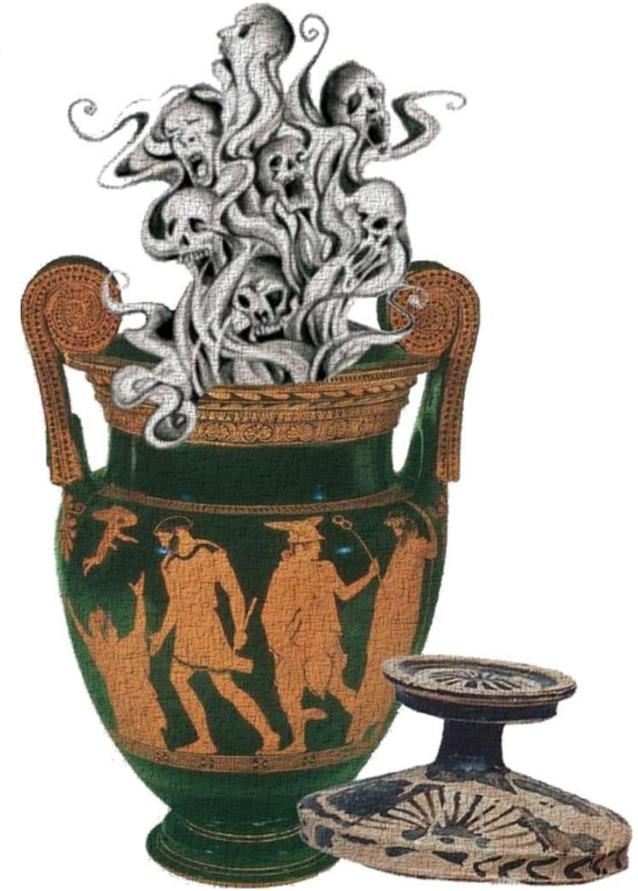




H.Fuseli, l'incubo, 1782

The origin of COVID: Did people or nature open Pandora's box? From the origin to variants



The origin of the pandemic remains uncertain: what scientific data is available, which gives clues to what happened ???
it is possible to evaluate the complex question of guilt if someone or something is to blame?

Let's talk about Spillover, let's talk about Laboratory

Massimo Ciccozzi. Full prof of epidemiology
Univ of biomedical campus

two main theories about its origin.

One is that it jumped naturally from wildlife to people.

The other is that the virus was studied in a laboratory, from which it escaped, due to human error

It is very important to know this for prevention if we want to avoid a second event of this type.

After the pandemic first broke out in December 2019, Chinese authorities reported that many cases had occurred in the wet market a place selling wild animals for meat - in Wuhan.



Fabrizio Gatti
L'infinito errore

La storia segreta
di una pandemia
che si doveva evitare

La nave di Iseo

Oceani

Natural o created in laboratory:
the mystery of the origin

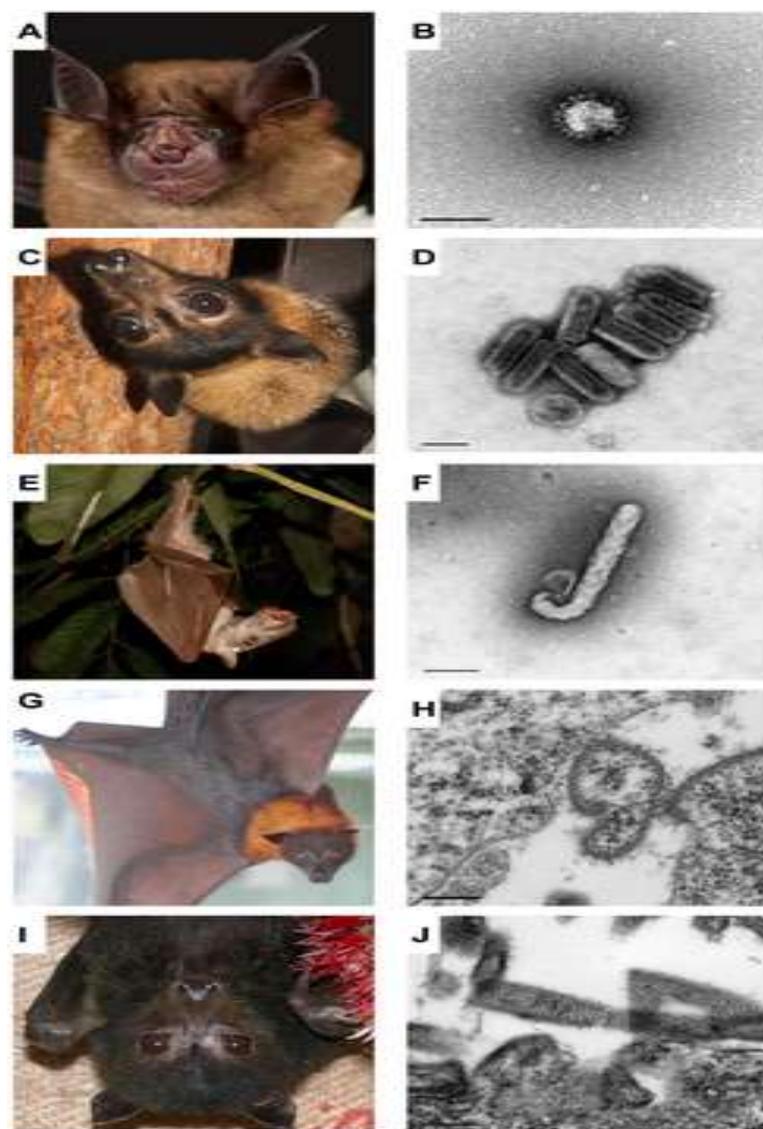




Coronaviruses have been detected in various members of the animal kingdom and, in multiple instances, they have been protagonists of zoonosis, sometimes resulting in very insidious human pathogens.

Bats and rodents are the most common gene source of most Alpha- and Beta- coronavirus, while birds are mostly of Gamma- and Delta-coronavirus. The majority of the currently known human coronaviruses and a large number of SARS-related coronaviruses have emerged from bats .

Indeed, bats are ideal “virus spreaders”, given their longevity, sociability, and ability to fly. **In this host, coronaviruses are well adapted, non-pathogenic but with a great genetic diversity and potential to infect humans.**



Bats are diverse, as are the viruses that infect them.

The Chinese horseshoe bat (A; *Rhinolophus sinicus*) is one of many *Rhinolophus* sp. that are a natural host of SARS-like coronaviruses (B; scale bar 100 nm).

The spectacled flying fox (C; *Pteropus conspicillatus*) along with other *Pteropus* sp. are reservoirs for the Australian Bat lyssavirus (D; scale bar 100 nm).

A number of African fruit bats including *Hypsignathus monstrosus* (E) have been found to host Ebola virus (F; Ebola Reston, scale bar 200 nm).

The Malayan flying fox (G; *Pteropus vampyrus*) is the natural host of Nipah virus (H; scale bar 200 nm).

All four pteropid Australian bat species including *Pteropus alecto* (I) have been found to carry Hendra virus (J; scale bar 200 nm).

The 2019-new coronavirus epidemic: Evidence for virus evolution

Domenico Benvenuto¹ | Marta Giovanetti² | Alessandra Ciccozzi¹ | Silvia Spoto³ | Silvia Angeletti⁴ | Massimo Ciccozzi²

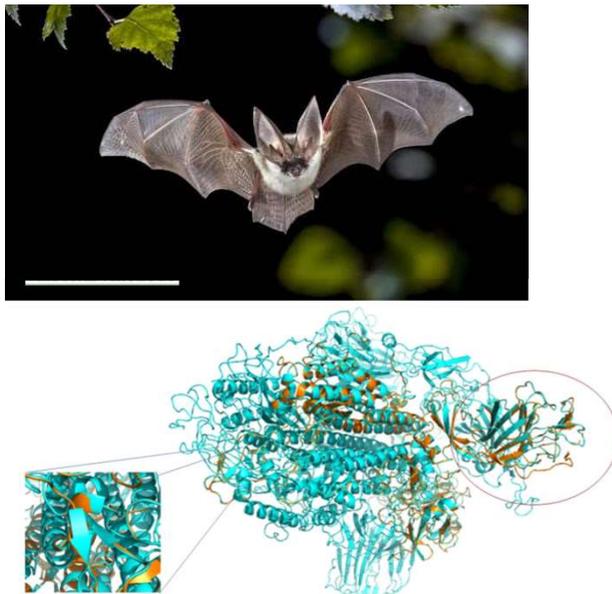
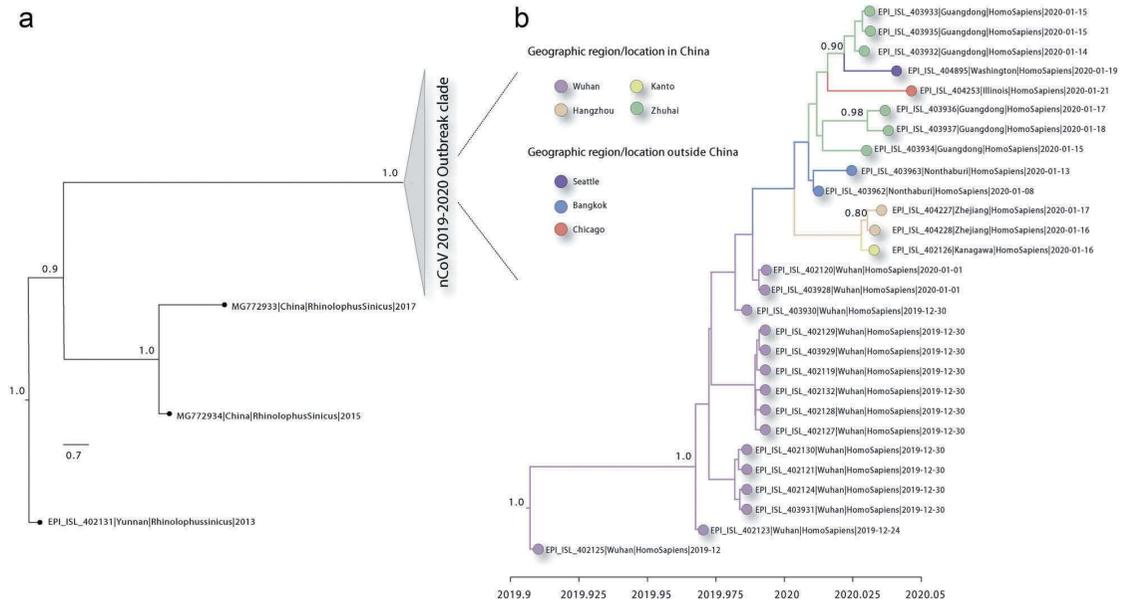


FIGURE 3 Cartoon model of the structural superposition between the homology model of the 2019-nCoV in blue and the spike glycoprotein of SARS coronavirus (PDB code 6acc.1) in orange. The red circle highlights the presence of a variable region on the 2019-nCoV at the beginning of the protein, whereas the blue square highlights the presence of two beta-sheets on the 2019-nCoV (401:KYR and 440:LND) that are not present on the SARS-CoV structure; SARS, severe acute respiratory syndrome

The global spread of 2019-nCoV: a molecular evolutionary analysis

Domenico Benvenuto¹*, Marta Giovanetti²*, Marco Salemi^{3,4}, Mattia Prospero^{3,4}, Cecilia De Flora³, Luiz Carlos Junior Alcantara⁵, Silvia Angeletti⁶* and Massimo Ciccozzi²*

¹Unit of Medical Statistics and Molecular Epidemiology, University Campus Bio-Medico of Rome, Italy; ²Laboratório de Flavivírus, Instituto Oswaldo Cruz, Fundação Oswaldo Cruz, Rio de Janeiro, Brazil; ³Department of Epidemiology, University of Florida, Gainesville, FL, USA; ⁴Emerging Pathogens Institute, University of Florida, Gainesville, FL, USA; ⁵Unit of Clinical Laboratory Science, University Campus Bio-Medico of Rome, Italy





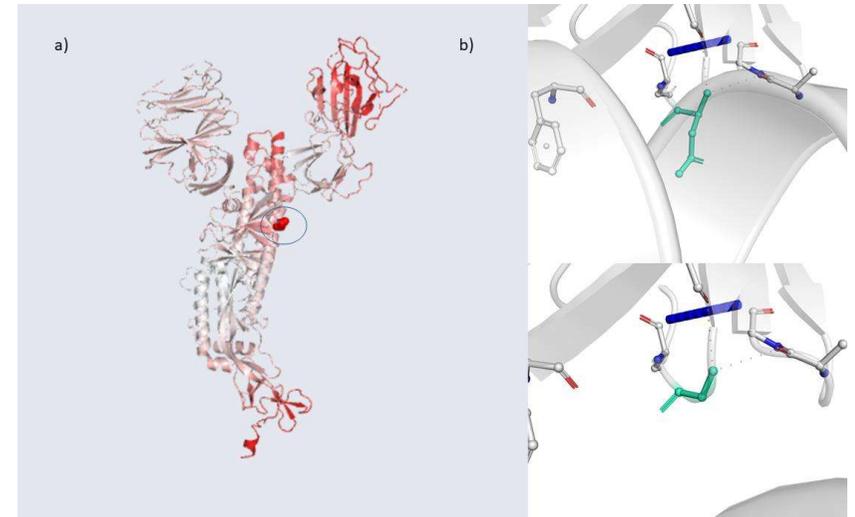
J Med Virol. 2020 Jun 3 : 10.1002/jmv.26104.
doi: [10.1002/jmv.26104](https://doi.org/10.1002/jmv.26104) [Epub ahead of print]
PMCID: PMC7300971
PMID: [32492183](https://pubmed.ncbi.nlm.nih.gov/32492183/)

Evidence for mutations in SARS-CoV-2 Italian isolates potentially affecting virus transmission

[Domenico Benvenuto](#),¹ [Ayse Banu Demir](#),² [Marta Giovanetti](#),³ [Martina Bianchi](#),⁴ [Silvia Angeletti](#),⁵ [Stefano Pascarella](#),⁴ [Roberto Cauda](#),⁶
⁷ [Massimo Ciccozzi](#),¹ and [Antonio Cassone](#)⁸

dg614

Figure 1, a) A model of spike glycoprotein monomer displaying the amino acids coloured according to the vibrational entropy change upon mutation, Red regions are those gaining in flexibility, The amino acidic mutation is blue circled; b) the top image shows the molecular interaction between the side chain of the wild-type amino acid and the side chains of the surrounding amino acid; the bottom image shows the molecular interaction between the side chain of the mutated amino acid and the side chains of the surrounding amino acid



A doubt of multiple introduction of SARS-CoV-2 in Italy: A preliminary overview

Marta Giovanetti^{1,2} | Silvia Angeletti³  | Domenico Benvenuto⁴  |
Massimo Ciccozzi⁴ 

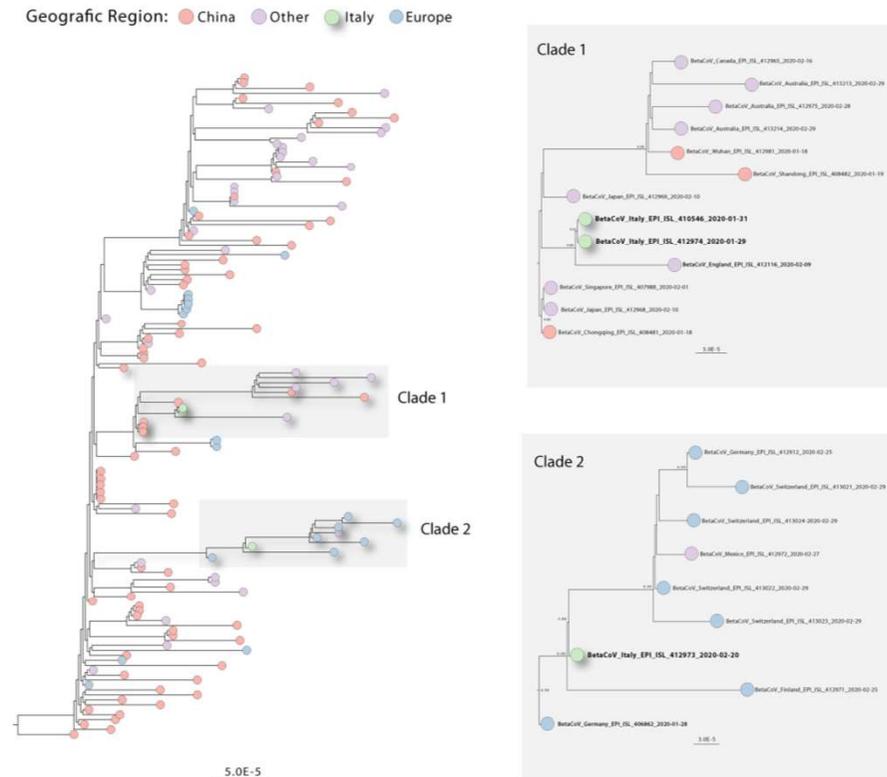


FIGURE 1 Maximum likelihood phylogeny, estimated from complete and near-complete coronavirus (CoV) genomes using genome data available in GISAID. Colors represent different locations (panels A and B represent expansions of the clades containing the Italian CoV isolates [green]) in clade 1 closely related with sequence from England, in clade B closely related with sequences from Germany

COVID-2019: The role of the nsp2 and nsp3 in its pathogenesis

Silvia Angeletti¹ | Domenico Benvenuto² | Martina Bianchi³ | Marta Giovanetti⁴ | Stefano Pascarella³ | Massimo Ciccozzi²

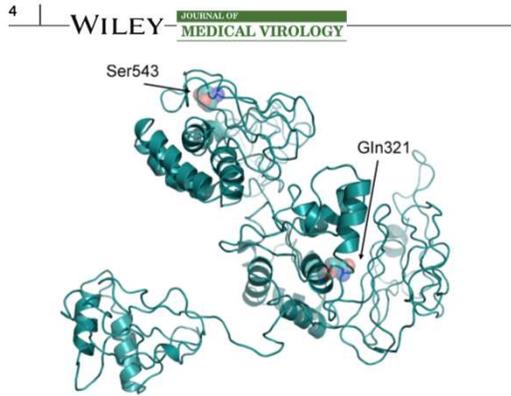


FIGURE 2 I-Tasser model of the COVID-2019 nsp2. Residues under positive selective pressure with a $P < .05$ are shown as sticks and transparent spheres and are marked by the corresponding labels. COVID-2019, novel Coronavirus; nsp2, non structural protein-2

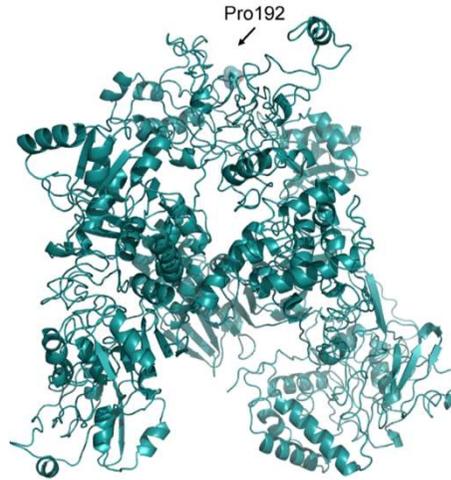


FIGURE 3 I-Tasser model of the COVID-2019 nsp3. The residue under positive selective pressure with a $P < .05$ is shown as sticks and transparent spheres and is marked by the corresponding label. COVID-2019, novel Coronavirus; nsp, non structural protein

Emerging SARS-CoV-2 mutation hot spots include a novel RNA-dependent-RNA polymerase variant

Maria Pachetti^{1,2*}, Bruna Marini^{1,4}, Francesca Benedetti⁵, Fabiola Giudici³, Elisabetta Mauro⁴, Paola Storici¹, Claudio Masciovecchio¹, Silvia Angeletti⁶, Massimo Ciccozzi⁶, Robert C. Gallo^{7,8}, Davide Zella^{5,9*} and Rudy Ippodirino^{4*}

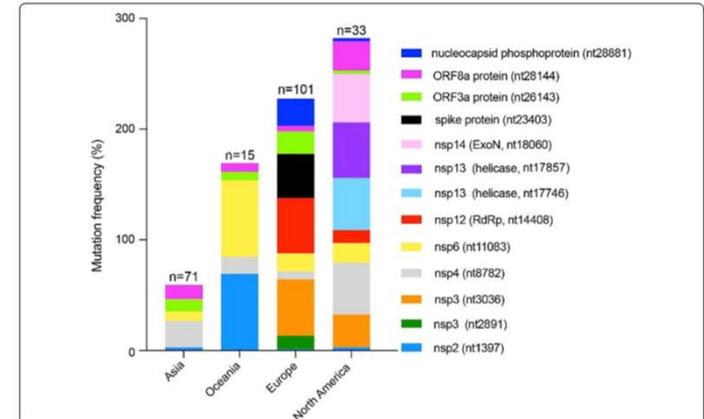
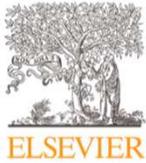


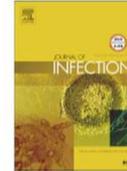
Fig. 1 SARS-CoV-2 mutation frequency in different geographic areas. Eight novel recurrent hotspots mutations (namely 1397, 2891, 14408, 17746, 17857, 18060, 23403 and 28881) and 5 hotspots already reported in literature (namely 3036, 8782, 11083, 28144 and 26143) were subdivided into 4 geographic areas: Asia (n=71), Oceania (n=15), Europe (n=101), North America (n=33). The mutation frequency was estimated for each of them, by normalizing the number of genomes carrying a given mutation in a geographic area, by the overall number of retrieved genomes per geographic area; the graph shows the cumulative mutation frequency of all given mutations present in each geographic area. Mutation locations in viral genes are reported in the legend as well as the proteins (i.e. non-structural protein, nsp) presenting these mutations. The figure shows that genomes from European and North American patients present an increase in mutation frequency compared to Asia. It is also possible to observe that European and North American show a differential pattern of mutations: mutation 14408 (red), 23403 (black), 28881 (electric blue) and 26143 (light green) are present mostly in Europe, whereas 18060 (pink), 17857 (purple) and 17746 (light blue) are present mostly in North America



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Evolutionary analysis of SARS-CoV-2: how mutation of Non-Structural Protein 6 (NSP6) could affect viral autophagy



Domenico Benvenuto^{a,*}, Silvia Angeletti^b, Marta Giovanetti^c, Martina Bianchi^d, Stefano Pascarella^d, Roberto Cauda^{e,f}, Massimo Ciccozzi^a, Antonio Cassone^g

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^c Laboratório de Flavivirus, Instituto Oswaldo Cruz, Fundação Oswaldo Cruz, Av. Brasil, 4365 - Manguinhos, Rio de Janeiro - RJ 21040-900, Brasil

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^f Department of Healthcare Surveillance and Bioethics, Catholic University of Sacred Heart, Largo Francesco Vito, 1, Rome 00168, Italy

^g Center of genomics, genetics and biology, University of Siena, Petriccio e Belriguardo, 35, Siena 53100, Italy

Thus, its role would be to limit autophagosome expansion, directly or indirectly by starvation or chemical inhibition

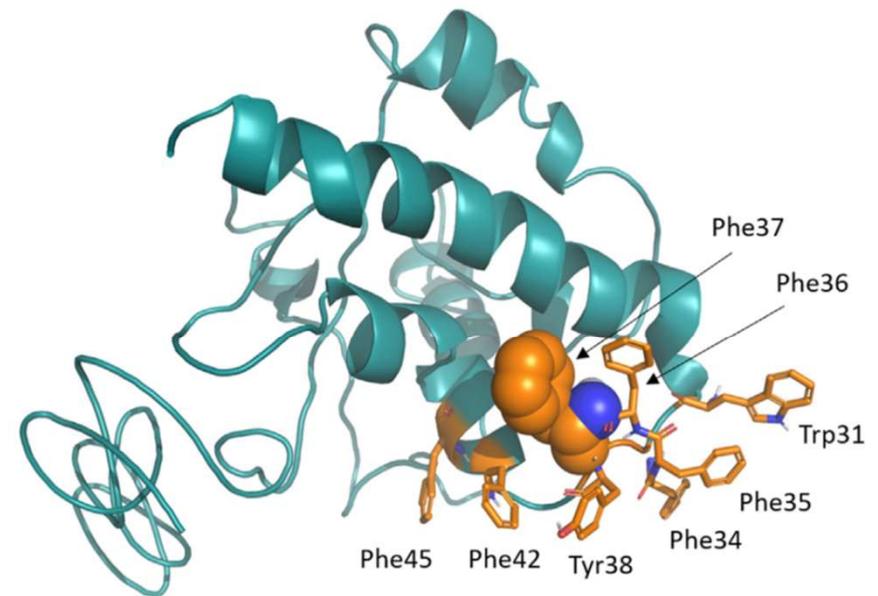


Fig. 1. I-TASSER model of NSP6. Residue under positive selective pressure with a $p < 0.05$ is shown as a sphere. Residues found in the structure proximity are shown in sticks. All residues are marked by the corresponding labels.

The origin of the Omicron variant have been a subject of controversy

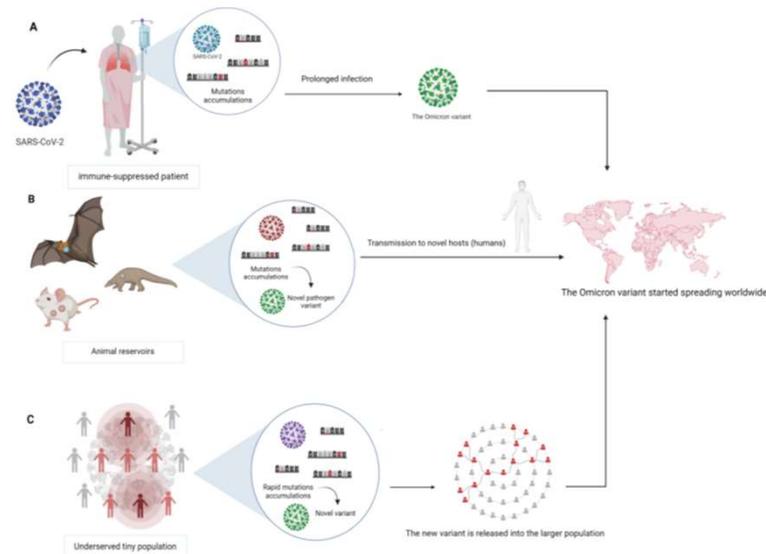
The three most likely scenarios for how the Omicron variant appeared.

A: Omicron could have developed and significantly mutated in an immune-suppressed patient with prolonged viral infections.

The idea was

B: The Omicron variant might have evolved in non-human reservoirs, such as animal source, and as of late spread from it to humans.

C: The virus might began circulating and changing in underserved places with a tiny population, where it had the opportunity to mutate rapidly, it could then have been released into the larger population, where it could have spread to different groups.



Early in the [COVID-19 pandemic](#), Andersen and other scientists were consulted by the [NIH](#) and [NIAID](#) about the possibility of a [lab leak](#).

Andersen, in an email to [Anthony Fauci](#) in January 2020, told Fauci, the government's top infectious disease expert, that **some features of the virus made him wonder whether it had been engineered**, and noted that he and his colleagues were planning to investigate further by analyzing the virus's genome.

Andersen and his colleagues initially suspected that **the virus could have escaped from a laboratory in Wuhan**, China, **after additional analyses and an accumulation of this scientific evidence**, Andersen and his co-authors concluded that the **hypothesis was unfounded**.

In a **2022** paper, Andersen concluded that animals sold in a market in [Wuhan](#), China, were most likely to be the source of the virus



correspondence Nature Medicine

The proximal origin of SARS-CoV-2 To the Editor —

Since the first reports of novel pneumonia (COVID-19) in Wuhan, Hubei province, China^{1,2}, there has been considerable discussion on the origin of the causative virus, SARS-CoV-2 (also referred to as HCoV-19)⁴. Infections with SARS-CoV-2 are now widespread, and as of 11 March 2020, 121,564 cases have been confirmed in more than 110 countries, with 4,373 deaths. SARS-CoV-2 is the seventh coronavirus known to infect humans; SARS-CoV, MERSCoV and SARS-CoV-2 can cause severe disease, whereas HKU1, NL63, OC43 and 229E are associated with mild symptoms⁶. Here we review what can be deduced about the origin of SARS-CoV-2 from comparative analysis of genomic data. We offer a perspective on the notable features of the SARS-CoV-2 genome and discuss scenarios by which they could have arisen. **Our analyses clearly show that SARS-CoV-2 is not a laboratory construct or a purposefully manipulated virus.**



CO DI BIDEN
TIARE FAUCI
Come il potere a Trump, Joe Biden
rappresentanti e sottofunzionari
di da "procedimenti giudiziari"
mente motivati"

Kristian G. Andersen^{1,2} ✉, Andrew Rambaut³, W. Ian Lipkin Edward C. Holmes^{4 5} and Robert F. Garry¹Department of Immunology and Microbiology,

A recent article published in Nature Medicine explains, thanks to a careful comparative analysis of the available genomic data, that SARS-CoV-2 cannot be a laboratory construct or a specially modified virus, and shows the probable scenarios from which it evolved.

The study of the structural and biochemical characteristics of the genome of this virus seems to suggest that: (i) it is optimized to bind to the human ACE2 receptor; and (ii) its surface protein “spike” has a functional polybasic cutting site (furin) at the junction between the two S1 – S2 subunits.

The receptor-binding domain (RBD) of the spike protein is the most variable part of the coronavirus genome. Six amino acids of RBD have been shown to be critical for binding to the ACE2 receptor and in determining the host spectrum of SARS-CoV-like viruses. Five of these residues appear to be different between SARS-CoV and SARS-CoV-2.

SARS-CoV-2 appears to have an RBD that binds with high affinity to ACE2 from humans, ferrets, cats, and other species with high receptor homology, even though computational analyses suggest a non-ideal spike-ACE2 interaction in the presence of an RBD other than that of SARS-CoV.

The high affinity binding of the SARS-CoV-2 spike protein to the human ACE2 receptor therefore appears to be the result of natural selection of the virus on a human or human-like receptor which has generated an alternative high affinity binding solution. This is clear evidence that SARS-CoV-2 is not the product of intentional manipulation.

The second feature of SARS-CoV-2 is a polybasic cleavage site (RRAR) at the junction of S1 and S2, which allows for effective cleavage by furin and other proteases and has a role in determining viral infectivity and the host spectrum of the virus.

Furthermore, a main proline is inserted into this site, the sequence is then PRRA; it is hypothesized that this insertion leads to the addition of O-glycans flanking the cleavage site.

The function of O-glycans is unclear, but they could create a “mucin-like domain” that protects key epitopes or residues on the spike protein.

In fact, numerous viruses use mucin-like domains as glycan shields involved in immune evasion.

In light of their analysis, the Authors consider it unlikely that SARS-CoV-2 emerged from the laboratory manipulation of a SARS-CoV-like coronavirus.

In fact, genomic data irrefutably show that the virus does not derive from any previously used viral structure.

The researchers propose, instead, two possible scenarios that can explain the origin of SARS-CoV-2: (i) natural selection in an animal host prior to zoonotic transfer; and (ii) natural selection in humans following zoonotic transfer.

<https://www.nature.com/articles/s41591-020-0820-9>

Fonte: Andersen, K.G., Rambaut, A., Lipkin, W.I. et al. The proximal origin of SARS-CoV-2. Nature Medicine, 2020.

Enhancing viruses to better study them is a scientifically accepted practice,

several clues and gaps leave open the hypothesis of origin in the laboratory.

the origin of the SARS-CoV-2 virus, with two predominant hypotheses: the natural one and that of escape from the laboratory. The latter hypothesis points to Gain-of-Function (GoF), or gain-of-function, studies involving genetic manipulation of organisms, often viruses. **These studies are aimed at evaluating the effects of increased pathogenicity, transmissibility, ability to evade the immune system or changes in host range or tropism.**

The controversial role of GoF studies

Proponents of GoF studies point to its potential to understand how a virus gains the ability to overcome the species barrier or become more lethal. These studies could allow scientists to anticipate future pandemic threats and develop vaccines and therapies.

For example, GoF studies of influenza viruses have allowed the identification of key mutations associated with increased transmissibility in mammals, providing valuable information for the development of proactive vaccines. Likewise, they could contribute to the development of targeted therapeutic countermeasures, anticipating the mechanisms by which a pathogen could evolve resistance to existing drugs.

ethical and biosafety concerns regarding the risk of accidentally or intentionally releasing enhanced pathogens, with potentially catastrophic consequences for global public health.

The debate over GoF studies has intensified in recent years as a result of the COVID-19 pandemic and discussions about the possible origins of the SARS-CoV-2 virus.

The concerns relate in particular to risks related to potential accidents due to human error or unforeseen events, which cannot be completely excluded,

The origin of the COVID-19 pandemic: a debate that is still open

GoF studies are closely linked to the debate over the origin of the COVID-19 pandemic. On March 17, 2020, the journal Nature Medicine published the article “The proximal origin of SARS-CoV-2”.

The authors, evaluating the genome sequence of SARS-CoV-2, concluded that the hypothesis of laboratory origin (that is, that the virus had escaped from the laboratory or that it had been manipulated intentionally) was not a plausible scenario, leaning towards a natural origin.

This paper played an influential, indeed, central role in communicating the narrative that science had established that SARS-CoV-2 had infected people through natural spillover. Since its publication, it has been consulted online more than five million times and widely cited in the media.

In 2022, a bipartisan Commission on the COVID-19 pandemic was established, tasked with examining whether government officials had unfairly and perhaps biasedly tipped the scales toward natural origin theory and whether scientific integrity had been ignored in favor of political expediency, perhaps to hide or belittle the government's relationship with the Wuhan Institute of Virology or to avoid blaming China for any complicity in triggering the pandemic.

On February 6, 2023, Biosafety, a non-governmental organization, launched a petition asking the journal Nature to withdraw the article “The proximal origin of SARS-CoV-2”, which was deemed a product of fraud and scientific misconduct. Another petition followed to prohibit GoF research on potential pandemic pathogens, reduce the number of high-level biocontainment laboratories, and strengthen biosecurity and biohazard management for pathogen research, in order to prevent future pandemics.

The clues supporting laboratory origin

The laboratory escape theory is now supported by solid evidence. Most recent documents obtained from US. Right to Know demonstrate that American scientists planned to work with the Wuhan Institute of Virology to design novel coronaviruses with the characteristics of SARS-CoV-2 the year before the outbreak of the pandemic.

This was under the DEFUSE grant proposal for conducting engineering experiments on coronaviruses, inter alia led by Peter Daszak, president of EcoHealth Alliance

– an international non-profit dedicated to the One Health approach to preventing pandemics– but also a lead member of the mission team that WHO sent to China to investigate the origin of SARS-CoV-2.

The laboratory origin hypothesis, at first thought to be heresy and largely ostracized, is currently recognized as a credible hypothesis.

In summary, as Richard Ebright, a US molecular biologist, documents in his oral testimony filed during the US Senate hearing, **multiple lines of evidence provide the evidence to support his assessment of the laboratory origin of SARS-CoV-2. In particular, studies on the insertion of a furin cleavage site into the human Spike protein and the fact that SARS-CoV-2 is the only one among the more than 800 SARS viruses known to possess such a site.** Mathematically, this, in itself, implies that the probability of finding a natural SARS virus with a furin cleavage site is less than 1 in 800. This constitutes an extremely strong case – a “smoking gun” – in favour of laboratory origin.

In November 2021, WHO established the SAGO (Scientific Advisory Group for the Origins of Novel Pathogens), a group of 27 international scientists with the objectives of investigating the origins of SARS-CoV-2 and other emerging pathogens, defining a framework for such investigations, and evaluating available scientific evidence. SAGO released its first findings and recommendations on June 9, 2022. In a more recent report, dated June 27, 2025 and titled “Independent assessment of the origins of SARS-CoV-2”, previous assessments are examined based on scientific publications, intelligence reports, presentations and expert discussions.

four main hypotheses examined are:

Natural zoonotic origin: The virus passed from animals to humans, either directly or via an intermediate host.

Laboratory accident: The virus has infected humans following exposure during field research or due to a violation of biosafety procedures.

Introduction via cold chain: The virus arrived in animal markets via frozen products and infected humans.

Deliberate laboratory manipulation: The virus was created in the laboratory and subsequently leaked

Excluding hypothesis 3, which is not supported by solid evidence, the report focuses on the others. Regarding hypothesis 2 of a laboratory accident, SAGO complains about the cooperation of the Chinese Government which did not provide WHO or SAGO with information crucial for a full assessment.

In conclusion, although zoonotic origin is the hypothesis most accredited by current scientific data, the precise origins of SARS-CoV-2 remain undetermined pending further scientific information and data.

Significant data gaps remain that prevent SAGO from establishing with certainty how SARS-CoV-2 initially entered the human population.

Cite as: S. Lytras *et al.*, *Science* 10.1126/science.abh0117 (2021).

The animal origin of SARS-CoV-2

Spyros Lytras¹, Wei Xia², Joseph Hughes¹, Xiaowei Jiang³, David L. Robertson¹

¹Medical Research Council–University of Glasgow Centre for Virus Research, Glasgow, UK. ²National School of Agricultural Institution and Development, South China Agricultural University, Guangzhou, China. ³Department of Biological Sciences, Xi'an Jiaotong–Liverpool University, Suzhou, China.
 Email: xiaowei.jiang@xjtlu.edu.cn; david.l.robertson@glasgow.ac.uk

Trading of animals susceptible to bat coronaviruses is the likely cause of the COVID-19 pandemic

Sarbecoviruses closely related to SARS-CoV-2

Coronaviruses that are evolutionarily closest to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have been sampled in China, Cambodia, Japan, and Thailand (5). The phylogenetic tree, inferred from a genomic region minimized for recombination (5), shows sarbecoviruses closely related to SARS-CoV-2. Host species for each virus, horseshoe bat (*Rhinolophus*), human (*Homo sapiens*), and pangolin (*Manis javanica*) and the year of sample collection are shown in the key. Longquan140 is inferred from another genomic region (5) (dashed line). See supplementary table S1 for more details.



The unresolved question on Covid-19 virus origin: the three cards game?

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²Department of Veterinary Medicine, University of Sassari, 07100 Sassari, Italy

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⁷Flavivirus Laboratory, Oswaldo Cruz Institute, Oswaldo Cruz Foundation, Rio de Janeiro 21040-360, Brazil

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⁹Department of Medicine Medical Statistics and Epidemiology Unit, Campus Bio-Medico University, Rome, Italy

The ongoing discussion about the real origin of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) feeds acrimonious debates. Where did SARS-CoV-2 come from? Was SARS-CoV-2 transmitted in the wild from an animal to a person before exploding in Wuhan or was it an engineered virus that escaped from research or a laboratory in Wuhan? Right now, we still don't know enough whether SARS-CoV-2 is human-made or not, and lab-leak theories remain essentially speculative. Many recent studies have pointed out several plausible scenarios. Anyhow, currently, even if suspicions by some about the possibility of lab-leak hypothesis still remain, the consensus view is that the pandemic probably started from a natural source and, to determine the real origin of the SARS-CoV-2 virus, further research is needed.

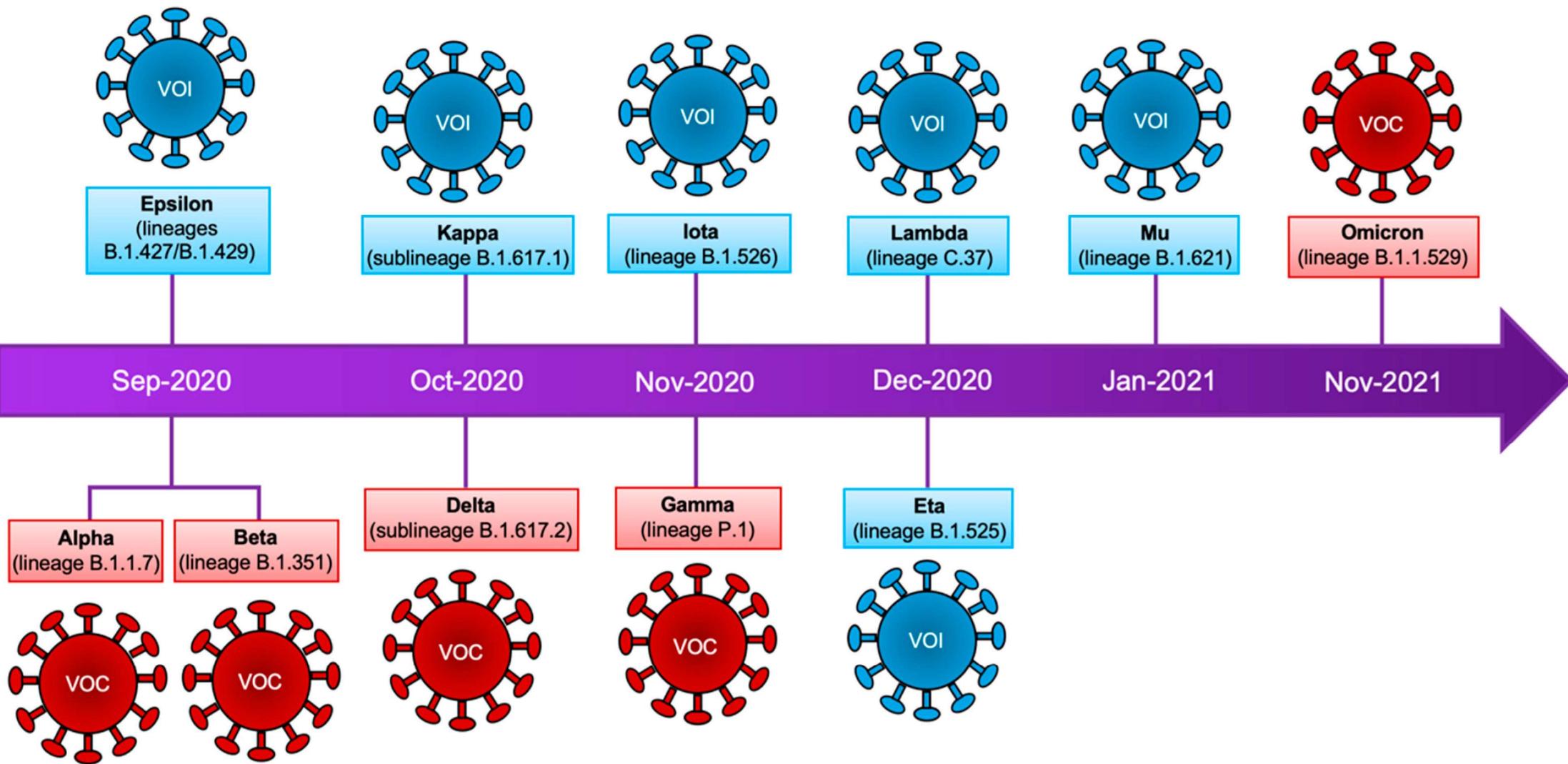
Many questions are open and need an answer..

VARIANTS ??????

THERAPY. ??

VACCINE. ?

ORIGIN. ???



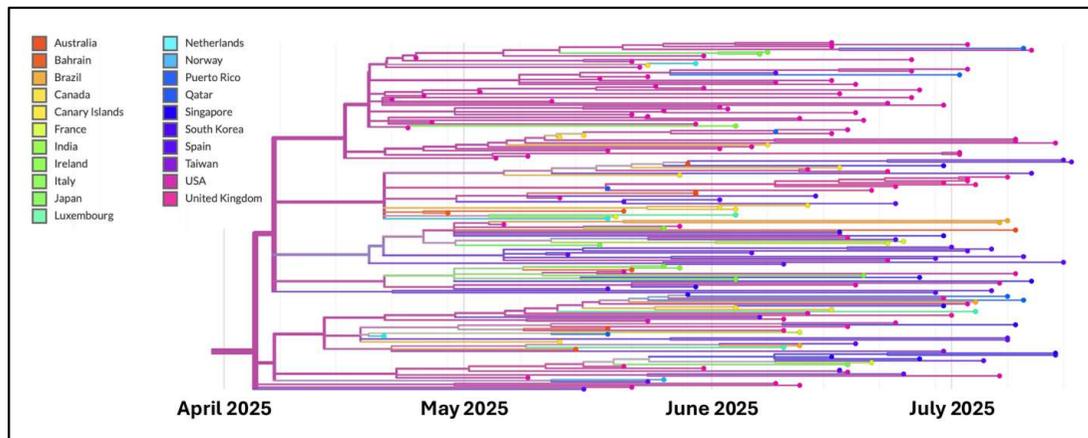
SARS-CoV-2 XFG: A Genomic Insight into the new recombinant

Authors: Francesco Brandal^{1,*}, Massimo Ciccozzi¹, Fabio Scarpa²

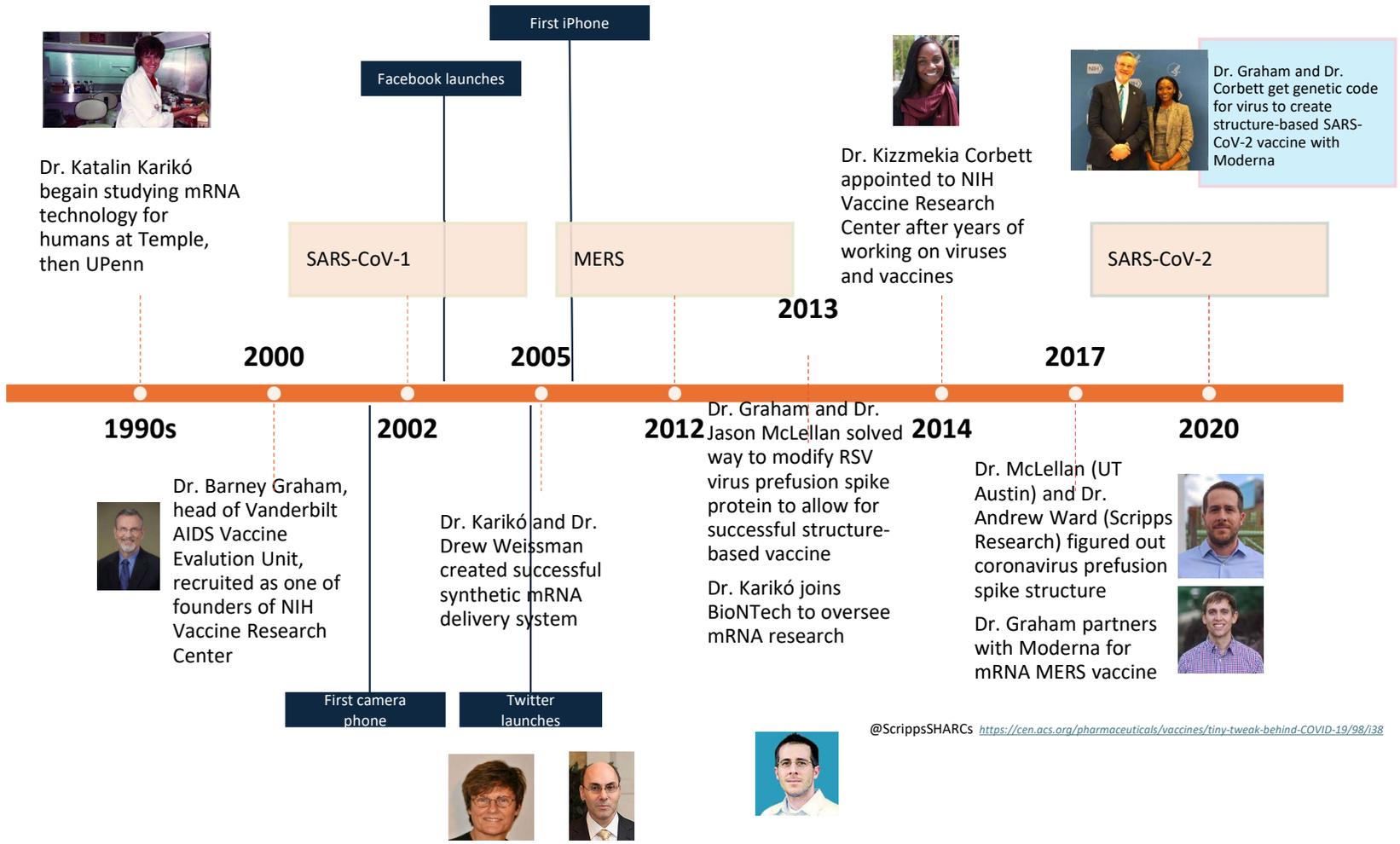
Affiliation

1. Unit of Medical Statistics and Molecular Epidemiology, University Campus Bio-Medico of Rome, Rome, Italy.
2. Department of Biomedical Sciences, University of Sassari, Sassari, Italy

The latest SARS-CoV-2 recombinant is represented by the one labeled XFG (nicknamed *Stratus*), which has arisen through recombination between LF.7 and LP.8.1.2 [5]. SARS-CoV-2 XFG was detected for the first time on 27 January 2025 [6] and in 25 June 2025 it has been designed as VuM (Variant under Monitoring) [5]. Compared to the currently dominant SARS-CoV-2 variant NB.1.8.1, the XFG variant carries several additional mutations in the Spike protein, including: S31P, K182R, R190S, R346T, K444R, V445R, T478K, N487D, and T572I. In contrast, when compared to the JN.1 variant, XFG displays the following Spike mutations: T22N, S31P, K182R, R190S, R346T, K444R, V445R, F456L, N487D, Q493E, and T572I



Timeline of mRNA technology and key figures in vaccine development



@ScrippsSHARCs <https://cen.acs.org/pharmaceuticals/vaccines/tiny-tweak-behind-COVID-19/98/i38>

“Health is not everything, but without health everything is nothing”.

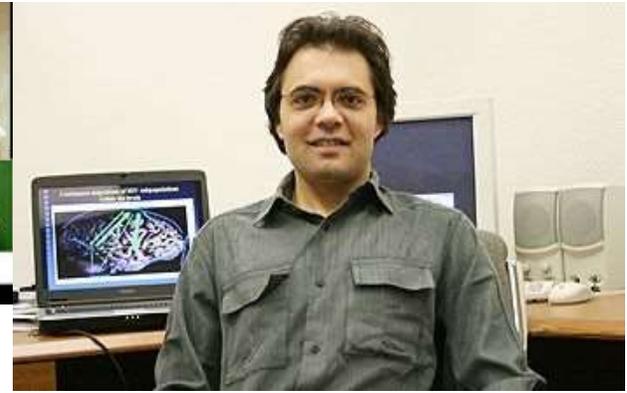
massimo

UP TO NOW



Completely conscious Ignorance is the prelude to any real progress in science

James Clerk Maxwell



147 papers
on Covid-19

