



**Songbirds are mysteriously dying across the eastern U.S. Scientists are scrambling to find out why** , 06 Jul 2021

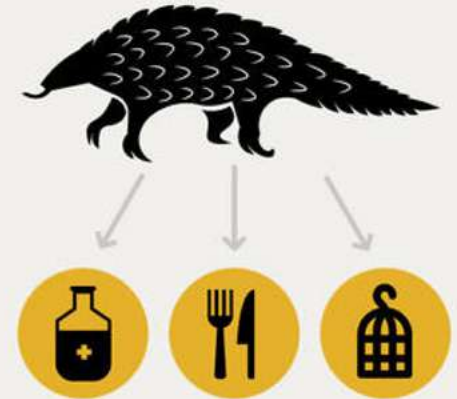
## HOW WILDLIFE MARKETS SPREAD DISEASE

# WHERE DID THE COVID-19 CORONAVIRUS COME FROM?

The spread of zoonotic diseases – those pathogens that jump from the species that it evolved with to a new host – is exacerbated by wildlife trafficking, habitat destruction and climate change. These threats drive humans and animals closer together. Coronavirus is just one example of a string of pathogens that has come from wildlife trafficking, including SARS, Ebola, Bird Flu, and more. | #COVID19

### 1. WILDLIFE TRAFFICKING & POACHING

Animals are hunted, trapped and taken to markets to be sold for traditional medicine, food and the pet trade.



GLOBAL WILDLIFE CONSERVATION



WCS



### 2. RESERVOIRS OF DISEASE

Wild animals that appear healthy can harbor diseases that can make other animals, including humans, sick. When animals are forced into markets, they can spread disease.

### 3. PATHOGEN EXCHANGE



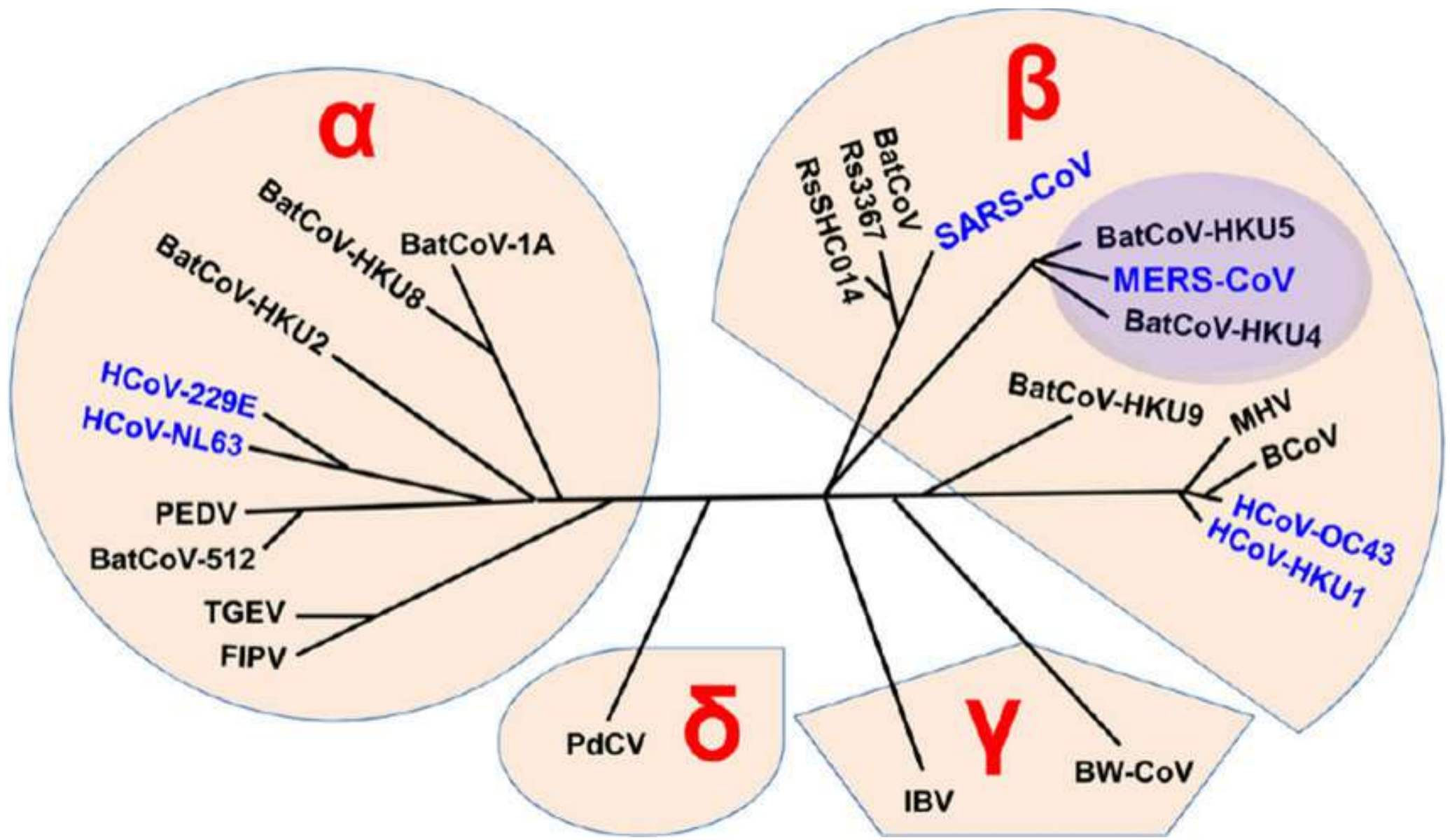
It's only when humans hunt wildlife or destroy their homes that these viruses and other pathogens jump species. We must combat trafficking of wild animals and change dangerous wildlife consumption behaviors, especially in cities.

## STOP THE SPREAD

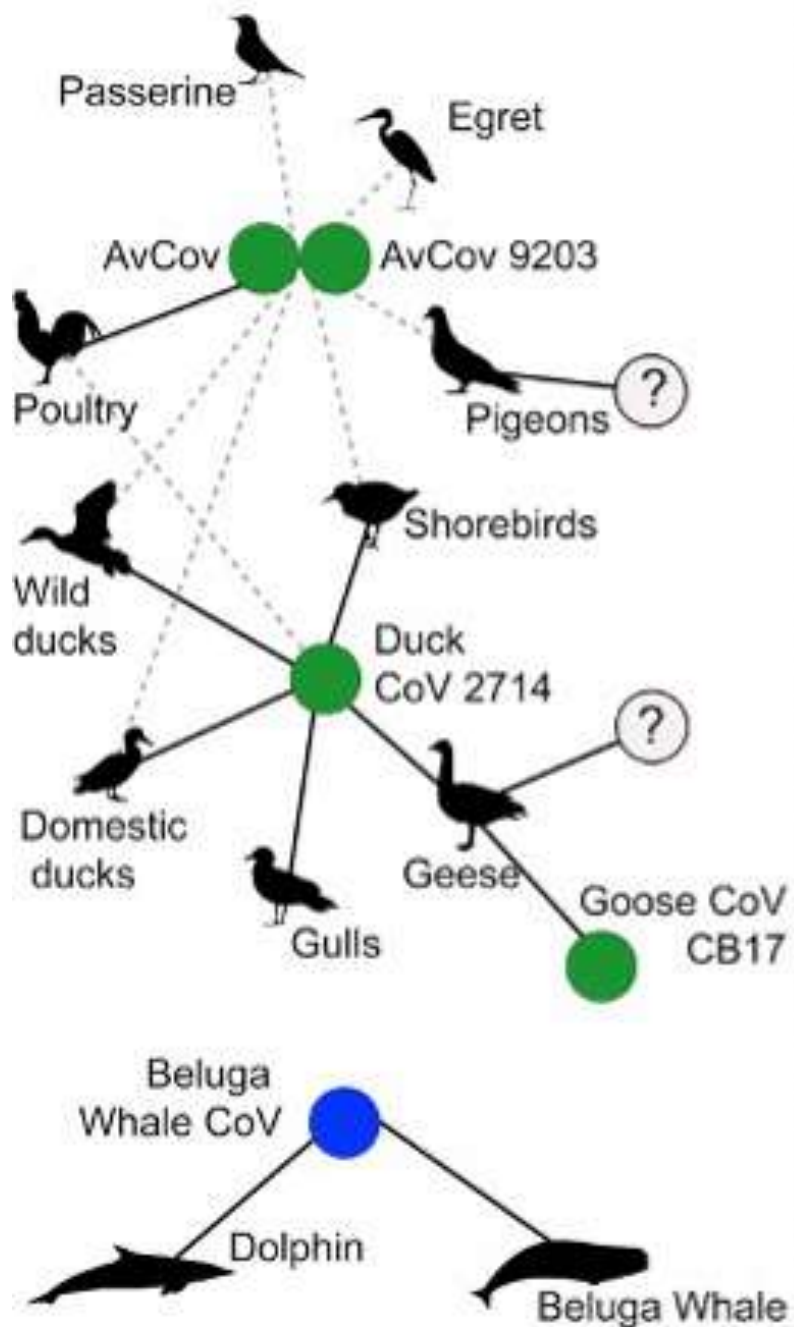


Ban live animal markets that trade in wildlife. Stop illegal trafficking and poaching of wild animals. Not only will this help prevent the spread of disease, it will address one of the major drivers of species extinction.

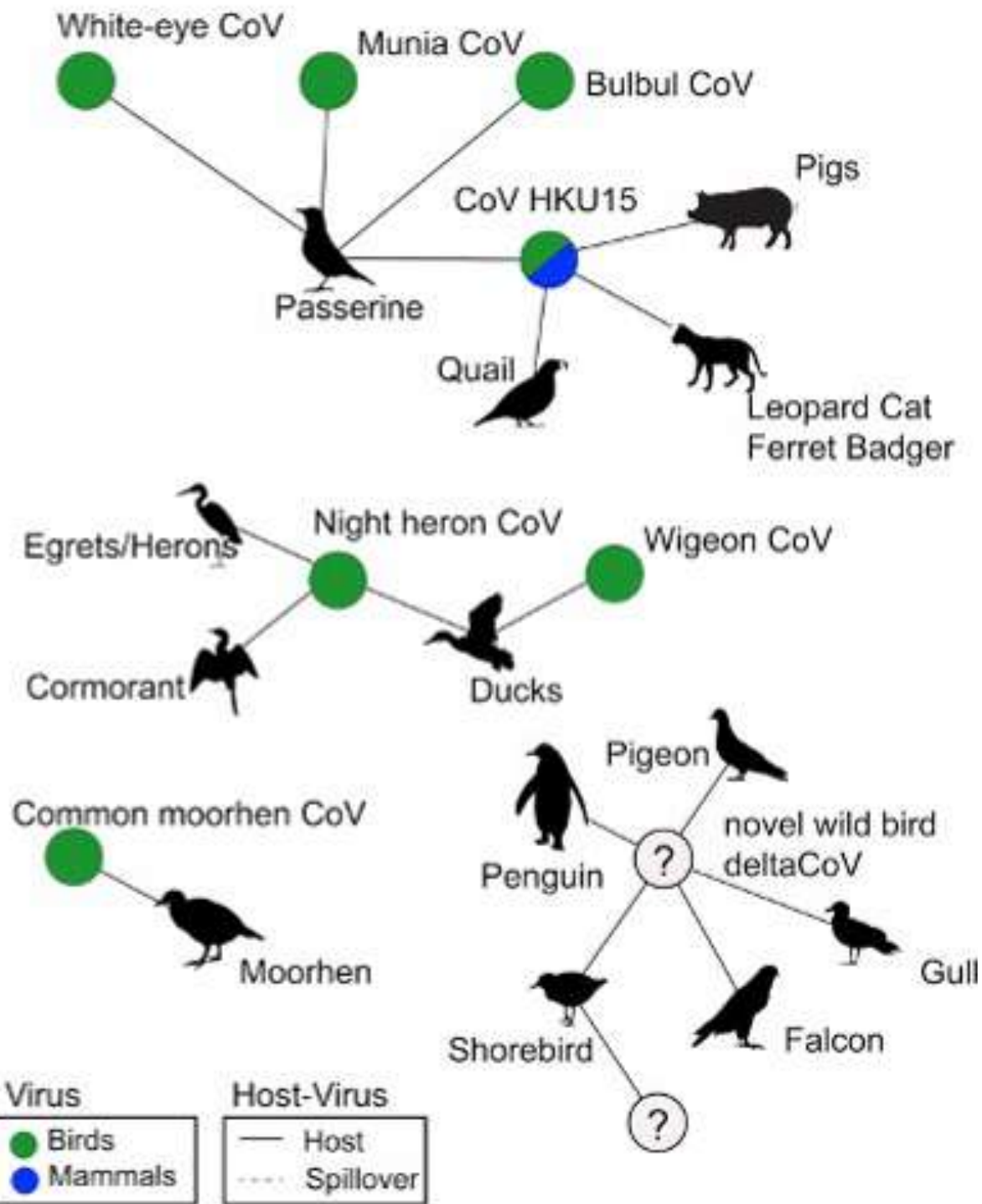




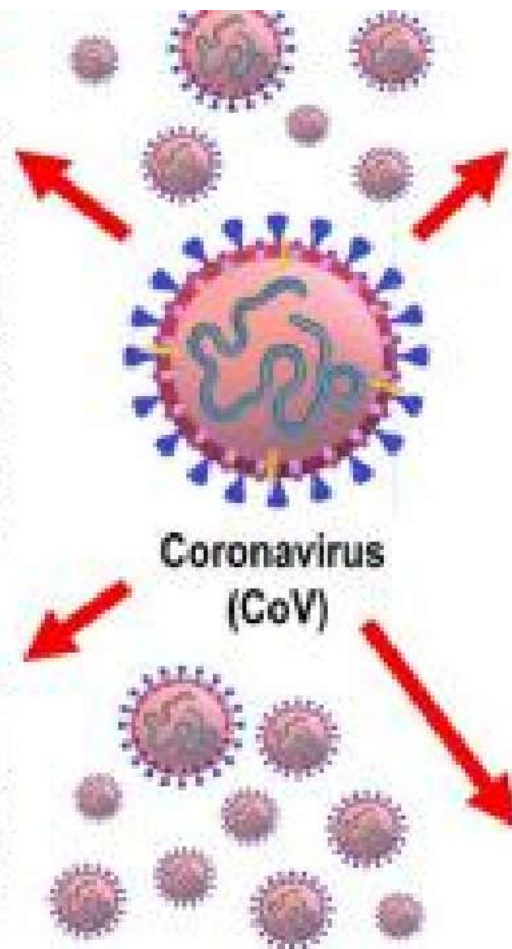
## Gammacoronavirus




## Deltacoronavirus
















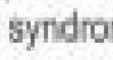








**Alphacoronavirus**

-  Canine coronavirus
-  Feline coronavirus
-  Bat coronavirus HKU2
-  Bat coronavirus HKU8
-  Porcine respiratory coronavirus
-  Porcine epidemic diarrhoea virus
-  Transmissible gastroenteritis virus
-  Human coronavirus 229E
-  Human coronavirus NL63

**Deltacoronavirus**

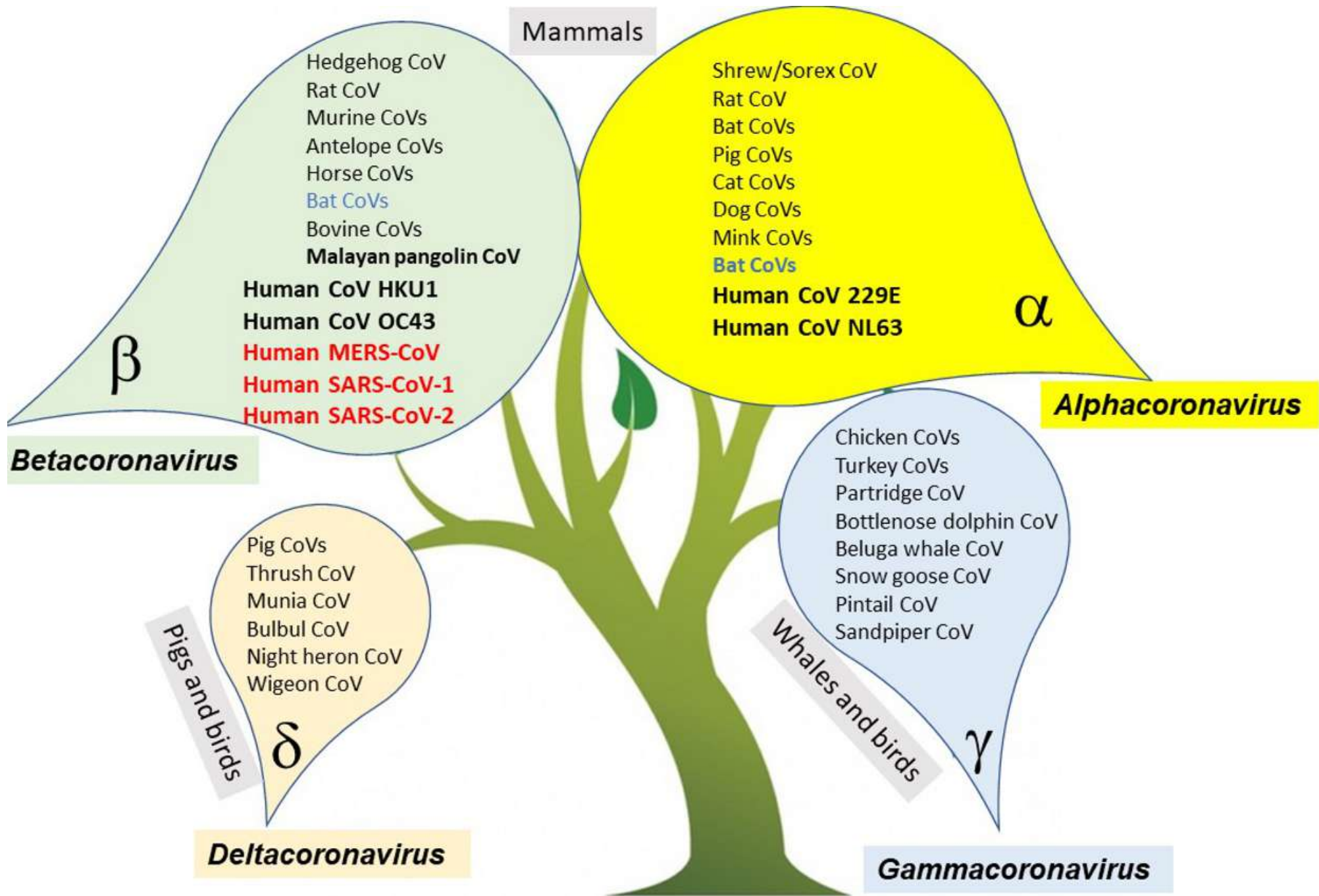
-  Bulbul coronavirus HKU11
-  Thrush coronavirus HKU12
-  Muntia coronavirus HKU13
-  Porcine coronavirus HKU15

**Betacoronavirus**

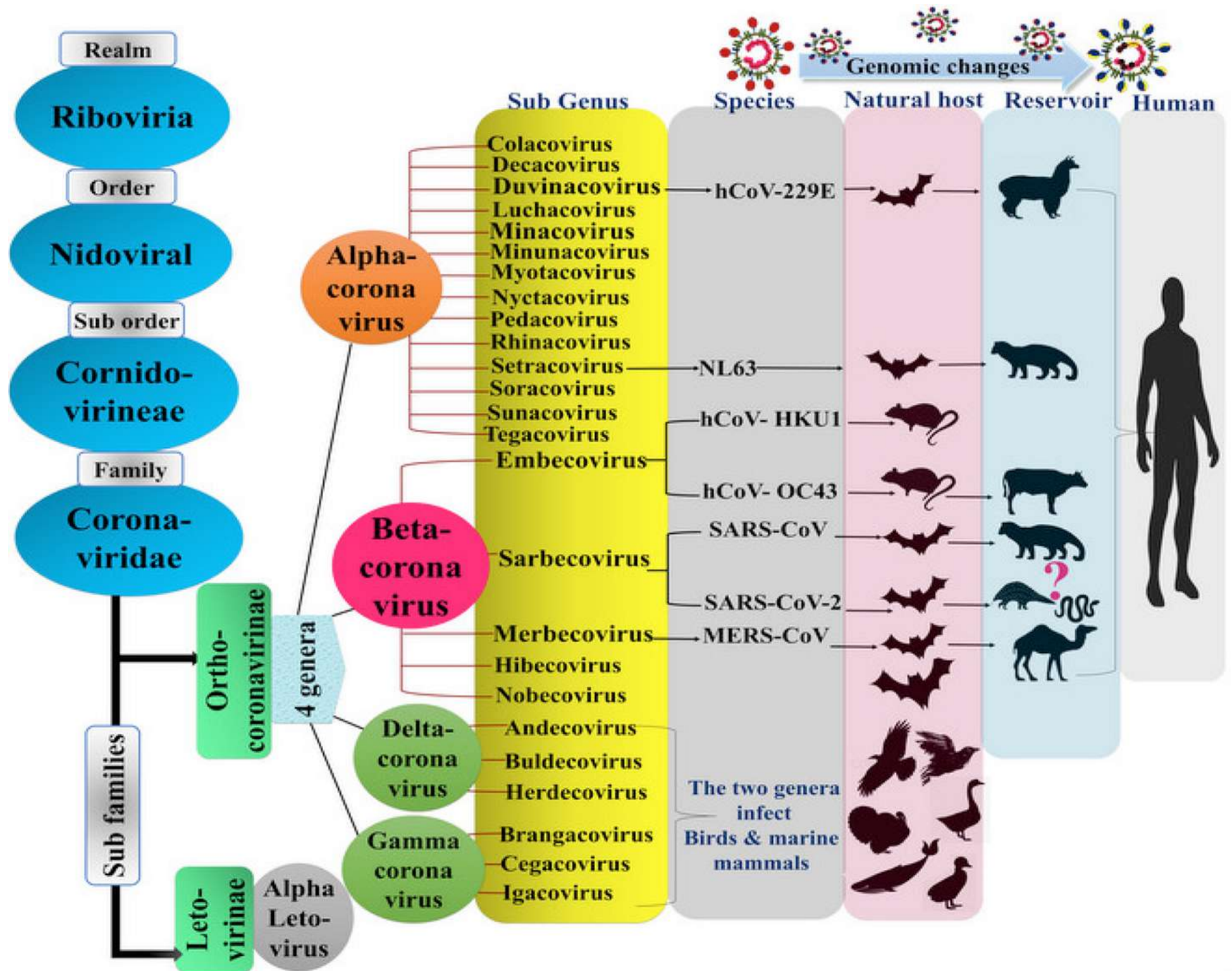
-  Human coronavirus OC43
-  Human coronavirus HKU1
-  Murine coronavirus
-  Bat coronavirus HKU4
-  Bat coronavirus HKU5
-  Bat coronavirus HKU9
-  Severe acute respiratory syndrome CoV (SARS-CoV)
-  Middle East respiratory syndrome CoV (MERS-CoV)
-  Severe acute respiratory syndrome CoV-2 (SARS-CoV-2)
-  Porcine haemagglutinating encephalomyelitis virus
-  Hedgehog coronavirus
-  Bovine coronavirus
-  Canine respiratory coronavirus
-  Equine coronavirus

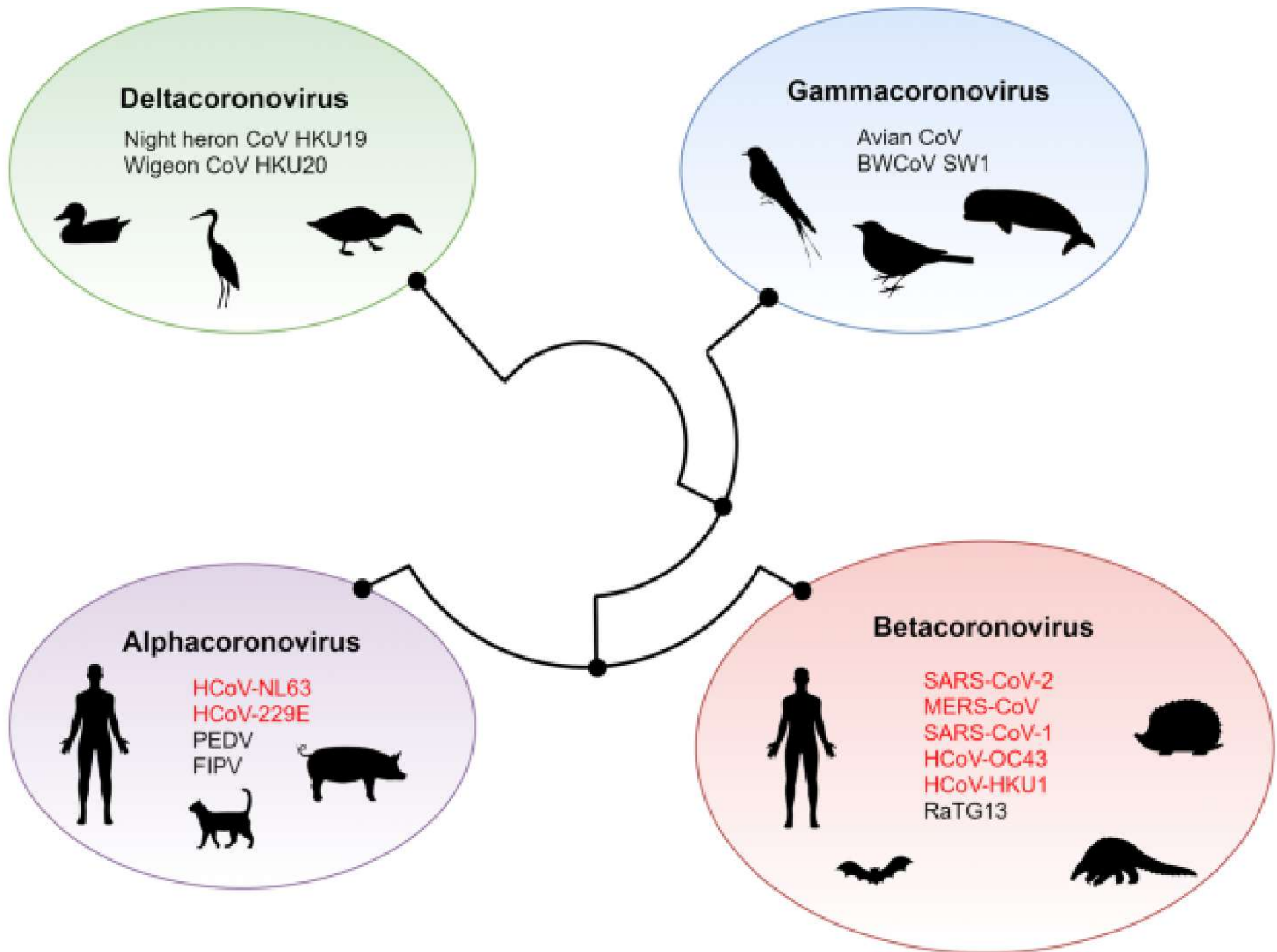
**Gammacoronavirus**

-  Infectious bronchitis virus
-  Beluga whale coronavirus SW1





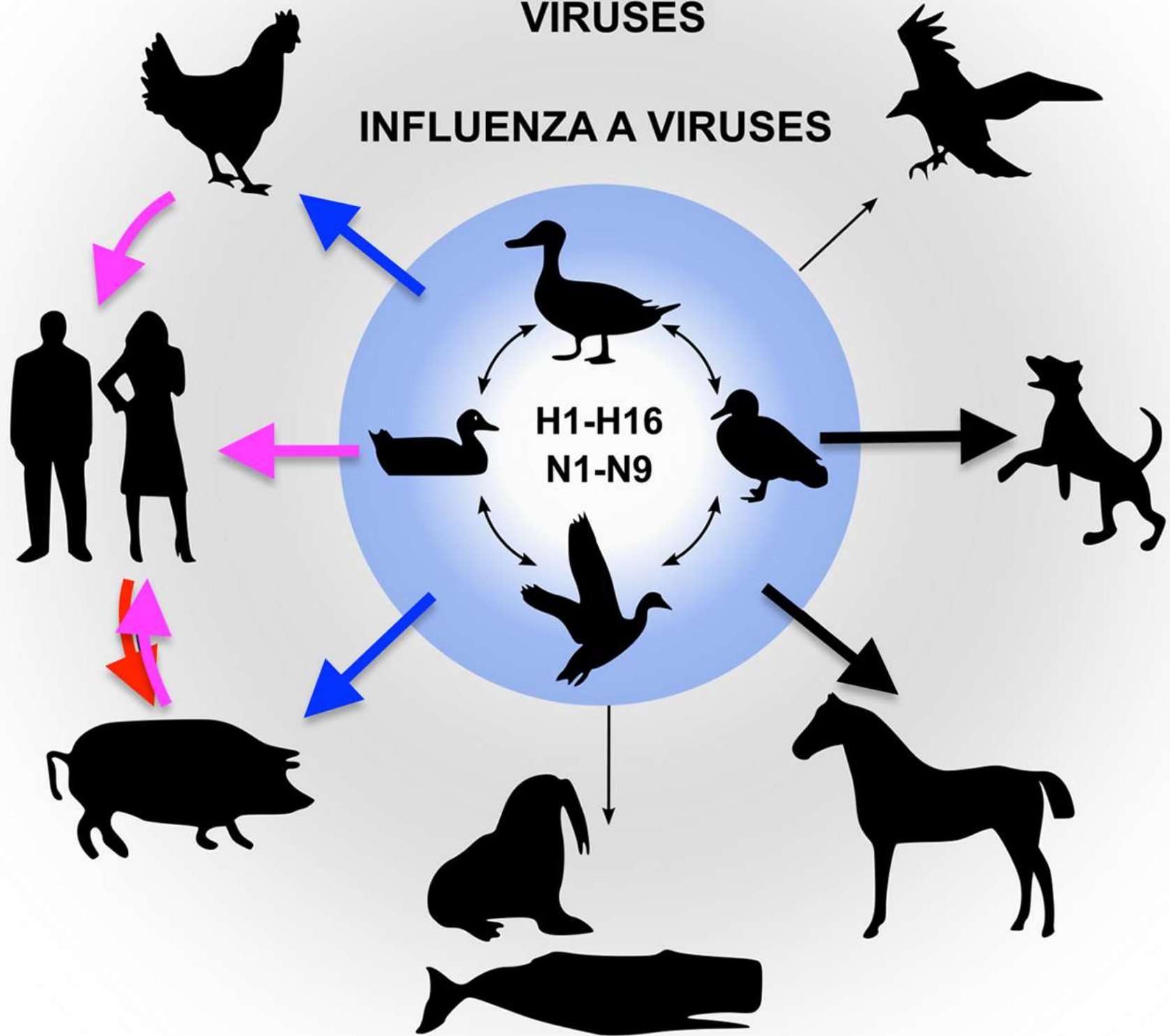






# VIRUSES

## INFLUENZA A VIRUSES



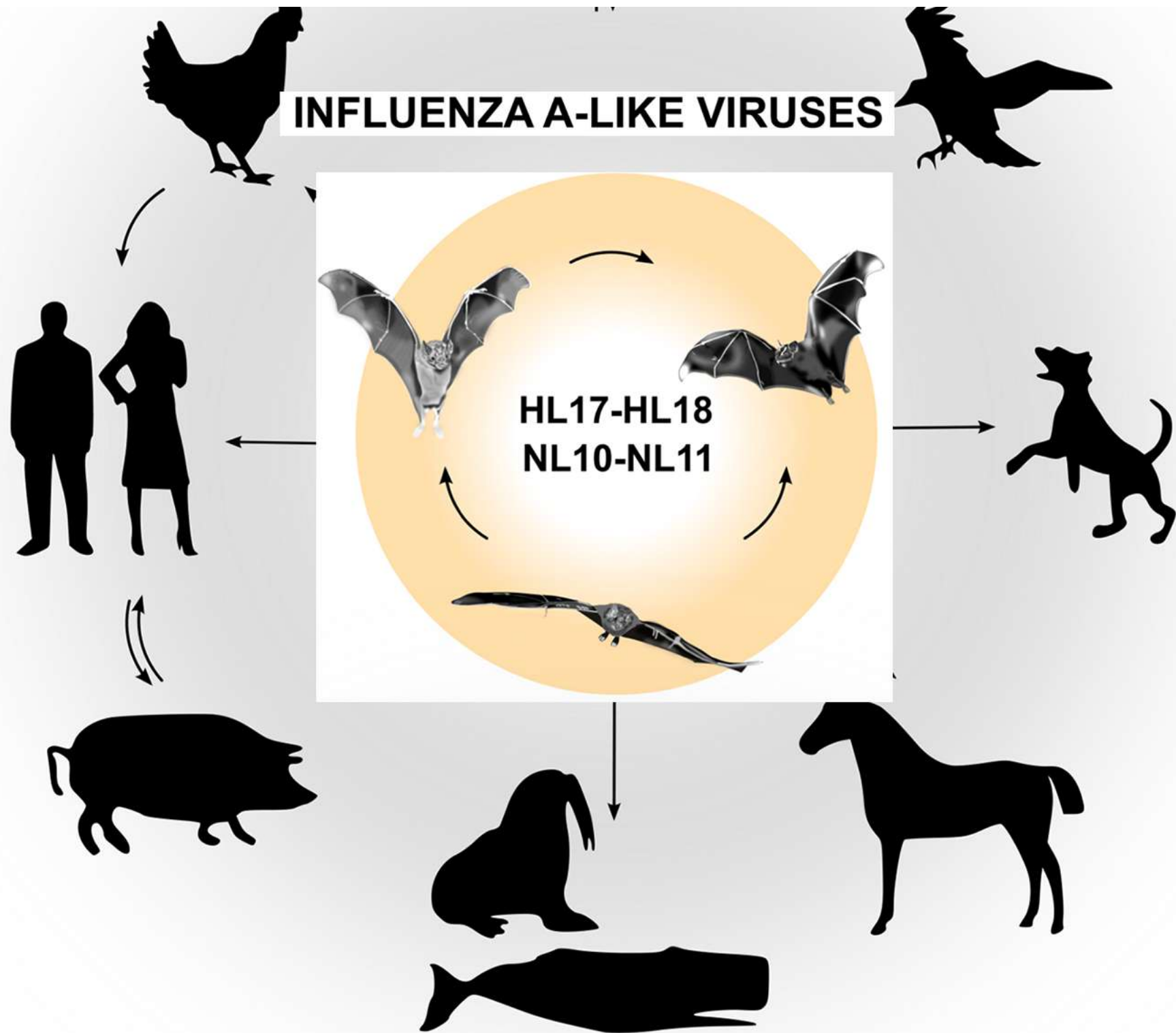








# INFLUENZA A-LIKE VIRUSES







# Les recombinauts du coronavirus

**1** Une personne est infectée au même moment par deux virus différents.

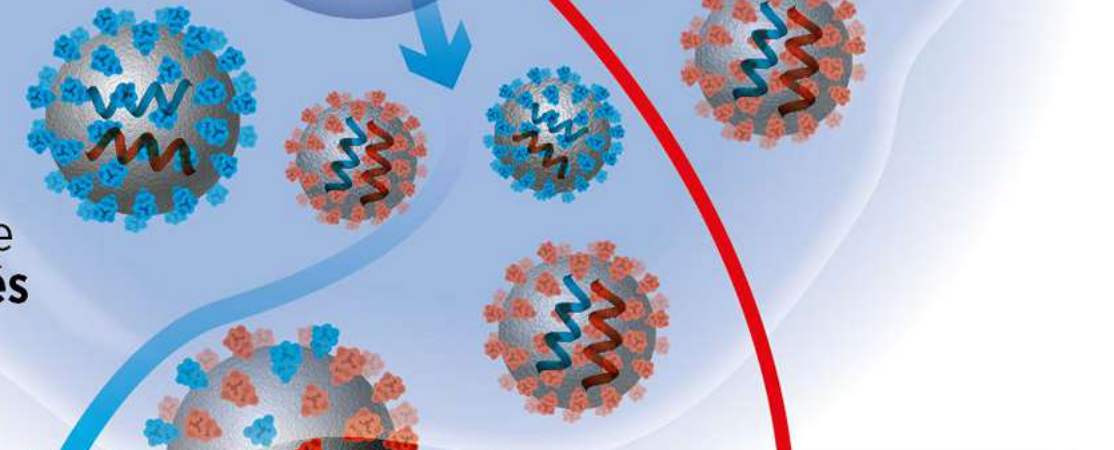
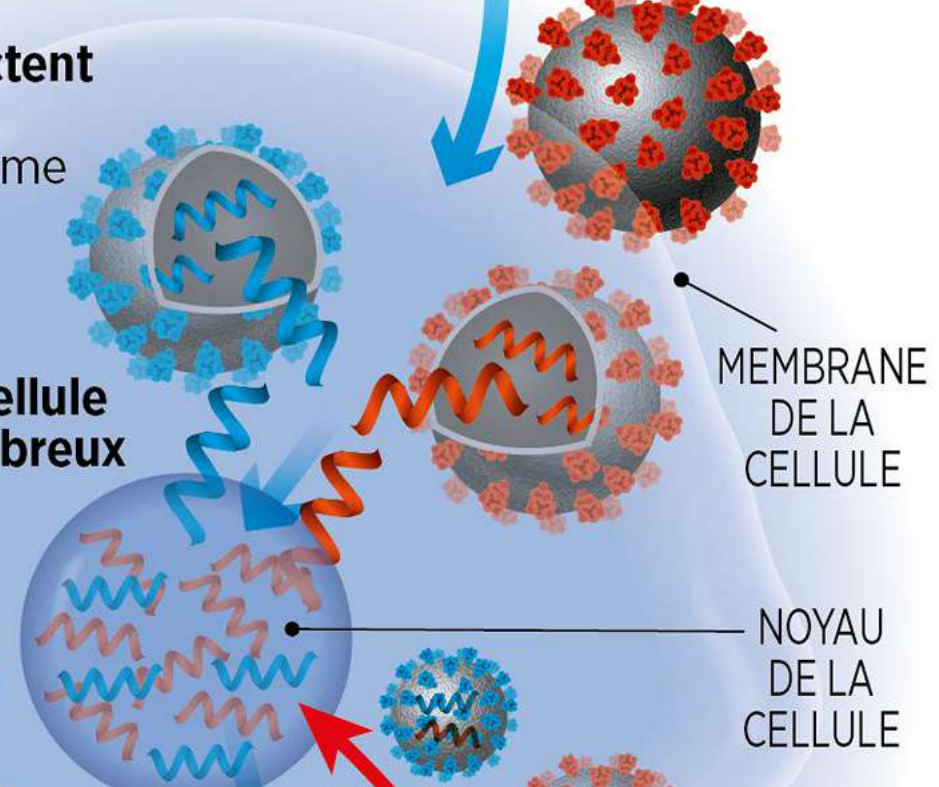
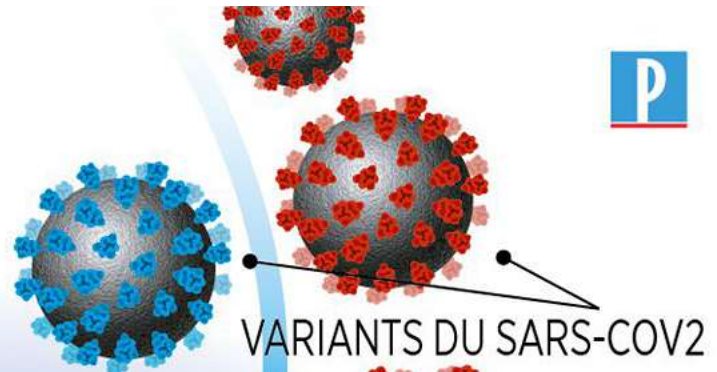
**2** Ces deux virus infectent une même cellule et libèrent leur génome (ARN).

CELLULE

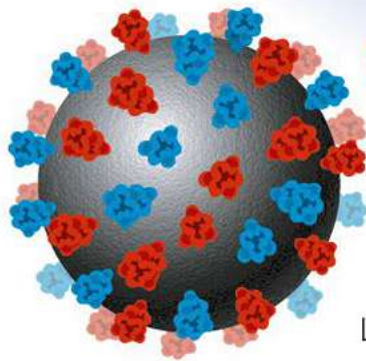
**3** Le noyau de la cellule réplique de nombreux ARN viraux.

**4** L'ARN se recrée une enveloppe virale dans la cellule infectée.

**5** Le nouveau virus présente des propriétés de ses deux « parents ».



**Durant la réplication, les génomes des deux virus se rencontrent et mutent pour former une nouvelle combinaison.**





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**Citation:** Shang J, Zheng Y, Yang Y, Liu C, Geng Q, Luo C, et al. (2018) Cryo-EM structure of infectious bronchitis coronavirus spike protein reveals structural and functional evolution of coronavirus spike proteins. *PLoS Pathog* 14(4): e1007009. <https://doi.org/10.1371/journal.ppat.1007009>

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**1** Department of Veterinary and Biomedical Sciences, College of Veterinary Medicine, University of Minnesota, Saint Paul, MN, United States of America, **2** Department of Diagnostic and Biological Sciences, School of Dentistry, University of Minnesota, Minneapolis, MN, United States of America, **3** Characterization Facility, College of Science and Engineering, University of Minnesota, Minneapolis, MN, United States of America

As cell-invading molecular machinery, coronavirus spike proteins pose an evolutionary conundrum due to their high divergence. In this study, we determined the cryo-EM structure of avian infectious bronchitis coronavirus (IBV) spike protein from the  $\gamma$ -genus. The trimeric IBV spike ectodomain contains three receptor-binding S1 heads and a trimeric membrane-fusion S2 stalk. While IBV S2 is structurally similar to those from the other genera, IBV S1 possesses structural features that are unique to different other genera, thereby bridging these diverse spikes into an evolutionary spectrum. Specifically, among different genera, the two domains of S1, the N-terminal domain (S1-NTD) and C-terminal domain (S1-CTD), diverge from simpler tertiary structures and quaternary packing to more complex ones, leading to different functions of the spikes in receptor usage and membrane fusion. Based on the above structural and functional comparisons, we propose that the evolutionary spectrum of coronavirus spikes follows the order of  $\alpha$ -,  $\delta$ -,  $\gamma$ -, and  $\beta$ -genus. This study has provided insight into the evolutionary relationships among coronavirus spikes and deepened our understanding of their structural and functional diversity.

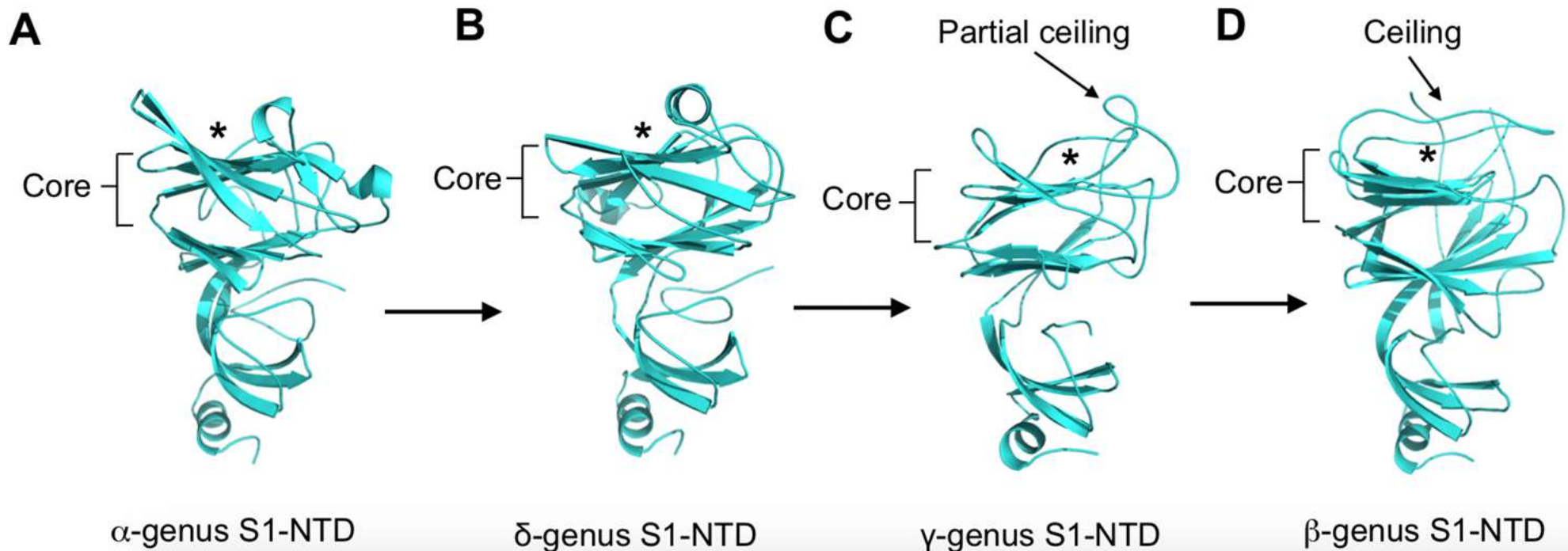


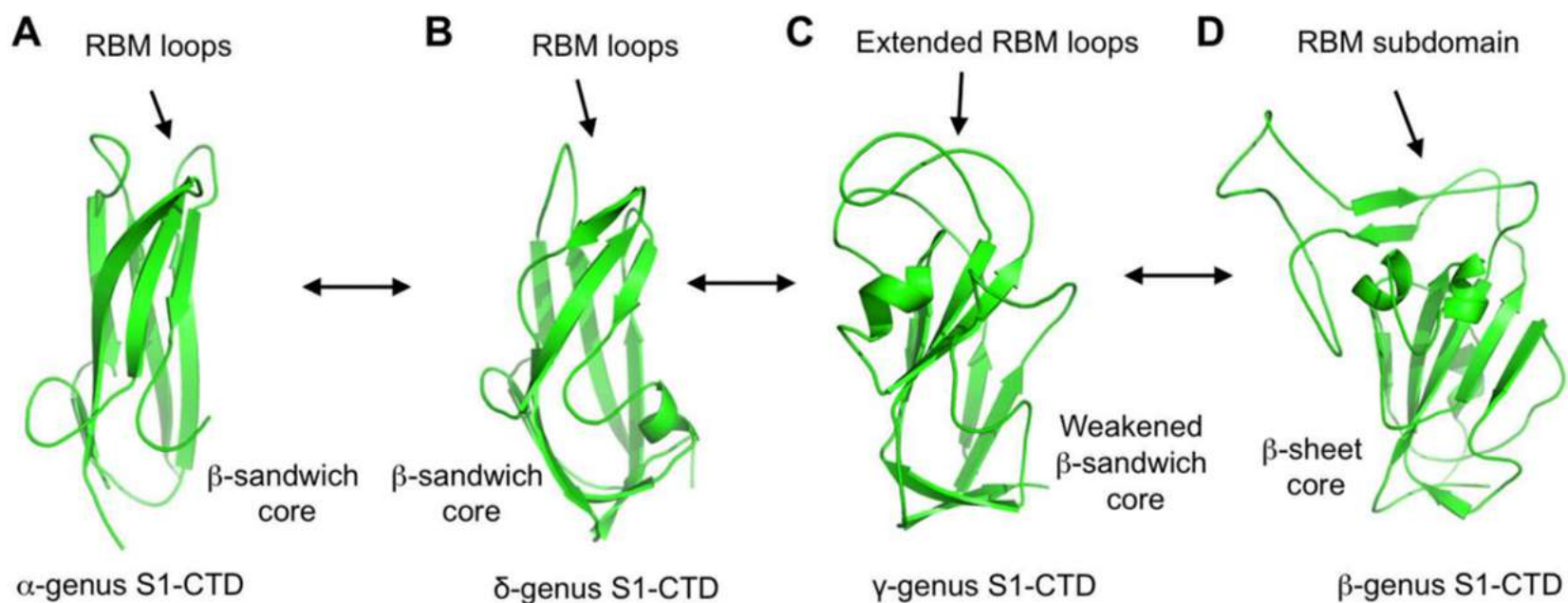
# Cryo-EM structure of infectious bronchitis coronavirus spike protein reveals structural and functional evolution of coronavirus spike proteins

Fig 3

Structural comparisons of S1-NTDs from four coronavirus genera.

(A) Structure of S1-NTD from  $\alpha$ -genus human coronavirus NL63 (PDB ID: 5SZS). Although each subunit of NL63 S1 contains two copies of S1-NTDs (i.e., S1-NTD1 and S1-NTD2), S1-NTD2 was used in structural comparisons with the S1-NTDs from the other genera because it occupies the same location as the S1-NTDs from the other genera in quaternary structures of the spikes (see Fig 5A). (B) Structure of S1-NTD from  $\delta$ -genus porcine delta coronavirus (PdCoV) (PDB ID: 6B7N). (C) Structure of S1-NTD from  $\gamma$ -genus IBV. (D) Structure of S1-NTD from  $\beta$ -genus SARS coronavirus (PDB ID: 5X58). \* indicates sugar-binding site or putative sugar-binding site in sugar-binding S1-NTDs from each genus. Core structure, partial ceiling, and extensive ceiling are labeled. Arrows from panels (A) to (D) indicate evolutionary direction. (E) Quantitative structural comparisons among S1-NTDs from different genera using software Dali [58]. Both Z-score and r.m.s.d. were calculated for each pair of the proteins. PDB IDs for NL63, PdCoV and SARS S1-NTDs are the same as in panels (A)-(D). PDB IDs for mouse hepatitis coronavirus (MHV) and MERS coronavirus are 3JCL and 5X5F, respectively. CEACAM1b (PDB ID: 5VST), whose  $\beta$ -sandwich fold is topologically different from that of coronavirus S1-NTDs [59], was used as a negative control. N.D.: no detectable structural similarity.





**E**

Z score/ rmsd (Å)	α-genus		β-genus		γ-genus	δ-genus	(-) control
	NL63	MHV	SARS	MERS	IBV	PdCoV	CEACAM1b
NL63		N.D.	N.D.	2.1/ 4.3	3.4/ 3.8	10.1/ 2.2	N.D.
MHV	N.D.		13.8/ 3.5	19.1/ 3.1	5.3/ 4.0	N.D.	N.D.
SARS	N.D.	13.8/ 3.5		14.2/ 3.2	4.7/ 3.7	2.9/ 4.4	N.D.
MERS	2.1/ 4.3	19.1/ 3.1	14.2/ 3.2		6.1/ 3.7	3.6/ 3.8	N.D.
IBV	3.4/ 3.8	5.3/ 4.0	4.7/ 3.7	6.1/ 3.7		7.1/ 3.5	N.D.
PdCoV	10.1/ 2.2	N.D.	2.9/ 4.4	3.6/ 3.8	7.1/ 3.5		N.D.
CEACAM1b	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	

**Fig 4. Structural comparisons of S1-CTDs from four coronavirus genera.** (A)-(D): Structures of S1-CTDs from different genera. Core structures and RBMs are labeled. (E) Quantitative structural comparisons among S1-CTDs from different genera. The PDB IDs are the same as those in Fig 3. Left right arrows from panels (A) to (D) indicate that evolution could go either way.





**L'Europe s'engage à interdire  
l'élevage en cage des animaux  
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