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NOVEL CORONAVIRUS (COVID-19) HISTORY, GENOME STRUCTURE AND LIFE CYCLE – A REVIEW

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Abstract: Pandemics such as influenza, smallpox, and plague have caused the damage of hundreds of millions of lives and have occurred for many centuries. Luckily, they have been largely removed by the use of vaccinations and drugs. The coronavirus disease 2019 (COVID-19) pandemic, which causes severe acute respiratory syndrome diseases, is currently the threat to the whole world. The epicenter of this disease initiates in December 2019 in Wuhan, China. Presently the virus has infected roughly 5,000,000 people and caused approximately 500000 deaths worldwide, and these numbers are increasing rapidly. In a review we shell, discuss the basic structure and life cycle of coronavirus.

Keywords: Coronavirus, Pandemic, epicenter, Structure, life cycle

INTRUDUCTION

Coronavirus represents a large family of viruses. These Viruses mostly causes respiratory problems in human beings and also some more serious illness such as Middle East Respiratory Syndrome (MERS) and Severe Acute Respiratory Syndrome (SARS Primary symptoms of illness includes common cold fever, sour throat, etc. [1] . The novel coronavirus (currently referred to as 2019-nCoV) was first detected in Wuhan, China, in December 2019 and appeared to originally affect people who had visited a seafood and animal market selling live games. China (Wuhan) is the epicenter of coronavirus [2]. Currently, this disease has affected 213 countries in the world [3].

Worldwide 521,512 deaths occurred by this disease. On 13 January 2020, the first death of COVID-19 wasconfirmed in Thailand. This is the first recorded case outside China. World Health Organization (WHO) on March 11, 2020, has declared the novel coronavirus (COVID-19) outbreak a global pandemic [4].

Like MARS and SARS, this group of viruses can be transmitted between animals and humans. Bats are the carriers of this disease. In the past, there have been more other deadly pandemic or epidemic diseases; primarily Black Death, smallpox [5], and influenza [6], each of these have killed millions of individuals.

STRUCTURE OF CORONAVIRUS (COVID-19 VIRUS)

The transmission of COVID-19 was increasing day by day. Researchers have moved quickly to describe 2019-nCoV and generally spread their discoveries among the universal exploration network as fast as could be expected under the circumstances. One significant cause of this is the homology models of the novel coronavirus cysteine protease delivered by Stoermer [7]. The quick accessibility of nCoV genomic information made conceivable the creation of original homology models for 3PLpro cysteine protease; a protein that is urgent for viral replication and has been investigated already as an objective for antiviral treatments in the treatment of another coronavirus, SARS. This research clarifies that the viral genome bears a nearby closeness to bat coronaviruses; the protease shows the nearest homology with SARS CoV protease a zoonotic infection that entered the human populace through civets.

In another method, Huang et al. used the crystallographic and biophysical methods. He conducts the structural and functional label of HKU9-RBD. HKU9-RBD is a bat virus [8]. The virus has not transmitted into human beings. The goal for these investigations was that bat beta coronaviruses (a sort that incorporates SARS and MERS) ought to be very much described if they wind up stuff the wellspring of the pursuit worldwide pandemic. Endless supply of the HKU9-RBD receptor-restricting zone (RBD) to the wieldy structures of the SARS-, MERS-, and HKU4-(another bat coronavirus) RBD. He found that even though the transformative chronicles of RNA infections will in unstipulated be nonflexible to decide considering of generous developmental weight, the coronaviruses in this investigation displayed a few saved tertiary vital highlights in the partway subdomain of the spike (S) protein. Investigations was that bat beta coronaviruses (a sort that incorporates SARS and MERS). Endless supply of the HKU9-RBD receptor-restricting zone (RBD) to the controllable structures of the SARS-, MERS-, and HKU4-(another bat coronavirus) RBD. It is very difficult to found plane the evolutionary record of the RNA virus tends to be difficult due to substantial evolutionary pressure, The coronavirus exhibited several conserved the coronaviruses in this study exhibited several structures features In the inside subdomain of coronavirus the spike (S) protein are found. This spike protein, at the Centre of virion surface, is a key factor in determining the all kinds of tropism of the virus as it is involved in receptor recognition and membrane fusion as part of the mechanism of infection. It is supported that the notion of the S protein of beta CoV divergently evolves from a worldwide ancestor, particularly in the external RBD region, and that this determines the potential of a particular betaCoV virus for interspecies transmission.

Lee et al self-mastery least 25000 screening of coronavirus infected samples. He found a small molecule dual inhibitor for chemical papain-like protease (PLpro) enzyme of MERS-CoV and SARS-CoV [9]. He identifies a recipe with inhibitory worriedness versus both enzymes even though the two enzymes withstand important similarities in their overall structures and catalytic sites, the identified recipe acts as a competitive inhibitor versus MERS-CoV PLpro, and an allosteric inhibitor versus SARS-CoV PLpro as unswayable using SPR. Further, though this suggests that the inhibitor recognition specificity of the recipe may differ for MERS-CoV PLpro and SARS-CoV PLpro,

the inhibitor was selective for both of these over two human homologs. Two residues identified through structure and sequence alignments, Y269 and Q270 of the SARS-CoV PLpro were replaced by T274 and A275 in MERS-CoV PLpro complicating the potential for hair-trigger tightness interactions. Considering this with the finding that none of the four tested SARS-CoV PLpro lead inhibitors was constructive versus MERS-CoV PLpro, it is notable that a dual functionality inhibitor was identified for both of the SARS and MERS papain-like proteases Table 1 & Fig 1.

Table1. Shows applications of nonstructural proteins [ref 10-15]

ASP	FUNCTION
asp1	mRNAdegradation and inhibiting IFN inhibition
asp2	Investigations underway
asp3	Peptide cleavage , blocking host immune response, promoting cytokine expression
asp4	DMV formation
asp5	Cleavage and IFN Signaling
asp6	Restricting autophagosomes
asp7	Cofactor with nsp8 and nsp12
asp8	Cofactor with nsp7 and nsp12
asp9	Dimerization and RNAbinding
asp10	Scaffold protein for nsp14 and nsp 16
asp11	Investigations underway
asp12	Primer depended RdRp
asp13	Exoribosenuclease inhibition
asp14	Exoribosenuclease inhibition
asp15	Regulating innate immunity

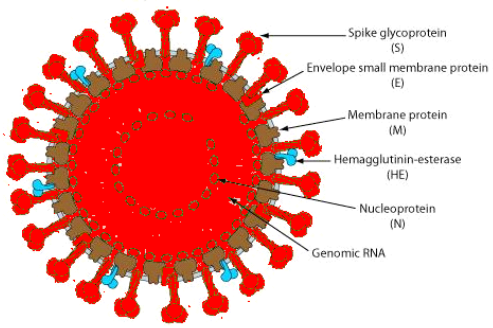

Fig 1 Structure of Corona Virus

CORONAVIRUS LIFE CYCLE (COVID-19 LIFE CYCLE)

Structurally the genome organization of SARS-CoV-2 to the other families of Beta –Coronaviruses. The beta-coronavirus also includes SARS-CoV and MERS-CoV. The structure of viruses containing RNA as genetic material consists of proteins. The proteins are classified into two classes. One structural and nonstructural protein. The structural protein contains Spike envelop, membrane, nucleocapsid, and hemagglutinin enzyme. These viral parts played important roles during infection [16].

The staring of the infection occurs with an interaction between the viral spike glycoprotein (S proteins) and a receptor, angiotensin an enzyme converts enzyme 2 (ACE2) on the host cell surface. ACE2is transmitted in various cell types including those in the lungs. The host called serine protease present in the membrane is important in this process. The key the Trimer S protein knows the serine protease. The Trimer S protein is present on the surface prior to cell entry [17]. The endocytic mechanism occurs for the cleavage of S proteins into two subunits. This process is required for viral and host membrane fusion prior to viral uptake.

During the engulfment, viral genetic material is released into the host cytoplasm prior to translation of the single-stranded viral RNA into long polypeptides that contains nonstructural proteins. These polypeptides are again cleavage into 16 individual nonstructural proteins the mechanism involved is recognized as auto processing mechanism. There are two cysteine like proteases on is known as accessory protease. The other called main protease also known as 3CLpro. The main protease gets cleaves itself and processes the remaining polypeptide into nonstructural proteins (7-16), which makes up the RNA replication-Transcription complex (

RTC) . There are few parts of the RTC, which supports the RNA – dependent RNA polymerase (RdRP) [18-19]. The RdRP is responsible for replicating extra copies of the RNA genome and transcribing several mRNA fragments then encoded structural or accessory proteins. Subsequent to several cycles of replication and translation, the viral particle accumulates and exists in the cell through the budding mechanism, which is known as scissor.

It is supposed that β-coronaviruses trusts on the host cell's endosomal sorting complex required for transport (ESCRT), but the exact method of egress is still not known. When virus particles assemble and areconverted into full scission. The host cell releases the virus to infect more cells and continue to replicate Fig 2.

CONCLUSION:

After SARS and MERS epidemics, great pains have been dedicated to the development of new antivirals targeting CoVs proteases, polymerases, MT ases, and entry proteins; however, none of them is efficacious in clinical trials. The review provides an overview of important molecular pathways involved in the viral life cycle of SARS-CoV-2, the infectious agent of COVID-19. We highlight past and recent findings in essential coronavirus proteins, including RNA polymerase machinery, proteases, and fusion proteins that offer opportunities for the design of novel inhibitors

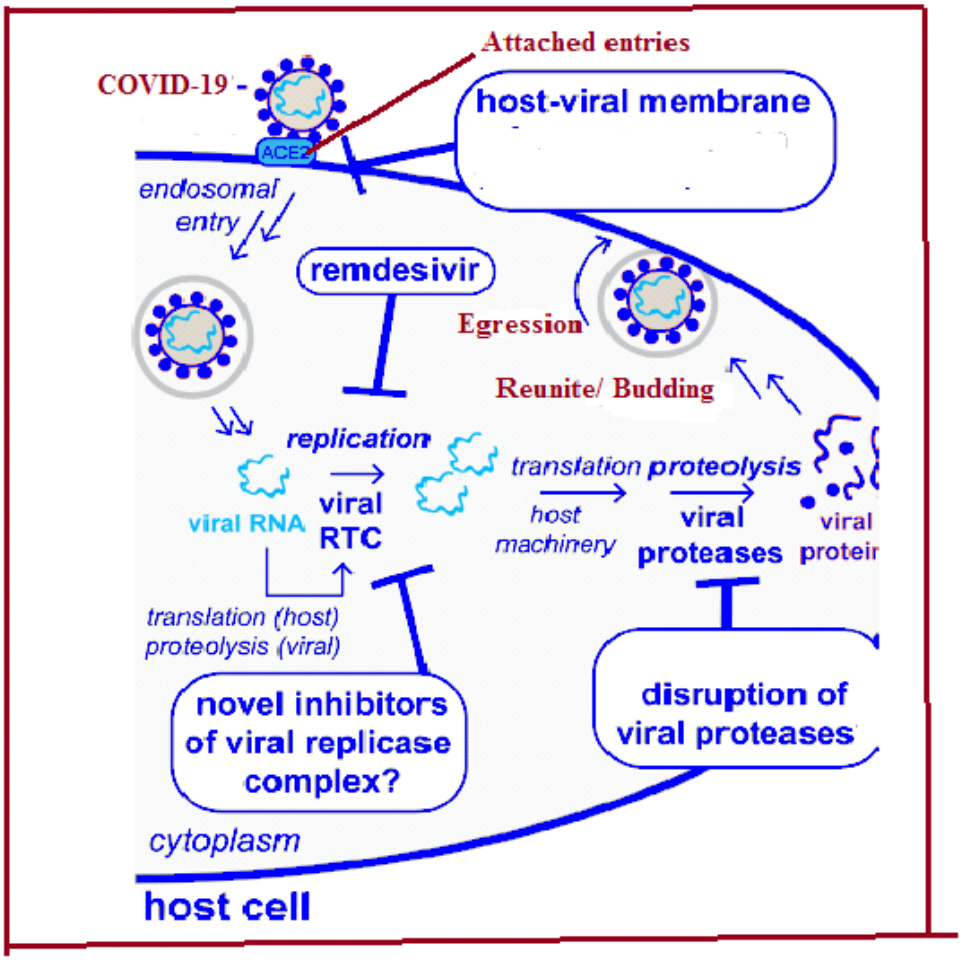

Fig 2 Life Cycle of COVID -19 Virus

of SARS-CoV-2 infection.

Abbreviations	Full form
COVID-19	Coronavirus disease 2019
MERS	Middle East respiratory syndrome
SARS	Severe acute respiratory syndrome
nCoV	Novel Corona Virus
RBD	Receptor binding domain
RNA	Ribose nucleic acid
PLpro	Papain- like protease
SPR	Surface Plasmon resonance
ACE	Angiotensin converting enzyme
RTC	Replication/transcription complex
RdRP	RNA dependent RNA polymerase
ESCRT	Endosomal sorting complexes required for transport

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