

SARS-COV-2 (COVID-19): Cellular and biochemical properties.

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Description

COVID-19 caused by SARS-COV-2 first appeared within the Wuhan City of China and commenced to spread rapidly among people. Rapid progression of the outbreak has led to a serious global public ill health of a potentially fatal disease.

It is documented that virus particles have a diameter of around 120 nm. The envelope of the virus in electron micrographs appears as a definite pair of electron-dense shells. Coronaviruses are single-stranded RNA virus genomes with the most important known viral RNA genome, ranging in size from 26 to 32 kilobases. The virion consists of genomic RNA embedded in phospholipid double layers and coated with two differing types of Nucleocapsid protein (N). The Membrane (M) protein (a type III transmembrane glycoprotein) and therefore the Envelope (E) protein are among the Surface (S) proteins within the virus envelope. The cellular structure of the SARS-CoV-2 and its interaction with the target cell. Especially, beta coronavirus subgroup A members even have a shorter spike-like surface protein called Hemagglutinin Esterase (HE). The lipid bilayer envelope protects membrane proteins and therefore the nucleocapsid of the virus while outside the host cell.

Cellular and biochemical properties

Human coronavirus infections are caused only by α and β coronaviruses. CoVs are quite common and approximately 30% to 60% of the population in China has anti-CoV antibodies. The virus can survive a minimum of 2 to three days on dry surfaces and a couple of to 4 days in faeces at temperature. S protein is involved within the binding and entry of the host cell. Therefore, it's the most target for neutralizing antibodies and antiviral peptides.

Infection by different coronaviruses (CoVs) causes alterations within the transcriptional and translational patterns, cell cycle, cytoskeleton, and apoptosis pathways of the host cells. Additionally, CoV infection may cause inflammation, alter immune and stress responses, and modify the coagulation pathways. The balance between the up- and downregulated genes could explain the pathogenesis caused by these viruses. We review specific aspects of CoV-host interactions. CoV genome replication takes place within the cytoplasm during a membrane-protected microenvironment and should control the cell machinery by locating a number of their proteins within the host nucleus [1]. CoVs initiate translation by cap-dependent and cap-independent mechanisms. CoV transcription involves a discontinuous RNA synthesis (template switching) during the extension of a negative copy of the subgenomic mRNAs. The need for base-pairing during transcription has been formally demonstrated in arteriviruses and CoVs. CoV N proteins have

RNA chaperone activity which will help initiate template switching. Both viral and cellular proteins are required for replication and transcription, and therefore the role of selected proteins is addressed.

SARS-CoV-2 is a component of Coronavirinae subfamily with one among the most important positive-sense single-stranded RNA genomes ~30 kilobases and over 10 Open Reading Frames (ORFs). Two polypeptides, polyprotein 1a (pp1a) and pp1ab, are synthesized through ribosomal frameshift between ORF1a and ORF1b during translation. Additionally to the papain-like protease, the 3- chymotrypsin-like protease (3CLpro), also referred to as the most protease, is vital for the posttranslational processing of SARS-CoV-2 polypeptides and therefore the production of 16 non-structural proteins (nsps). The nsps play fundamental roles in replication, transcription, and virus recombination during an infection, where inhibiting the proteases will block the discharge of the nsps and inhibit the maturation and infectivity of SARS-CoV-2. As a result, 3CLpro of SARS-CoV-2 is a beautiful target for the planning of broad-spectrum of antivirals against COVID-19 [2].

In the course of virus epidemics, the power to adapt to external pressure is a crucial factor affecting the spread of the virus. Regarding the envelope S protein, recombination or mutation within the gene of its RBD can occur to market transmission between different hosts and cause a better death rate. Mutation of the aspartate (D) at position 614 to glycine (G614) leads to a more pathogenic strain of SARS-CoV-2, which makes it harder to develop antibodies or vaccines that focus on nonconservative regions [3]. To effectively prevent disease, combinations of various mAbs that identify different epitopes on the SARS-CoV-2 S surface are often assessed to neutralize a good range of isolates, including escape mutants [4].

Conclusion

Currently, no specific therapeutic or prophylactic has been used clinically to treat or prevent SARS-CoV-2 infection. Nonspecific antiviral drugs, like IFN- α (recombinant human IFN- α 1b, IFN- α 2a), remdesivir, chloroquine, favipiravir, and lopinavir-ritonavir (Aluvia), are clinically wont to treat COVID-19 in China. Nevertheless, NIAID-VRC scientists are developing a candidate vaccine expressing SARS-CoV-2 S protein in mRNA vaccine platform technology. Clinical trials of the vaccine are expected within the coming months. Continued strengthening of the monitoring of the SARS-CoV-2 S protein is of great significance for subsequent new drug development and protection against COVID-19.

References

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