An official EU website

European Centre for Disease Prevention and Control

Coronaviruses

General background

(Last update 08/09/2020)

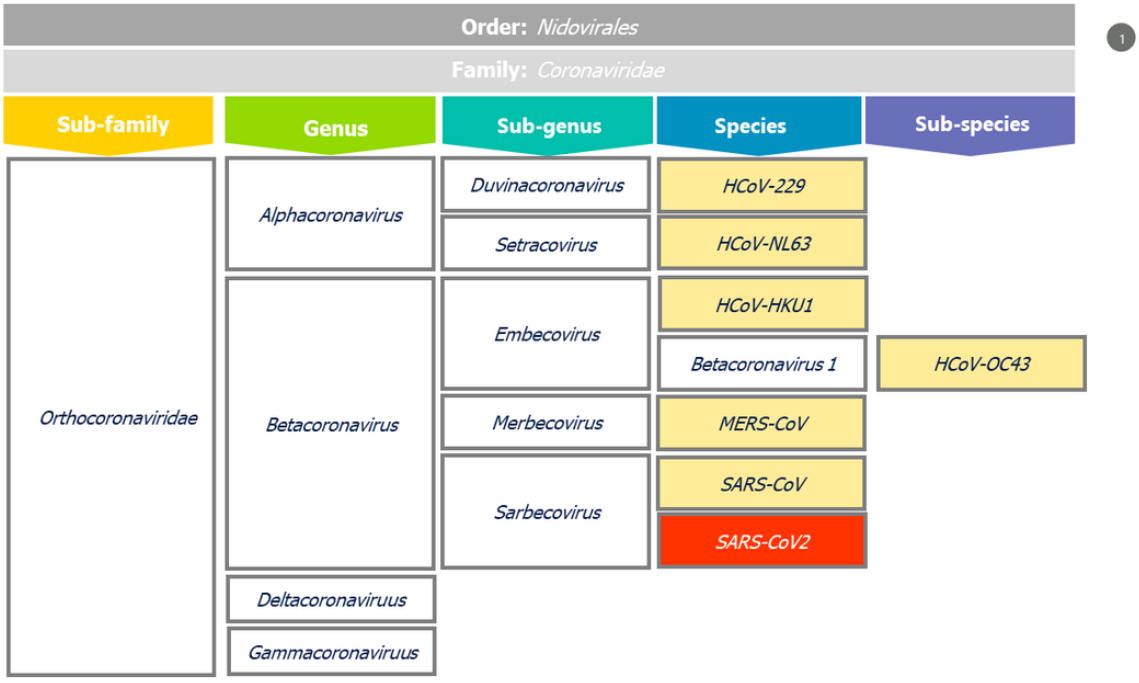
Coronaviruses (CoV) were identified as human pathogens in the 1960s. They are enveloped positive stranded RNA viruses in the order of *Nidovirales* (Figure) [1]. With their characteristic surface, the virions have a crown-like appearance under the electron microscope, which is why the viruses are named after the Latin word corona, meaning 'crown' or 'halo'.

Most coronaviruses infect animals, (i.e., bats, birds and mammals), which act as an intermediate host reservoir. Sometimes they change host and infect humans.

There are currently seven coronaviruses known to infect humans (Figure). Four of them cause mild to moderate disease. More specifically, HCoV-OC43, HCoV-HKU1 and HCoV-229E cause common colds, and severe lower respiratory tract infections in the youngest and oldest age groups, while HCoV-NL63 is an important cause of (pseudo) croup and bronchiolitis in children [2]. The other three cause more severe and even fatal disease and have emerged more recently (in the last 20 years): SARS-CoV responsible for the Severe Acute Respiratory Syndrome (SARS) in 2002, MERS-CoV the Middle East Respiratory Syndrome (MERS) in 2012 and SARS-CoV-2, identified with a cluster of pneumonia cases in Wuhan, China in late 2019 [3,4].

Illness in humans mostly affects the respiratory tract, with symptoms ranging from those of a common cold to very severe lower respiratory infections [5].

Figure: Human coronavirus taxonomy



The human coronavirus are depicted in yellow and SARS-CoV-2 responsible for COVID-19 in red. Source: based on the International Committee on Taxonomy of Viruses (ICTV)

SARS-CoV-2 virus evolution

(Last update 03/09/2020)

At the end of June 2020, the GISAID EpiCoV database held more than 57 000 genome sequences (www.gisaid.org (http://www.gisaid.org/)) of SARS-CoV-2.

A meta-analysis of time estimates to the last common ancestor of the virus suggested that the pandemic started sometime between 6 October and 11 December 2019 [6]. Retrospective analysis of sewage samples from Milan and Turin showed that the virus was already present in northern Italy on 18 December 2019 [7].

Three different time-calibrated phylogenetic analysis of closely related coronaviruses suggested that the lineage giving rise to SARS-CoV-2 diverged from the most similar known bat coronavirus between 1948 and 1982 [8]. Bats are therefore the most likely original animal reservoir of the virus, with an intermediate animal host involved in the transmission to humans [9-11]. However, the recombinant nature of coronaviruses complicates longer-term phylogenetic analysis. Genomic evidence also indicates it is unlikely that the virus is a product of in-vitro manipulation, passaging in cell-culture, or that it is of synthetic origin [12,13].

No evidence currently supports that mutations accumulated since the introduction of the SARS-CoV-2 virus in the human population have caused increased disease severity. In contrast, a variant with a 382-nucleotide deletion circulating in Singapore from January to March 2020 was associated with reduced disease severity [14], but this variant has not been found in Singapore or elsewhere since. There is, however, some evidence indicating that the variant of the virus carrying a mutation (substitution from aspartate to glycine at position 614) in the spike glycoprotein but not located in the receptor-binding domain of the protein could affect the transmissibility of the virus. Researchers are still debating the effect of this mutation [15-18]. Studies of viral cell entry with spike glycoprotein position 614 variants showed that the glycine substitution is associated with an increased rate of cell entry by 2.2 to 7.7-fold depending on the experimental setting [19-21]. The frequency of reporting viral sequences carrying this mutation has increased in the GISAID database over time, with sequences reported from all parts of the world.

Mutations in the receptor-binding domain of the spike glycoprotein are of particular interest, as they may affect infectivity and host-specificity [22]. Some mutations in this domain have been reported but so far rare and are not present in any of the major SARS-CoV-2 clades [23]. Other mutations of potential interest are those that have occurred independently several times, but preliminary findings showed that none of these mutations provided any fitness advantage [24].

Mutations in primer binding sites used for reverse transcription polymerase chain reaction (RT-PCR) detection assays have significant implications for accurate testing but, have been rare so far. These mutations are shown in the ECDC Primerscan tool (https://primerscan.ecdc.europa.eu/ (https://primerscan.ecdc.europa.eu/)) [25]. Issues with potential design flaws of a few publicly available primer- and probe sets have recently been reported [26].

SARS-CoV-2 virus seasonality

(Last update: 08/09/2020)

Human coronavirus infection rates show peaks in the winter months, similar to influenza and human respiratory syncytial viruses (RSV) and are therefore, according to their seasonality, classified as winter viruses [27]. Low temperature and dry air impair and disrupt the integrity of the epithelial layer of the lungs, which might explain the winter seasonality of respiratory viruses [27].. Other factors that might contribute to transmission are increased indoor activities during the winter months, which increases susceptible host proximity. Such behavioural factors have been implicated in other winter viruses such as influenza [28,29]. Since SARS-CoV-2 is also a human coronavirus it can be presumed to be a winter virus.

A cohort study of cities with substantial spread of COVID-19 from around the world examined the relationship between temperature and humidity on the epidemiology of COVID-19 [30]. These cities displayed consistently similar weather patterns, with mean temperatures of between 5 and 11°C, combined with low specific humidity and low absolute humidity [30]. An analysis from Spain supported the hypothesis that COVID-19 incidence is lower at higher temperatures and higher levels of humidity [31]. A study from China, Hong Kong, and Singapore, found that high temperature mitigated the transmission of COVID-19 [32].

However, in temperate climate, (moderately) higher temperature might not necessarily reduce transmission, as observed this summer in many European countries. . An analysis of transmission in four major provinces in Canada between January and May 2020 did not find a significant association between ambient temperature and transmission [33]. It is possible that during the winter and spring season, the threshold was not reached at which the effects of temperature on viral activity would be detected. These findings were consistent with a Spanish study of temperature and COVID-19 transmission where lower seasonal temperatures did presumably not reach the (higher) temperature threshold needed to affect COVID-19 transmission [34].

Modelling the SARS-CoV-2 transmission dynamic based on other human coronaviruses suggests that effective reproductiveness can drop from winter to summer by 20% but can still generate substantial outbreaks (R0>1) if no control measures are in place [35]

References

Supporting document: List of references ▸ (https://www.ecdc.europa.eu/sites/default/files/documents/References-coronaviruses-11-September.pdf)

Coronavirus (/en/search?f%5B0%5D=diseases%3A2943) | COVID-19 (/en/search?f%5B0%5D=diseases%3A2942) | Public health threat (/en/search?f%5B0%5D=public_health_areas%3A1583)

Page last updated 11 Sep 2020

© European Centre for Disease Prevention and Control (ECDC) 2021