Corona Virus Disease- A Pandemic

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Abstract:

Coronavirus disease (COVID-19) is an infectious disease caused by a newly discovered coronavirus. The power of the coronavirus to create an upheaval in people's lives mainly depends on by breathing in the virus if we are within close proximity of someone who has COVID-19, or by touching a contaminated surface and then your eyes, nose or mouth. The present paper illustrates in breif about the virus in general and also about the corona virus. The world is currently face to face with a pandemic which is spreading rapidly across the globe caused by SARS-CoV-2, a strain of Coronaviruses (CoVs) belonging to subgenus Sarbecovirus of genus Betacoronavirus. World Health Organisation (WHO) on 11 Feb 20 named this disease caused by SARS-CoV-2 as Covid-19. This pandemic is spreading rapidly and as on today 47,89,205 cases have occurred globally. The human Coronaviruses discovered in 1960s were considered potentially harmless endemic viruses with seasonal distribution before late 2002. The CoVs are found in a large number of domestic and wild animals and birds.

Keywords:

Virus, Corona virus, Infectious, Pandemic, Health, Epidemic, Transmission, Prevention, Malnutrition and Immunosuppressive.

Introduction and Discussion:

A virus is a sub-microscopic infectious agent that replicates only inside the living cells of an organism. Viruses can infect all types of life forms, from animals and plants to microorganisms, including bacteria and archaea. Scientific opinions differ on whether viruses are a form of life, or organic structures that interact with living organisms.[3] They have been described as "organisms at the edge of life",[2] since they resemble organisms in that they possess genes, evolve by natural selection,[4] and reproduce by creating multiple copies of themselves through self-assembly. Although they have genes, they do not have a cellular structure, which is often seen as the basic unit of life. Viruses do not have their own metabolism, and require a host cell to make new products. They therefore cannot naturally reproduce outside a host cell[5]—although bacterial species such as rickettsia and chlamydia are considered living organisms despite the same limitation.[6] Accepted forms of life use cell division to reproduce, whereas viruses spontaneously assemble within cells. They differ from autonomous growth of crystals as they inherit genetic mutations while being subject to natural selection. Virus self-assembly within host cells has implications for the study of the origin of life, as it lends further credence to the hypothesis that life could have started as self-assembling organic molecules.[1]

Genetic mutation: Viruses undergo genetic change by several mechanisms. These include a process called antigenic drift where individual bases in the DNA or RNA mutate to other bases. Most of these point mutations are "silent"—they do not change the protein that the gene encodes—but others can confer evolutionary advantages such as resistance to antiviral drugs.[7][8] Antigenic shift occurs when there is a major change in the genome of the virus. This can be a result of recombination or reassortment. When this happens with influenza viruses, pandemics might result.[9] RNA viruses often exist as quasispecies or swarms of viruses of the same species but with slightly different genome nucleoside sequences. Such quasispecies are a prime target for natural selection.[10] Segmented genomes confer evolutionary advantages; different strains of a virus with a segmented genome can shuffle and combine genes and produce progeny viruses (or offspring) that have unique characteristics. This is called reassortment or 'viral sex'.[11]Genetic recombination is the process by which a strand of DNA is broken and then joined to the end of a different DNA molecule. This can occur when viruses infect cells simultaneously and studies of viral evolution have shown that recombination has been rampant in the species studied. Recombination is common to both RNA and DNA viruses.[12]

Coronaviruses are a group of related viruses that cause diseases in mammals and birds. In humans, coronaviruses cause respiratory tract infections that can be mild, such as some cases of the common cold (among other possible causes, predominantly rhinoviruses), and others that can be lethal, such as SARS, MERS, and COVID-19. Symptoms in other species vary: in chickens, they cause an upper respiratory tract disease, while in cows and pigs they cause diarrhea. There are yet to be vaccines or antiviral drugs to prevent or treat human coronavirus infections. Coronaviruses constitute the subfamily Orthocoronavirinae, in the family Coronaviridae, order Nidovirales, and realm Riboviria.[13] They are enveloped viruses with a positive-sense single-stranded RNA genome and a nucleocapsid of helical symmetry. The genome size of coronaviruses ranges from approximately 27 to 34 kilobases, the largest among known RNA viruses.[14] The name coronavirus is derived from the Latin corona, meaning "crown" or "halo", which refers to the characteristic appearance reminiscent of a crown or a solar corona around the virions (virus particles) when viewed under two-dimensional transmission electron microscopy, due to the surface being covered in club-shaped protein spikes. A peplomer is a glycoprotein spike on a viral capsid or viral envelope. These protrusions will only bind to certain receptors on the host cell; they are essential for both host specificity and viral infectivity.

Discovery of Corona Virus: Human coronaviruses were first discovered in the late 1960s.[15] The earliest ones discovered were an infectious bronchitis virus in chickens and two in human patients with the common cold (later named human coronavirus 229E and human coronavirus OC43). Other members of this family have since been identified, including SARS-CoV in 2003, HCoV NL63 in 2004, HKU1 in 2005, MERS-CoV in 2012, and SARS-CoV-2 (formerly known as 2019-nCoV) in 2019. Most of these have involved serious respiratory tract infections.

Genome: Coronaviruses contain a positive-sense, single-stranded RNA genome. The genome size for coronaviruses ranges from approximately 27 to 34 kilobases.[14] The genome size is one of the largest among RNA viruses. The genome has a 5′ methylated cap and a 3′ polyadenylated tail.[16] The genome organization for a coronavirus is 5′-leader-UTR-replicase/transcriptase-spike (S)-envelope (E)-membrane (M)-nucleocapsid (N)-3′UTR-poly (A) tail. The open reading frames 1a and 1b, which occupy the first two-thirds of the genome, encode the replicase/transcriptase polyprotein. The replicase/transcriptase polyprotein self cleaves to form the nonstructural proteins (nsps).[17] The later reading frames encode the four major structural proteins: spike, envelope, membrane, and nucleocapsid.[18] Interspersed between these reading frames are the reading frames for the accessory proteins. The number of accessory proteins and their function is unique depending on the specific coronavirus.[19]

Entry: Infection begins when the viral spike (S) glycoprotein attaches to its complementary host cell receptor. After attachment, a protease of the host cell cleaves and activates the receptor-attached spike protein. Depending on the host cell protease available, cleavage and activation allows the virus to enter the host cell by endocytosis or direct fusion of the viral envelop with the host membrane.[19] On entry into the host cell, the virus particle is uncoated, and its genome enters the cell cytoplasm. The coronavirus RNA genome has a 5′ methylated cap and a 3′ polyadenylated tail, which allows the RNA to attach to the host cell's ribosome for translation. The host ribosome translates the initial overlapping open reading frame of the virus genome and forms a long polyprotein. The polyprotein has its own proteases which cleave the polyprotein into multiple nonstructural proteins.[17]

Replication: A number of the nonstructural proteins coalesce to form a multi-protein replicase-transcriptase complex (RTC). The main replicase-transcriptase protein is the RNA-dependent RNA polymerase (RdRp). It is directly involved in the replication and transcription of RNA from an RNA strand. The other nonstructural proteins in the complex assist in the replication and transcription process. The exoribonuclease non-structural protein, for instance, provides extra fidelity to replication by providing a proofreading function which the RNA-dependent RNA polymerase lacks.[20] One of the main functions of the complex is to replicate the viral genome. RdRp directly mediates the synthesis of negative-sense genomic RNA from the positive-sense genomic RNA. This is followed by the replication of positive-sense genomic RNA from the negative-sense genomic RNA.[17] The other important function of the complex is to transcribe the viral genome. RdRp directly mediates the synthesis of negative-sense subgenomic RNA molecules from the positive-sense genomic RNA. This is followed by the transcription of these negative-sense subgenomic RNA molecules to their corresponding positive-sense mRNAs.[17]

Release: The replicated positive-sense genomic RNA becomes the genome of the progeny viruses. The mRNAs are gene transcripts of the last third of the virus genome after the initial overlapping reading frame. These mRNAs are translated by the host's ribosomes into the structural proteins and a number of accessory proteins.[17] RNA translation occurs inside the endoplasmic reticulum. The viral structural proteins S, E, and M move along the secretory

pathway into the Golgi intermediate compartment. There, the M proteins direct most protein-protein interactions required for assembly of viruses following its binding to the nucleocapsid.[21] Progeny viruses are then released from the host cell by exocytosis through secretory vesicles.[21]

Transmission: Human to human transmission of coronaviruses is primarily thought to occur among close contacts via respiratory droplets generated by sneezing and coughing.[22] The interaction of the coronavirus spike protein with its complement host cell receptor is central in determining the tissue tropism, infectivity, and species range of the virus.[23][24] The SARS coronavirus, for example, infects human cells by attaching to the angiotensin-converting enzyme 2 (ACE2) receptor.[25]

Conclusion:

Viruses similar to the pathogen fuelling the current global pandemic have been found in trafficked pangolins and bats as scientists warned that the scaly mammal needs to be banned from animal markets to prevent another coronavirus outbreak in the future. A study from the University of Sydney has shown that Sars-CoV-2, the virus that causes Covid-19, bears genetic similarities to a different strain of coronavirus currently infecting the Malayan pangolin population of southern China and handling these animals requires considerable caution. Therefore its clear that wildlife contains many coronaviruses that could potentially emerge in humans in the future. A crucial lesson from this pandemic to help prevent the next one is that humans must reduce their exposure to wildlife, for example by banning ‘wet markets’ and the trade in wildlife.

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