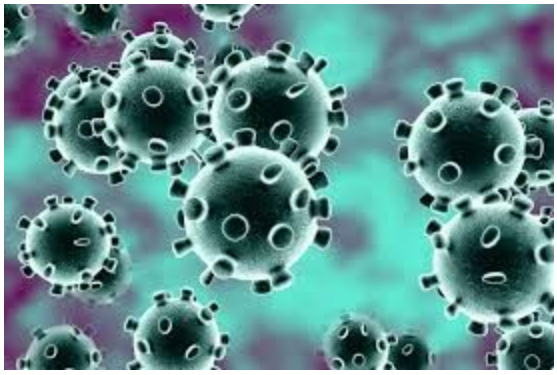
novelCoronavirus



Coronavirus

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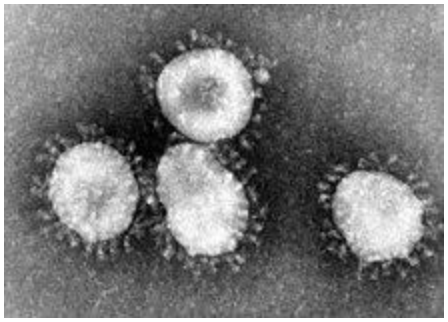
This article is about the group of viruses. For the ongoing disease involved in the COVID-19 pandemic, see Coronavirus disease 2019. For the virus that causes this disease, see Severe acute respiratory syndrome coronavirus 2. For the upcoming Indian film, see Coronavirus (film).

Orthocoronavirinae



Transmission electron micrograph of avian infectious bronchitis virus

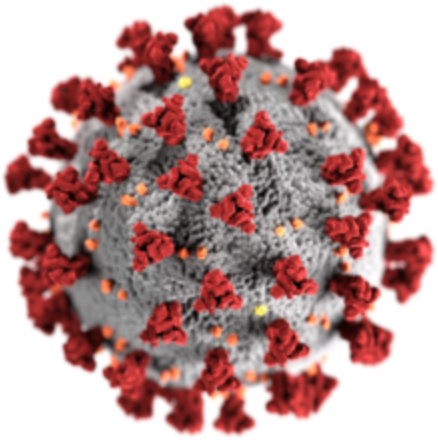


Illustration of the morphology of coronaviruses; the club-shaped viral spike peplomers (red) create the look of a corona surrounding the virion when seen with an electron microscope.

Virus classification

(unranked):	Virus
Realm:	*Riboviria*
Kingdom:	*Orthornavirae*
Phylum:	*Pisuviricota*
Class:	*Pisoniviricetes*
Order:	*Nidovirales*
Family:	*Coronaviridae*

Subfamily:	***Orthocoronavirinae***

Genera[1]

- *Alphacoronavirus*
- *Betacoronavirus*
- *Gammacoronavirus*
- *Deltacoronavirus*

Synonyms[2][3][4]

- *Coronavirinae*

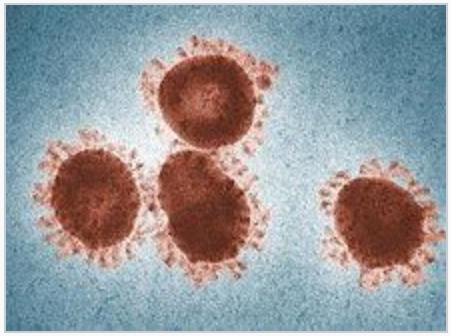
Coronaviruses are a group of related RNA viruses that cause diseases in mammals and birds. In humans, these viruses cause respiratory tract infections that can range from mild to lethal. Mild illnesses include some cases of the common cold (which is also caused by other viruses, predominantly rhinoviruses), while more lethal varieties can cause 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 as yet no 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*.[5][6] They are enveloped viruses with a positive-sense single-stranded RNA genome and a nucleocapsid of helical symmetry.[7] The genome size of coronaviruses ranges from approximately 26 to 32 kilobases, one of the largest among RNA viruses.[8] They have characteristic club-shaped spikes that project from their surface, which in electron micrographs create an image reminiscent of the solar corona, from which their name derives.[9]

Etymology

The name "coronavirus" is derived from Latin *corona*, meaning "crown" or "wreath", itself a borrowing from Greek κορώνη *korṓnē*, "garland, wreath".[10][11] The name was coined by June Almeida and David Tyrrell who first observed and studied human coronaviruses.[12] The word was first used in print in 1968 by an informal group of virologists in the journal *Nature* to designate the new family of viruses.[9] The name refers to the characteristic appearance of virions (the infective form of the virus) by electron microscopy, which have a fringe of large, bulbous surface projections creating an image reminiscent of the solar corona or halo.[9][12] This morphology is created by the viral spike peplomers, which are proteins on the surface of the virus.[13]

History

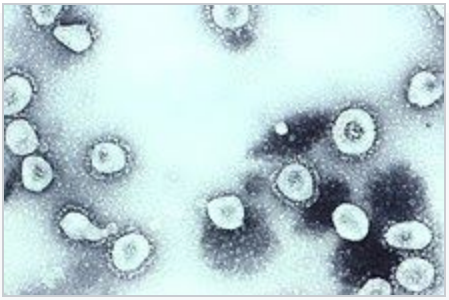


Transmission electron micrograph of infectious bronchitis viruses

Coronaviruses were first discovered in the 1930s when an acute respiratory infection of domesticated chickens was shown to be caused by infectious bronchitis virus (IBV).[14] Arthur Schalk and M.C. Hawn described in 1931 a new respiratory infection of chickens in North Dakota. The infection of new-born chicks was characterized by gasping and listlessness. The chicks' mortality rate was 40–90%.[15] Fred Beaudette and Charles Hudson six years later successfully isolated and cultivated the infectious bronchitis virus which caused the disease.[16] In the 1940s, two more animal coronaviruses, mouse hepatitis virus (MHV) and transmissible gastroenteritis virus (TGEV), were isolated.[17] It was not realized at the time that these three different viruses were related.[18]

Human coronaviruses were discovered in the 1960s.[19][20] They were isolated using two different methods in the United Kingdom and the United States.[21] E.C. Kendall, Malcom Byone, and David Tyrrell working at the Common Cold Unit of the British Medical Research Council in 1960 isolated from a boy a novel common cold virus B814.[22][23][24] The virus was not able to be cultivated using standard techniques which had successfully cultivated rhinoviruses, adenoviruses and other known common cold viruses. In 1965, Tyrrell and Byone successfully cultivated the novel virus by serially passing it through organ culture of human embryonic trachea.[25] The new cultivating method was

introduced to the lab by Bertil Hoorn.[26] The isolated virus when intranasally inoculated into volunteers caused a cold and was inactivated by ether which indicated it had a lipid envelope.[22][27] Around the same time, Dorothy Hamre[28] and John Procknow at the University of Chicago isolated a novel cold virus 229E from medical students, which they grew in kidney tissue culture. The novel virus 229E, like the virus strain B814, when inoculated into volunteers caused a cold and was inactivated by ether.[29]



Transmission electron micrograph of organ cultured coronavirus OC43

The two novel strains B814 and 229E were subsequently imaged by electron microscopy in 1967 by Scottish virologist June Almeida at St. Thomas Hospital in London.[30][31] Almeida through electron microscopy was able to show that B814 and 229E were morphologically related by their distinctive club-like spikes. Not only were they related with each other, but they were morphologically related to infectious bronchitis virus (IBV).[32] A research group at the National Institute of Health the same year was able to isolate another member of this new group of viruses using organ culture and named the virus strain OC43 (OC for organ culture).[33] Like B814, 229E, and IBV, the novel cold virus OC43 had distinctive cluMicrobiology

Structure

Cross-sectional model of a coronavirus

Cross-sectional model of a coronavirus

Coronaviruses are large, roughly spherical, particles with bulbous surface projections.[41] The average diameter of the virus particles is around 125 nm (.125 μm). The diameter of the envelope is 85 nm and the spikes are 20 nm long. The envelope of the virus in electron micrographs appears as a distinct pair of electron-dense shells (shells that are relatively opaque to the electron beam used to scan the virus particle).[42][43]

The viral envelope consists of a lipid bilayer, in which the membrane (M), envelope (E) and spike (S) structural proteins are anchored.[44] The ratio of E:S:M in the lipid bilayer is approximately 1:20:300.[45] On average a coronavirus particle has 74 surface spikes.[46] A subset of coronaviruses (specifically the members of betacoronavirus subgroup A) also have a shorter spike-like surface protein called hemagglutinin esterase (HE).[5]

The coronavirus surface spikes are homotrimers of the S protein, which is composed of an S1 and S2 subunit. The homotrimeric S protein is a class I fusion protein which mediates the receptor binding and membrane fusion between the virus and host cell. The S1 subunit forms the head of the spike and has the receptor binding domain (RBD). The S2 subunit forms the stem which anchors the spike in the viral envelope and on protease activation enables fusion. The E and M protein are important in forming the viral envelope and maintaining its structural shape.[43]

Inside the envelope, there is the nucleocapsid, which is formed from multiple copies of the nucleocapsid (N) protein, which are bound to the positive-sense single-stranded RNA genome in a continuous beads-on-a-string type conformation.[43][47] The lipid bilayer envelope, membrane proteins, and nucleocapsid protect the virus when it is outside the host cell.[48]

Genome

See also: Severe acute respiratory syndrome-related coronavirus § Genome

Schematic representation of the genome organization and functional domains of S protein for SARS-CoV and MERS-CoV

Coronaviruses contain a positive-sense, single-stranded RNA genome. The genome size for coronaviruses ranges from 26.4 to 31.7 kilobases.[8] The genome size is one of the largest among RNA viruses. The genome has a 5′ methylated cap and a 3′ polyadenylated tail.[43]

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 (pp1ab). The replicase-transcriptase polyprotein self cleaves to form 16 nonstructural proteins (nsp1–nsp16).[43]

The later reading frames encode the four major structural proteins: spike, envelope, membrane, and nucleocapsid.[49] 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.[43]

Replication cycle

Cell entry

The life cycle of a coronavirus

Infection begins when the viral spike protein 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.[50]

Genome translation

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 frames ORF1a and ORF1b of the virus genome into two large overlapping polyproteins, pp1a and pp1ab.[43]

SARS-CoV genome organization

The larger polyprotein pp1ab is a result of a -1 ribosomal frameshift caused by a slippery sequence (UUUAAAC) and a downstream RNA pseudoknot at the end of open reading frame ORF1a.[51] The ribosomal frameshift allows for the continuous translation of ORF1a followed by ORF1b.[43]

The polyproteins have their own proteases, PLpro and 3CLpro, which cleave the polyproteins at different specific sites. The cleavage of polyprotein pp1ab yields 16 nonstructural proteins (nsp1 to nsp16). Product proteins include various replication proteins such as RNA-dependent RNA polymerase (nsp12), RNA helicase (nsp13), and exoribonuclease (nsp14).[43]

Replicase-transcriptase

Replicase-transcriptase complex

A number of the nonstructural proteins coalesce to form a multi-protein replicase-transcriptase complex. 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 nonstructural protein, for instance, provides extra fidelity to replication by providing a proofreading function which the RNA-dependent RNA polymerase lacks.[52]

Replication – 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.[43]

Transcription of nested mRNAs

Nested set of subgenomic mRNAs

Transcription – 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 process is followed by the transcription of these negative-sense subgenomic RNA molecules to their corresponding positive-sense mRNAs.[43] The subgenomic mRNAs form a "nested set" which have a common 5'-head and partially duplicate 3'-end.[53]

Recombination – The replicase-transcriptase complex is also capable of genetic recombination when at least two viral genomes are present in the same infected cell.[53] RNA recombination appears to be a major driving force in determining genetic variability within a coronavirus species, the capability of a coronavirus species to jump from one host to another and, infrequently, in determining the emergence of novel coronaviruses.[54] The exact mechanism of recombination in coronaviruses is unclear, but likely involves template switching during genome replication.[54]

Assembly and 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.[43] 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. Progeny viruses are then released from the host cell by exocytosis through secretory vesicles. Once released the viruses can infect other host cells.[55]

Transmission

Infected carriers are able to shed viruses into the environment. The interaction of the coronavirus spike protein with its complementary cell receptor is central in determining the tissue tropism, infectivity, and species range of the released virus.[56][57] Coronaviruses mainly target epithelial cells.[5] They are transmitted from one host to another host, depending on the coronavirus species, by either an aerosol, fomite, or fecal-oral route.[58]

Human coronaviruses infect the epithelial cells of the respiratory tract, while animal coronaviruses generally infect the epithelial cells of the digestive tract.[5] SARS coronavirus, for example, infects via an aerosol route,[59] the human epithelial cells of the lungs by binding to the angiotensin-converting enzyme 2 (ACE2) receptor.[60] Transmissible gastroenteritis coronavirus

(TGEV) infects, via a fecal-oral route,[58] the pig epithelial cells of the digestive tract by binding to the alanine aminopeptidase (APN) receptor.[43]

Classification

For a more detailed list of members, see Coronaviridae.

Phylogenetic tree of coronaviruses

The scientific name for coronavirus is Orthocoronavirinae or Coronavirinae.[2][3][4] Coronaviruses belong to the family of Coronaviridae, order Nidovirales, and realm Riboviria.[5][6] They are divided into alphacoronaviruses and betacoronaviruses which infect mammals – and gammacoronaviruses and deltacoronaviruses, which primarily infect birds.[61][62]

Genus: Alphacoronavirus;[58] type species: Alphacoronavirus 1 (TGEV)

Species: Alphacoronavirus 1, Human coronavirus 229E, Human coronavirus NL63, Miniopterus bat coronavirus 1, Miniopterus bat coronavirus HKU8, Porcine epidemic diarrhea virus, Rhinolophus bat coronavirus HKU2, Scotophilus bat coronavirus 512

Genus Betacoronavirus;[59] type species: Murine coronavirus (MHV)

Species: Betacoronavirus 1 (Bovine Coronavirus, Human coronavirus OC43), Hedgehog coronavirus 1, Human coronavirus HKU1, Middle East respiratory syndrome-related coronavirus, Murine coronavirus, Pipistrellus bat coronavirus HKU5, Rousettus bat coronavirus HKU9, Severe acute respiratory syndrome-related coronavirus (SARS-CoV, SARS-CoV-2), Tylonycteris bat coronavirus HKU4

Genus Gammacoronavirus;[16] type species: Avian coronavirus (IBV)

Species: Avian coronavirus, Beluga whale coronavirus SW1

Genus Deltacoronavirus; type species: Bulbul coronavirus HKU11

Species: Bulbul coronavirus HKU11, Porcine coronavirus HKU15

Origin

Origins of human coronaviruses with possible intermediate hosts

The most recent common ancestor (MRCA) of all coronaviruses is estimated to have existed as recently as 8000 BCE, although some models place the common ancestor as far back as 55 million years or more, implying long term coevolution with bat and avian species.[63] The most recent common ancestor of the alphacoronavirus line has been placed at about 2400 BCE, of the betacoronavirus line at 3300 BCE, of the gammacoronavirus line at 2800 BCE, and of the deltacoronavirus line at about 3000 BCE. Bats and birds, as warm-blooded flying vertebrates, are an ideal natural reservoir for the coronavirus gene pool (with bats the reservoir for alphacoronaviruses and betacoronavirus – and birds the reservoir for gammacoronaviruses and deltacoronaviruses).

The large number and global range of bat and avian species that host viruses has enabled extensive evolution and dissemination of coronaviruses.[64]

Many human coronaviruses have their origin in bats.[65] The human coronavirus NL63 shared a common ancestor with a bat coronavirus (ARCoV.2) between 1190 and 1449 CE.[66] The human coronavirus 229E shared a common ancestor with a bat coronavirus (GhanaGrp1 Bt CoV) between 1686 and 1800 CE.[67] More recently, alpaca coronavirus and human coronavirus 229E diverged sometime before 1960.[68] MERS-CoV emerged in humans from bats through the intermediate host of camels.[69] MERS-CoV, although related to several bat coronavirus species, appears to have diverged from these several centuries ago.[70] The most closely related bat coronavirus and SARS-CoV diverged in 1986.[71] A possible path of evolution of SARS coronavirus and keen bat coronaviruses is that SARS-related coronaviruses coevolved in bats for a long time. The ancestors of SARS-CoV first infected leaf-nose bats of the genus Hipposideridae; subsequently, they spread to horseshoe bats in the species Rhinolophidae, then to Asian palm civets, and finally to humans.[72][73]

Unlike other betacoronaviruses, bovine coronavirus of the species Betacoronavirus 1 and subgenus Embecovirus is thought to have originated in rodents and not in bats.[65][74] In the 1790s, equine coronavirus diverged from the bovine coronavirus after a cross-species jump.[75] Later in the 1890s, human coronavirus OC43 diverged from bovine coronavirus after another cross-species spillover event.[76][75] It is speculated that the flu pandemic of 1890 may have been caused by this spillover event, and not by the influenza virus, because of the related timing, neurological symptoms, and unknown causative agent of the pandemic.[77] Besides causing respiratory infections, human coronavirus OC43 is also suspected of playing a role in neurological diseases.[78] In the 1950s, the human coronavirus OC43 began to diverge into its present genotypes.[79] Phylogentically, mouse hepatitis virus (Murine coronavirus), which infects the mouse's liver and central nervous system,[80] is related to human coronavirus OC43 and bovine coronavirus. Human coronavirus HKU1, like the aforementioned viruses, also has its origins in rodents.[65]

Infection in humans

Illustration of SARSr-CoV virion

Coronaviruses vary significantly in risk factor. Some can kill more than 30% of those infected, such as MERS-CoV, and some are relatively harmless, such as the common cold.[43] Coronaviruses can cause colds with major symptoms, such as fever, and a sore throat from swollen adenoids.[81] Coronaviruses can cause pneumonia (either direct viral pneumonia or secondary bacterial pneumonia) and bronchitis (either direct viral bronchitis or secondary bacterial bronchitis).[82] The human coronavirus discovered in 2003, SARS-CoV, which causes severe acute respiratory syndrome (SARS), has a unique pathogenesis because it causes both upper and lower respiratory tract infections.[82]

Six species of human coronaviruses are known, with one species subdivided into two different strains, making seven strains of human coronaviruses altogether.

Seasonal distribution of HCoV-NL63 in Germany shows a preferential detection from November to March

Four human coronaviruses produce symptoms that are generally mild:

Human coronavirus OC43 (HCoV-OC43), β-CoV

Human coronavirus HKU1 (HCoV-HKU1), β-CoV

Human coronavirus 229E (HCoV-229E), α-CoV

Human coronavirus NL63 (HCoV-NL63), α-CoV

Three human coronaviruses produce symptoms that are potentially severe:

Middle East respiratory syndrome-related coronavirus (MERS-CoV), β-CoV

Severe acute respiratory syndrome coronavirus (SARS-CoV), β-CoV

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), β-CoV

Common cold

Main article: Common cold

The human coronaviruses HCoV-OC43, HCoV-HKU1, HCoV-229E, and HCoV-NL63 continually circulate in the human population and produce the generally mild symptoms of the common cold in adults and children worldwide.[83] These coronaviruses cause about 15% of common colds,[84] while 40 to 50% of colds are caused by rhinoviruses.[85] The four mild coronaviruses have a seasonal incidence occurring in the winter months in temperate climates.[86][87] There is no preponderance in any season in tropical climates.[88]

Severe acute respiratory syndrome (SARS)

Main article: Severe acute respiratory syndrome

Characteristics of zoonotic coronavirus strains

MERS-CoV, SARS-CoV, SARS-CoV-2,

and related diseases

MERS-CoV	SARS-CoV	SARS-CoV-2
Disease	MERS	SARS	COVID-19
Outbreaks	2012, 2015,		

2018 2002–2004 2019–2020

pandemic

Epidemiology

Date of first

identified case June

2012 November

2002 December

2019[89]

Location of first

identified case Jeddah,

Saudi Arabia Shunde,

China Wuhan,

China

Age average 56 44[90][a] 56[91]

Sex ratio (M:F) 3.3:1 0.8:1[92] 1.6:1[91]

Confirmed cases 2494 8096[93] 6,632,985[94][b]

Deaths 858 774[93] 391,136[94][b]

Case fatality rate 37% 9.2% 5.9%[94]

Symptoms

Fever 98% 99–100% 87.9%[95]

Dry cough 47% 29–75% 67.7%[95]

Dyspnea 72% 40–42% 18.6%[95]

Diarrhea 26% 20–25% 3.7%[95]

Sore throat 21% 13–25% 13.9%[95]

Ventilatory use 24.5%[96] 14–20% 4.1%[97]

Notes

Based on data from Hong Kong.

Data as of 5 June 2020.

vte

In 2003, following the outbreak of severe acute respiratory syndrome (SARS) which had begun the prior year in Asia, and secondary cases elsewhere in the world, the World Health Organization (WHO) issued a press release stating that a novel coronavirus identified by a number of laboratories was the causative agent for SARS. The virus was officially named the SARS coronavirus (SARS-CoV). More than 8,000 people were infected, about ten percent of whom died.[60]

Middle East respiratory syndrome (MERS)

Main article: Middle East respiratory syndrome

In September 2012, a new type of coronavirus was identified, initially called Novel Coronavirus 2012, and now officially named Middle East respiratory syndrome coronavirus (MERS-CoV).[98][99] The World Health Organization issued a global alert soon after.[100] The WHO update on 28 September 2012 said the virus did not seem to pass easily from person to person.[101] However, on 12 May 2013, a case of human-to-human transmission in France was confirmed by the French Ministry of Social Affairs and Health.[102] In addition, cases of human-to-human transmission were reported by the Ministry of Health in Tunisia. Two confirmed cases involved people who seemed to have caught the disease from their late father, who became ill after a visit to Qatar and Saudi Arabia. Despite this, it appears the virus had trouble spreading from human to human, as most individuals who are infected do not transmit the virus.[103] By 30 October 2013, there were 124 cases and 52 deaths in Saudi Arabia.[104]

After the Dutch Erasmus Medical Centre sequenced the virus, the virus was given a new name, Human Coronavirus—Erasmus Medical Centre (HCoV-EMC). The final name for the virus is Middle East respiratory syndrome coronavirus (MERS-CoV). The only U.S. cases (both survived) were recorded in May 2014.[105]

In May 2015, an outbreak of MERS-CoV occurred in the Republic of Korea, when a man who had traveled to the Middle East, visited four hospitals in the Seoul area to treat his illness. This caused one of the largest outbreaks of MERS-CoV outside the Middle East.[106] As of December 2019, 2,468 cases of MERS-CoV infection had been confirmed by laboratory tests, 851 of which were fatal, a mortality rate of approximately 34.5%.[107]

Coronavirus disease 2019 (COVID-19)

Main article: Coronavirus disease 2019

In December 2019, a pneumonia outbreak was reported in Wuhan, China.[108] On 31 December 2019, the outbreak was traced to a novel strain of coronavirus,[109] which was given the interim name 2019-nCoV by the World Health Organization (WHO),[110][111][112] later renamed SARS-CoV-2 by the International Committee on Taxonomy of Viruses.

As of 5 June 2020, there have been at least 391,136[94] confirmed deaths and more than 6,632,985[94] confirmed cases in the COVID-19 pandemic. The Wuhan strain has been identified as a new strain of Betacoronavirus from group 2B with approximately 70% genetic similarity to the SARS-CoV.[113] The virus has a 96% similarity to a bat coronavirus, so it is widely suspected to originate from bats as well.[114][115] The pandemic has resulted in travel restrictions and nationwide lockdowns in many countries.

Infection in animals

Coronaviruses have been recognized as causing pathological conditions in veterinary medicine since the 1930s.[17] They infect a range of animals including swine, cattle, horses, camels, cats, dogs, rodents, birds and bats.[116] The majority of animal related coronaviruses infect the intestinal tract and are transmitted by a fecal-oral route.[117] Significant research efforts have been focused on elucidating the viral pathogenesis of these animal coronaviruses, especially by virologists interested in veterinary and zoonotic diseases.[118]

Farm animals

Infectious bronchitis virus (IBV) causes avian infectious bronchitis.[119] The virus is of concern to the poultry industry because of the high mortality from infection, its rapid spread, and affect on production.[116] The virus affects both meat production and egg production and causes substantial economic loss.[120] In chickens, infectious bronchitis virus targets not only the respiratory tract but also the urogenital tract. The virus can spread to different organs throughout the chicken.[119] The virus is transmitted by aersol and food contaminated by feces. Different vaccines against IBV exist and have helped to limit the spread of the virus and its variants.[116] Infectious bronchitis virus is one of a number of strains of the species Avian coronavirus.[121] Another strain of avian coronavirus is turkey coronavirus (TCV) which causes enteritis in turkeys.[116]

Coronaviruses also affect other branches of animal husbandry such as pig farming and the cattle raising.[116] Swine acute diarrhea syndrome coronavirus (SADS-CoV), which is related to bat coronavirus HKU2, causes diarrhea in pigs.[122] Porcine epidemic diarrhea virus (PEDV) has emerged around the world and similarly causes diarrhea in pigs.[123] Transmissible gastroenteritis virus (TGEV) is a coronavirus which causes diarrhea in young pigs.[124][125] In the cattle industry, bovine coronavirus (BCV) is responsible for severe profuse enteritis in young calves.[116]

Domestic pets

There are two forms of feline coronavirus. Feline enteric coronavirus is a pathogen of minor clinical significance, but spontaneous mutation of this virus can result in feline infectious peritonitis (FIP), a disease with high mortality.

There are two types of canine coronavirus (CCoV), one that causes mild gastrointestinal disease and one that has been found to cause respiratory disease. Pantropic canine coronavirus.[clarification needed]

There are two types of coronavirus that infect ferrets: Ferret enteric coronavirus causes a gastrointestinal syndrome known as epizootic catarrhal enteritis (ECE), and a more lethal systemic version of the virus (like FIP in cats) known as ferret systemic coronavirus (FSC).[126][127]

Labratory animals

Mouse hepatitis virus (MHV) is a coronavirus that causes an epidemic murine illness with high mortality, especially among colonies of laboratory mice.[128] Prior to the discovery of SARS-CoV, MHV was the best-studied coronavirus both in vivo and in vitro as well as at the molecular level. Some strains of MHV cause a progressive demyelinating encephalitis in mice which has been used as a murine model for multiple sclerosis.[118]

Sialodacryoadenitis virus (SDAV) is highly infectious coronavirus of laboratory rats, which can be transmitted between individuals by direct contact and indirectly by aerosol. Acute infections have high morbidity and tropism for the salivary, lachrymal and harderian glands.[129]

Rabbit enteric coronavirus causes acute gastrointestinal disease and diarrhea in young European rabbits. Mortality rates are high.[130]

Prevention and treatment

There are no vaccines or antiviral drugs to prevent or treat human coronavirus infections. Treatment is only supportive. A number of antivirial targets have been identified such as viral proteases, polymerases, and entry proteins. Drugs are in development which target these proteins and the different steps of viral replication. A number of vaccines using different methods are also under development for different human coronaviruses.[43]

There are no antiviral drugs to treat animal coronaviruses. Vaccines are available for IBV, TGEV, and Canine CoV, although their effectiveness is limited. In the case of outbreaks of highly contagious animal coronaviruses, such as PEDV, measures such as destruction of entire herds of pigs may be used to prevent transmission to other herds.[43]

See also

Bat-borne virus

Zoonosis

SARS-CoV-2 (Wikimedia colors).svgCoronavirus disease 2019 portalWHO Rod.svgMedicine portalSida-aids.pngViruses portal

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Nextstrain, phylogenetic tree of Beta-CoV

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The IBV-like novel cold viruses were soon shown to be also morphologically related to the mouse hepatitis virus.[17] This new group of IBV-like viruses came to be known as coronaviruses after their distinctive morphological appearance.[9] Human coronavirus 229E and human coronavirus OC43 continued to be studied in subsequent decades.[36][37] The coronavirus strain B814 was lost. It is not known which present human coronavirus it was.[38] Other human coronaviruses have since been identified, including SARS-CoV in 2003, HCoV NL63 in 2004, HCoV HKU1 in 2005, MERS-CoV in 2012, and SARS-CoV-2 in 2019.[39][40] There have also been a large number of animal coronaviruses identified since the 1960s.[5]