may offer some protection, though it is far from total. The trend does not prove the existence of a causal relationship, but it tends to suggest the existence of a correlation. However, numbers keep nudging us down this path (Wu et al., 2020). Some people with type O blood seem resistant to the virus, possibly because their blood serum contains a form of IgG that controls inflammation from the outset. Research indicates that people with type A face worse outcomes more often than others. Type O; it tends to show up less in infection tallies. However, the death rates in different studies do not match exactly. One thing some data agree on is that having type A might raise the chances of getting hit hard, even fatally (Sol Pardo & Susana, 2025).

5.2. Mechanisms for the association between ABO blood types and COVID-19:

Some think that blood type shapes who are more susceptible to the virus. Not everyone agrees with that, but one idea suggests the built-in defenses, such as anti-A shields found in certain people. Another angle suggests the virus might carry sugarcoated markers matching those on red cells. Blood groups also affect the fast clotting form, a detail that could shift outcomes. Subtle differences in the DNA behind blood typing may also affect the level of risk. Furthermore, it is known that the

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glycosyltransferases produced by the A and B alleles alter glycosylation patterns in different cell types, including respiratory epithelial cells. There is growing evidence that people with blood groups O and B inherently have anti-A antibodies (Miotto et al, 2021). People with blood type O may have better resistance to SARS-CoV-2 because they carry type anti-A antibodies. These molecules are usually found in people with O or B blood, but not at all in people with blood type A. Instead of binding directly, the virus might get blocked when these antibodies interfere with how its spike protein binds to ACE2 receptors. Data from population studies indicate fewer infections among groups carrying either O or B blood types, those equipped with anti-A shields; however, a high number of cases accumulate where these defenses are lacking. Interestingly, lab work later indicated that the version made by blood type O bodies tends to be more resistant to invasion compared to the one from blood type B. Because anti-A antibodies found in people with blood type O tend to be IgG, while in blood type B, they're mostly IgM, the stronger shield may be due to this difference in antibody type(Gérard et al., 2020). Preventing the spike (S) protein of SARS-CoV from attaching to the ACE2 receptor on cells, this blocking effect is clearly demonstrated when A antibodies intervene (Yan et al., 2020). Furthermore, it has been reported that IgM-class antibodies are associated with phenotypic glycosylation patterns in non-O blood groups, which may contribute to reduced isoagglutinin activity. Another proposed mechanism suggests that during replication within host epithelial cells, SARS-CoV-2 may acquire host-like glycan antigens (Zaidi et al., 2020). When SARS-CoV-2 acquires specific glycan antigens reflective of the blood type of the original host, subsequent transmission to a person with a different blood type may result in the presence of identical natural antibodies. These antibodies can potentially inhibit the interaction between the viral spike (S) protein and the ACE2 receptor on host cells (Yamamoto et al., 2020). The blood group (ABO) system has been linked to variations in angiotensin-converting enzyme (ACE) activity. One reason ties back to a specific genetic pattern in the ABO gene - called GATC - that carries four distinct variations: rs495828, rs8176740, rs8176746, and rs12683493. People with blood type O often have less ACE enzyme in their bodies, yet more interleukins appear. Because they naturally produce anti-A antibodies, the virus may have difficulty binding to ACE2 through its spike proteins. Then the TMPRSS2 enzyme helps the invading virus enter cells. Recent findings suggest that blood group could influence just how much help this virus gives (Mehran et al., 2024).

6. Conclusion:

SARS-CoV-2 has disrupted financial systems and strained healthcare networks globally. Many individuals contracted the virus from the outset; some were asymptomatic, while others died from the illness. The relationship between ABO blood types and who contracts the virus and how sick they become is now being investigated. Research suggests that your blood type may have an effect on your risk of contracting an infection, its severity, and even your chances of surviving. Individuals with type O blood appear to be less susceptible to infection. When they do, their symptoms are usually modest. Conversely, those with type A have a higher risk of contracting the disease; in addition, worse results are more prevalent there. However, it is advisable to be cautious when interpreting these patterns. Small groups were used in some experiments. Different methods have also been used in other research to select control groups. Furthermore, the distribution of blood types varies across geographical regions.

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The observed results may be affected by this variation. The prevalence of each blood type varies by location, and for this reason, broad judgments may be inaccurate. To obtain more answers that are accurate in the future, more research is required and should include larger populations. However, it is unclear what physical processes underlie this.

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