Review on viral bioflims against SARS-CoV-2 (COVID-19).

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Abstract

Corona viruses are important human and animal pathogens. At the end of 2019, a novel coronavirus was identified as the cause of a cluster of pneumonia cases in Wuhan, a city in the Hubei Province of China. It rapidly spread, resulting in an epidemic throughout China, followed by an increasing number of cases in other countries throughout the world. In February 2020, the World Health Organization designated the disease COVID-19, which stands for coronavirus disease 2019. The virus that causes COVID-19 is designated Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2); previously, it was referred to as 2019-nCoV.

Today, efforts by various countries are based on the rapid diagnosis, isolation of patients and care that can combat the disease's most severe effects. The number of COVID-19 confirmed infections is still that. Unfortunately, no drugs or vaccines have been approved for the treatment of human coronaviruses but thorough research on emerging human infectious coronaviruses is urgently needed. The advancement of successful prevention and treatment strategies will be facilitated by clarification routes and pathogenic pathways and the identification of possible drug therapy targets. Due to public health emergencies, it is important to research the potential effects of therapeutic methods on SARS-CoV-2 in the absence of established successful therapies. This review summarizes COVID-19's epidemiologic features, pathogenesis, virus structure and targeting strategies. In the meantime, this review also focuses on viral biofilms as a therapeutics effect that can be used to treat novel COVID-19.

Keywords: Pathogens, Virus, COVID-19, RNA, Genome, Oxidative stability.

Introduction

Coronavirus (CoV), which belongs to the Coronaviridae family, has spikes at the external surface, which make it look like a crown. Its an enveloped viruses consist of a genomic single-strand RNA material together with a helical nucleocapsid in continuous conformation with the RNA bead and string type. It's about 65 nm-125 nm in diameter and vary in length from 26 kbs-32 kbs. This family of viruses consists of subsets based on its genomic structure: α, β, da CoV. Until now, four human circulatory coVs that have low pathogenicity and mild respiratory symptoms such as NL63 and 229E have been reported that are α CoVs; OC43 and HKU1, which are β-CoVs. Two serious Respiratory Tract Infections (RTIs) in the 21st century. Severe ARS (SARS) caused by SARS-CoV (β-CoV) emerged in the province of Guangdong China in 2002-2003; and MERS-CoV (β-CoV) in the Middle East emerged in 2012. Both were of bat origin and had a fatality rate of 11% and 34% respectively. Palm civet cats were the intermediate hosts for bats to humans in SARS, and dromedary camels in MERS. Air discomfort and lung disease contribute to pulmonary disorder and mortality is triggered by SARS and MERS.

Literature Review

COVID-19 is originated from Wuhan, capital of Hubei province, China, clusters of cases of extreme pneumonia were reported due to unknown causes in late December 2019. Many of the initial cases were reported as having popular access to the Huanan seafood market that included the selling of dead seafood animals and live animal trading. Because China had a rapid surveillance program after the SARS outbreak, the patient's respiratory samples were sent for etiological analysis to reference laboratories [1]. Patient screening for viral pneumonia was conducted by testing the broncho-alveolar lavage fluid using polymerase chain reaction, full genome sequencing, and cell culture. The Chinese government told the World Health Organization (WHO), meanwhile closing down the Huanan seafood market on January 1st 2020. After then, the number of cases has started to grow dramatically, including to those without access to the seafood market, suggesting human transmission to humans [2]. On 11th January the first fatality was identified. It turned out to be an epidemic, initially spreading to other countries including Thailand, South Korea and Japan as Chinese New Year's Eve sparked major migration. On 7 January, this virus was reported as being β-CoV. Bat coronavirus, namely the RaTG13 genome, had 96.2% homology while SARS coronavirus had 79.5 % homology. [3]

Discussion

Biofilms protect and improve the survival of microorganisms by enclosing them in an extracellular matrix. Previous research has shown that viruses can colonise preexisting biofilms secondarily, and viral biofilms have also been described. We

argue in this review that CoVs can infect bats indefinitely due to their association with biofilm structures [4]. This phenomenon may provide an ideal environment for nonpathogenic and well-adapted viruses to interact with the host and for viral recombination [5]. Biofilms can also improve virion viability in extracellular environments like fomites and aquatic sediments, allowing for viral persistence and spread. Furthermore, understanding the biofilm lifestyle of CoVs in reservoirs may help to answer several pressing questions about the persistence and transmissibility of highly pathogenic emerging CoVs (Table 1).

Table 1. *The similarity between COVID-19, SARS, and MERS.*

SARS		MERS	COVID 19
Symptoms	Fever, Headache, Dry Cough and Shortness of Breath, Without Upper Respiratory System	Fever, Cough and Shortness of Breath	Fever, Cough and Shortness of Breath
Incubation Period	2 Days-10 Days	14 Days	2-14 Days
X-Ray	Confirmed by Pneumonia by Day 7-10	Not Always Seen	May be Confirmed

Pathogenesis

MERS-CoV and SARS-CoV are considered highly pathogenic and were transmitted by bats to humans by palm civets or dromedary camels. SARS-CoV-2 characterization and genomic sequencing showed that the novel CoVs were 88% identical to the two previously described bat-derived SARS-like CoVs, but differed from SARS-CoV (79%) and MERS-CoV (50%), suggesting that they originated from bats but the intermediate source is not yet established [6]. The phylogenetic study showed that SARS-CoV-2 was genetically dissimilar from MERS-CoV and SARS-CoV. Coronavirus genome size ranges from approx. 26,000 to 32,000 bases containing 6-11 ORFs. The first ORF containing approx non-structural proteins. Although remaining ORF encodes structural proteins and accessory proteins, 67% of the total genome. Among the four main structural glycoproteins Spike glycoprotein(S) plays a critical part in host receptor binding. Homology simulations shows that SARS-CoV-2 and SARS-CoV penetrate the lung use the same human cell receptor [7]. ACE-2 ignores main residue variation in amino acid, while MERS-CoV enters the host cell *via* DPP4. Six mutations occurred in SARS-CoV-2 Receptor-Binding Domain (RBD) variable parts, but no amino acid replacements were found in RBD specifically interacting with host cell ACE-2 receptors.

Analysis of receptor affinity claims that the SARS-CoV strain binds less efficiently than the novel SARS-CoV-2 strain, and further mutation in RBD's nucleotide sequence can increase pathogenicity. It is uncertain whether higher SARS-CoV-2 receptor affinity than SARS-CoV may lead to more serious lung contact [8]. This should be further investigated because studies *in vitro* inoculating SARS-CoV-2 in human airway epithelial cells showed a cytopathic effect with cessation of cilia movements. ACE-2 receptor is not only strongly expressed in the lungs but also in the small intestines, kidney, testis and liver, and thus SARS-CoV-2 may also be affected (Figure 1).

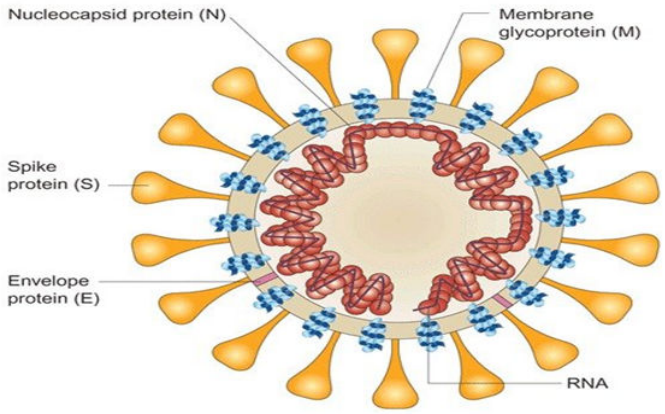

Figure 1. Structure of Corona Virus.

Important factors about COVID-19

Some of the SARS-CoV-2 proteins identified by the 3b gene, which suppresses part of our immune response to viral infections, a couple of publications analyzed variants last month. This specific version of or f3b does this more successfully than the original, and in the absence of an successful immune response will allow the virus more room to reproduce. The consequence can be a sicker patient [9].

A pair of publications last month examined variants in one of the SARS-CoV-2 proteins identified by the ORF3b gene which suppresses part of our immune response to virus infections. Another specific version of ORF3b does this more efficiently than the original, and in the absence of an successful immune response may give the virus more opportunity to reproduce [10]. The outcome may be a ill patient.

Through our experience of other viruses such as HIV-1 and influenza we realize that we are not meeting a stagnant opponent. As we struggle to control the pandemic, the novel coronavirus is changing. SARS-CoV-2 has already proven a powerful foe. While our success today though arguably limited-is important, we should not lose sight of the long battle that lies ahead against microbes.

Preventions: To prevent COVID-19 from spreading,: Clean your hands very often. Using soap and water, or a hand massage of alcohol. Maintain a healthy distance from someone coughes or sneezes. Should not cross the head, mouth or nose. If you cough or sneeze, protect your nose and mouth with your bent forearm or a tissue. When you feel unwell, return put.If you have a fever, cough and difficulty breathing, seek medical attention. Call in advance. Follow the directions of your local health authority.

Clinical features

The clinical features are as follows:

- Most common symptoms
- fever
- dry cough
- tiredness
- Less common symptoms
- aches and pains
- sore throat
- diarrhoea
- conjunctivitis
- headache
- loss of taste or smell
- A skin infection, or finger or toe discolouration
- Serious symptoms
- difficulty breathing or shortness of breath
- chest pain or pressure
- loss of speech or movement

Conclusion

We propose that viruses may form extracellular assemblies in structure, organization, and distribution that mimic bacterial biofilms. Viral biofilms are formed in close balance with the infected cell, which provides the machinery for the synthesis and assembly of the components of the virus and the components of the matrix.. Eventually, the infected cells have the initial surface adhered to by the biofilmsExpression viral genome pressure would drive matrix components expression and post-translation modifications, while cell-surface rearrangements (e.g. ruffles and filopodia) would help preserve viral biofilms on the cell surface. A closely controlled balance between adhesion, cohesiveness and dispersion will ensure a balance between the generation of viral biofilm and its spread to other cells. An interesting theory is that viral biofilm development may be an antiviral mechanism formed by the infected cell to enclose infectious viral particles, but hijacked for efficient spread by some viruses. Modulated ECM development in infected cells through viral-genome expression will benefit both viral particles stuck on the surface of the infected cells, and viruses transmitted *via* cell contacts.

In addition, as extracellular infections are involved in the spread of viruses, Could virus biofilms can be possible targets for therapy?

This could be envisaged in two ways firstly, virus biofilm formation could be blocked as a means of reducing virus transmission; secondly, and perhaps more importantly, viral biofilms could be modified as a way to enable or improve unique host immune responses that would minimize chronic infection.

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