

Evolución UniAndes 2020-10

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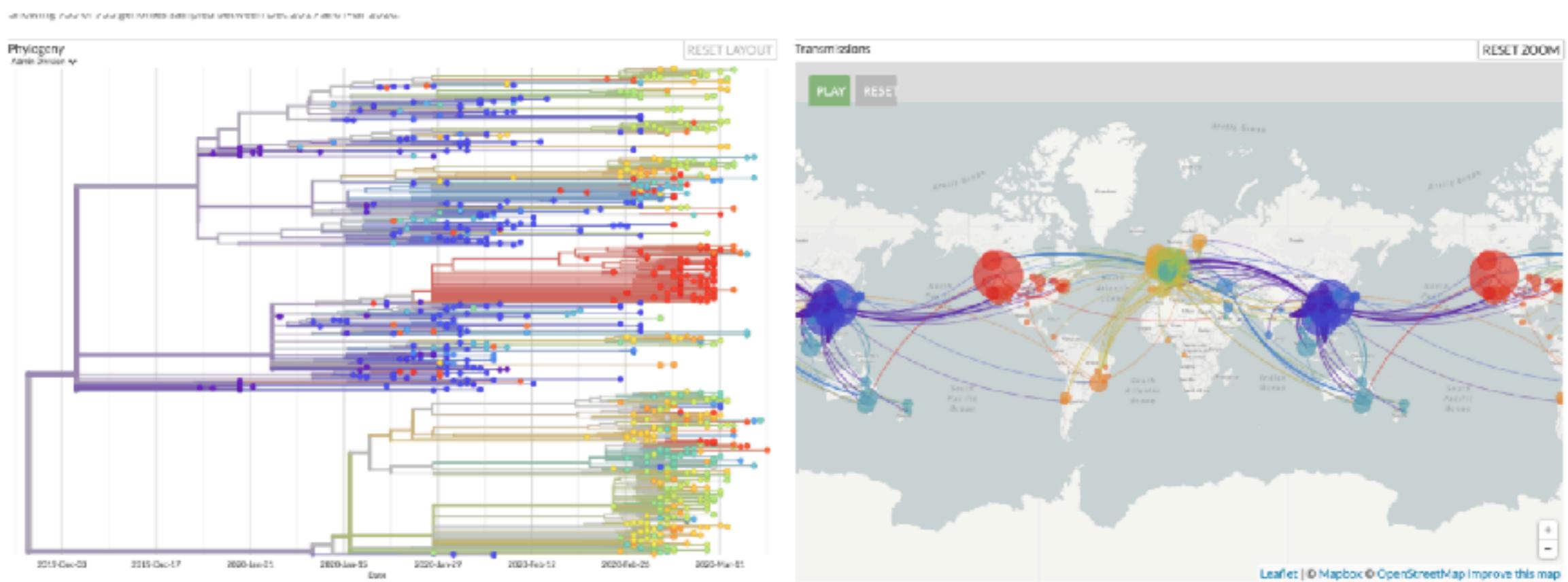
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Materiales para curso virtual de Evolución, Universidad de los Andes

Horario: martes, miércoles y jueves, 9:30-10:20 am

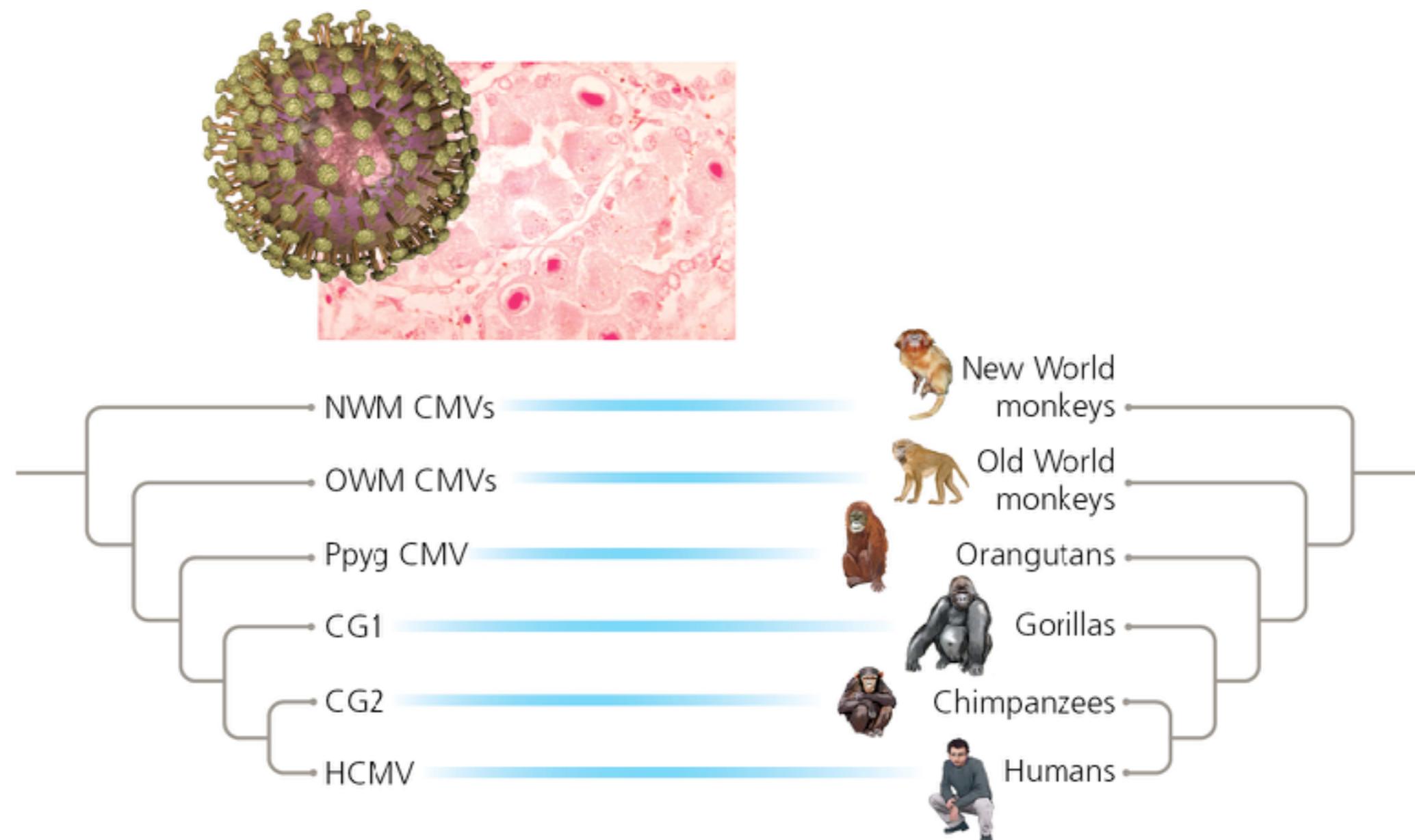
Aula Virtual en SicuaPlus

La Teoría Evolutiva Aplicada a Comprender una Pandemia: el caso de COVID-19



Distribución geográfica y filogenia basada en genomas del corononavirus SARS-CoV-2, causante de la pandemia de COVID-19. Imagen tomada el 23 de marzo de 2020 del proyecto [Nexstrain](#).

filogenias de virus y hospederos sugieren codiversificación (e.g. herpes)



NWM = New World monkeys

OWM = Old World monkeys

CG = chimpanzee and gorilla isolates

Zimmer & Emlen
macmillan learning

BUSH IS DETAILING ALL CONTRIBUTIONS ON INTERNET SITE

RECORD \$49 MILLION SUM

Critics Call It Public Relations Gesture — Rivals Are Not Likely to Follow Suit

By FRANK BRUNI

WASHINGTON, Sept. 9 — Gov. George W. Bush of Texas, the frontrunner for the Republican Presidential nomination, began posting on his Internet site today a detailed list of the size and source of every contribution to his campaign, a gesture of voluntary disclosure that campaign finance experts said was unprecedented in Presidential politics.

While the Federal Election Commission requires all candidates to provide such information shortly after each quarter — and the Government posts the data on the Internet — Mr. Bush said his campaign would update the details of its fund-raising daily. A brief time lag will occur, however, between the receipt of a donation and its appearance on the site, the Bush campaign said.

The information Mr. Bush's aides posted today covered contributions through Aug. 26, and although it did not provide a total, aides said the figure was slightly more than \$49 million, a record sum.

Mr. Bush said the daily Internet postings underscored his support for more openness in the campaign finance system.

"Americans will be able to look for themselves to find out who is helping to fund my campaign," Mr. Bush said today in a statement.

But several advocates of changes in campaign finance laws said the Bush campaign was also making a savvy public relations move amid clear public concern about the influence of money in politics.

These advocates said Mr. Bush was trying to counter criticism that his campaign was a financial steamroller, driven by the wealthiest Americans.

"He realizes that there's negative reaction to how much money he's raised and he's trying to take the



Andrea Mohin/The New York Times

Looking Good

Third graders Dariel Lanfranco, center, and Maximo Rodriguez were among the many students who opted to wear their new uniforms to class at Public School 7 in Cypress Hills, Brooklyn. Their school is new, but other schools in New York City reopened yesterday to overcrowding. Page B1.

PRESIDENT UNVEILS GUN BUYBACK PLAN

\$15 Million to Be Spent in Bid to Combat U.S. Violence

By ERIC SCHMITT

WASHINGTON, Sept. 9 — Stepping up the Federal Government's fight against gun violence, President Clinton today announced a \$15 million program to buy back firearms from private owners.

Under the first Federal effort of its kind, the Department of Housing and Urban Development would provide grants of up to \$500,000 to police departments around the country to buy back and destroy as many as 300,000 weapons, mainly in and around public housing projects. The housing department is suggesting that the authorities offer \$50 a gun, either in cash, food, gift certificates, toys or tickets to sporting events.

Continued on Page A22

Spraying Expands in New York Encephalitis Fight

By JODI WILGOOREN

New York City vastly expanded its week-old air raid on mosquitoes yesterday with pesticide spraying scheduled over the next three days for all of Brooklyn and Queens and much of the Bronx, as the number of suspected cases of the potentially deadly St. Louis encephalitis grew to 60, including, for the first time, 3 residents of Manhattan.

Mayor Rudolph W. Giuliani said that ground spraying of malathion, which kills mosquitoes, was planned for most of Manhattan in the coming days, and that the aerial assault would continue until the first frost, probably at least a month away, over the rest of the city, including Staten Island.

Air spraying is impractical in Manhattan because of the density of the buildings and the evening crowds outdoors, officials said.

"I ask you not to create any undue or unnecessary alarm or panic," Mr.

To Cover Entire City — Number of Suspected Cases Grows to 60

Giuliani said at a news conference yesterday morning at City Hall. "There's no point in not spraying, because there's no harm in spraying. So even if we're overdoing it, there's no risk to anyone in overdoing it."

"The more dead mosquitoes," he added, "the better."

Roger S. Nasci, a research entomologist at the Centers for Disease Control and Prevention, said the current outbreak — with 9 confirmed cases, 3 of them fatal — ranks among the smaller incidences of St. Louis encephalitis, which infected about 2,200 people throughout the Mississippi Valley in 1975. But he said that this epidemic presented a special challenge.

"Given that this is New York City, you have a very large population at risk," said Mr. Nasci, who is assisting local officials in fighting the disease. "That's the reason for this very aggressive aerial spray approach to control the mosquitoes."

City officials said they would not name the victims, to protect their privacy.

Spraying will take place only when mosquitoes tend to be active, from about 5 to 11 P.M. and from about 4 to 8 A.M., city officials said. Last night, the spraying began in parts of Queens and the Bronx but was aborted because of heavy rain.

This morning, three helicopters were scheduled to spray malathion throughout much of Brooklyn, from Prospect Park on south to the water; in the Bronx, between the Bronx River Parkway and the Hutchinson River Parkway; and in Queens, from Bay Terrace south to Hollis Hill and east to the Nassau County line. To-

Continued on Page B6

Late Edition

New York: Today, rain, heavy at times, high 79. Tonight, clear, low 63. Tomorrow, ample sun, high 79. Yesterday, high 82, low 71. Weather map and details appear on page D1.

PRESIDENT ASSERTS JAKARTA MUST ACT OR ADMIT TROOPS

TIMOR CHAOS CONTINUES

A U.S. Role in Peacekeeping Is Undefined and Clinton Does Not Threaten to End Aid

By PHILIP SHENON

WASHINGTON, Sept. 9 — President Clinton demanded today that Indonesia permit an international peacekeeping force to try to restore order in East Timor if the Indonesian military is unable to end "this madness" — the wave of violence that has taken hundreds of lives across the tiny province since it voted for independence last week.

"If Indonesia does not end the violence, it must invite, it must invite the international community to assist in restoring security," the President said at the White House as he prepared to leave for the Asia-Pacific economic summit meeting in New Zealand.

But the President did not threaten an immediate cutoff of economic assistance to Indonesia, as some lawmakers and human rights groups had warned him. Nor did he cut commercial arms sales to Indonesia, which are expected to total about \$15 million over the next year.

Instead, he said that he had ordered the Pentagon to suspend its few formal contacts with the Indonesia military, and that he would consider economic sanctions if the killing in East Timor did not stop. "My own willingness to support future assistance will depend very strongly on the way Indonesia handles this situation," he said. (Excerpts, page A12.)

He said the United States was prepared to assist Australia in its efforts to form an international peacekeeping force for East Timor, an idea Indonesia has rejected. Mr. Clinton made clear that no decision had been made on the extent of American involvement in the mission. The Pentagon has said there are no plans to deploy American ground troops.

The Administration has faced a difficult calculation in deciding how to react to the bloodletting that began after the independence referendum in East Timor. Hundreds of





south pasadena news

Late Edition

New York: Today, partial sunshine, high 74. Tonight, patchy fog, low 67. Tomorrow, partly sunny with late showers, high 73. Yesterday, high 71, low 59. Weather map is on page D4.

Outbreak of Virus in New York Much Broader Than Suspected

Officials Fear Migrating Birds May Transmit It

By JENNIFER STEINHAUER

Health and wildlife officials said yesterday that nearly twice as many people in the New York region as had previously been recognized had been stricken by a mosquito-borne illness, and they said the virus might be present in a far larger geographic area than they originally suspected.

The new assessment came as experts confirmed that the virus was not St. Louis encephalitis, as first believed, but a strain called West Nile virus that had never before been reported in the Western Hemisphere. Officials also attributed a fourth death to the outbreak: a woman in her 60's from New Rochelle in Westchester County.

The virus is transmitted to humans from mosquitoes that have bitten infected birds. That fact has led experts to suspect that the virus may exist in many parts of the region, since scores of dead crows have been turning up from Connecticut to Suffolk County on Long Island.

"I have never seen crows dying in this kind of pattern in 30 years of studying them," said Ward Stone, the chief wildlife pathologist for the State Department of Environmental Conservation.

What is more, as birds continue their fall migration, some scientists are concerned that birds carrying the virus will take it with them as they move south.

"I think this is something we need to be very concerned about," said Dr. Duane Gubler, the director of the division of vector-borne infectious disease at the Federal Centers for Disease Control and Prevention.

There are now 37 confirmed cases of the West Nile virus in neonates in the

who died since the outbreak began in August for traces of the illness. Further, 162 cases remain under investigation in New York City.

One bird from Westport, Conn., has tested positive for the illness, as has one crow from Scarsdale, officials said. Last Friday, health officials from the Centers for Disease Control confirmed that several birds from the Bronx Zoo and its surrounding area were infected with West Nile.

The state has received 523 reports of dead birds around the region; many more are being reported among bird-watchers, conservationists and the like.

"These viruses do travel with birds, and bird migration has been in process now for a better part of the month," Dr. Gubler said. "There is a good possibility that this virus has already been taken to areas further south. We are going to rethink our whole surveillance approach."

Other Federal agencies have put states bordering the metropolitan region on alert to look for large pockets of dead birds.

Mayor Rudolph W. Giuliani said yesterday that the change in diagnosis, and the rise in numbers of the sick, would not change the city's plans to spray for mosquitoes periodically as the need arises, nor should it worry New Yorkers, because the illnesses are similar and are prevented the same way.

"I should emphasize that this is just reclassification," the Mayor said yesterday.

Symptoms of the illness, including fever and headache, are usually mild. But in rare cases, the virus can cause neurological disorders and



Photographs by Associated Press

One In, One Out

Senator John McCain of Arizona, left, formally entered the race for the Republican Presidential nomination yesterday as former Vice President Dan Quayle withdrew from it. Page A22.

Spending Bills Cannot Meet The Deadline, G.O.P. Admits

By TIM WEINER

WASHINGTON, Sept. 27 — With regretted ever since,

Tobacco Giant Gave to Backer Of Pataki Trips

MUSEUM CHAIRMAN BROACHED REMOVAL OF VIRGIN PAINTING

CONCESSION TO GIULIANI

But Talks With City Fall Apart
— Brooklyn Museum Aides
Reject Idea of a Deal

By DAVID BARSTOW
and DAVID M. HERZENHORN

Seeking to end a dispute with Mayor Rudolph W. Giuliani over a controversial art exhibition, the chairman of the Brooklyn Museum of Art discussed with city officials yesterday the possibility of removing the painting that had become the flash point of the controversy: a dung-stained portrait of the Virgin Mary.

But after city officials disclosed the discussions to reporters, museum officials responded with dismay, and the museum chairman said last night the exhibition would proceed as planned.

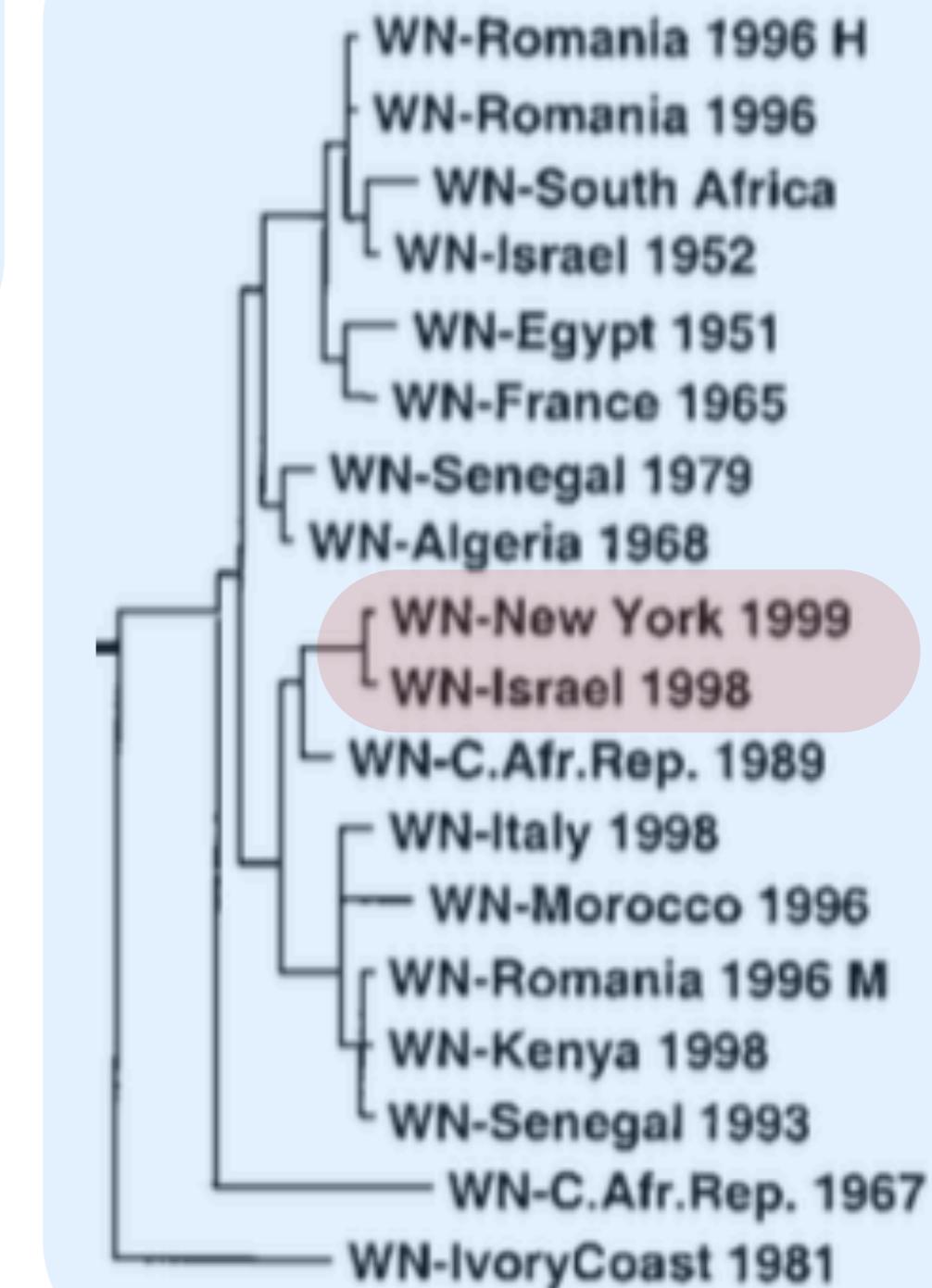
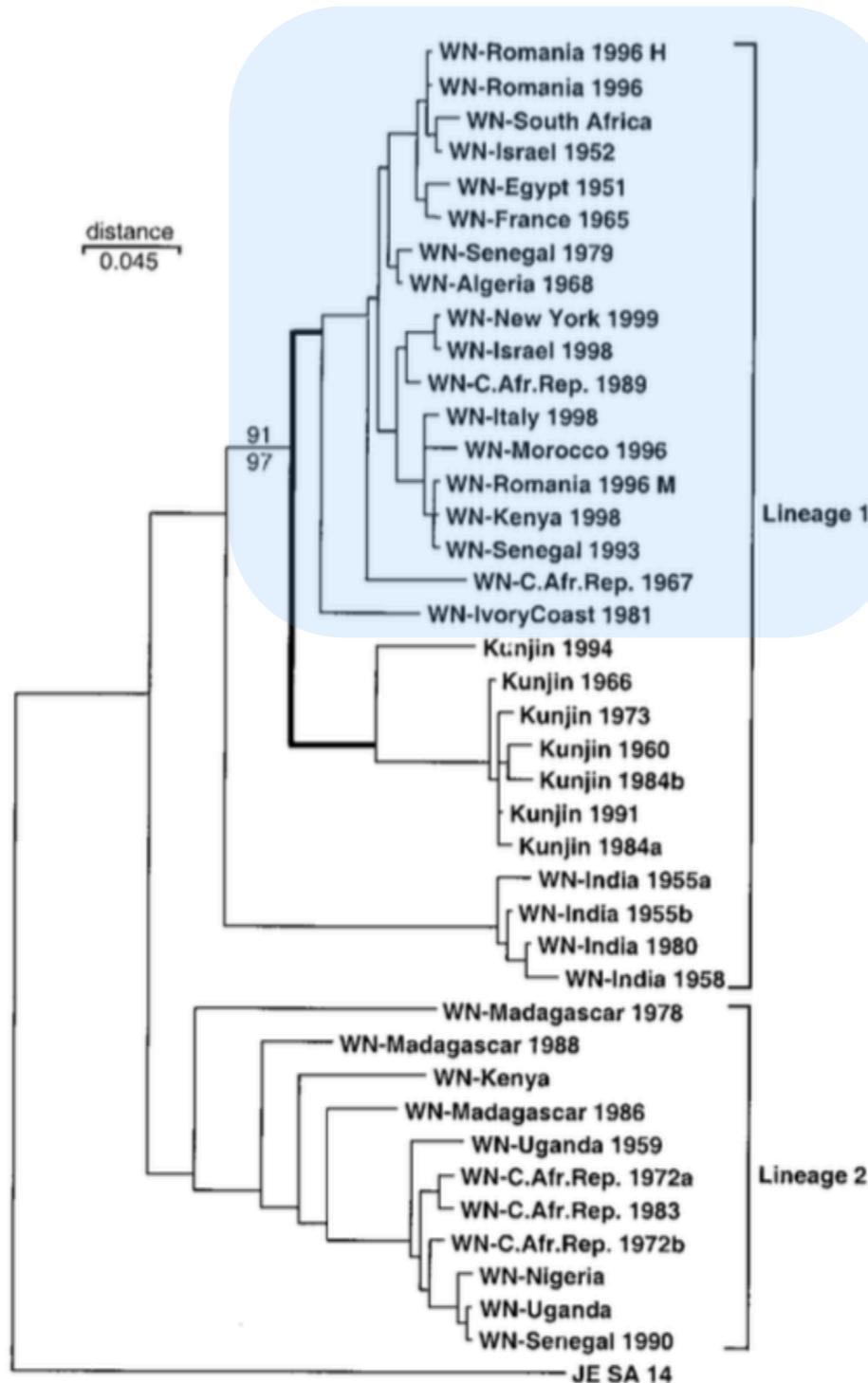
"Whatever I discussed is now off the table," Robert S. Rubin, chairman of the museum's board, said last night, clearly upset that word of his negotiations had been made public before he had discussed them with other top museum officials.

The development appeared to deepen a battle that the Mayor has embraced with gusto, and that spilled yesterday into his likely Senate campaign against Hillary Rodham Clinton, who was forced to speak out on the issue. [Page B5]

Arnold Lehman, director of the museum, said last night that he remained opposed to removing the Vir-



Fig. 2. Phylogenetic tree based on E-glycoprotein nucleic acid sequence data (255 base pairs). The tree was constructed with the program MEGA by neighbor-joining with Kimura two-parameter distance (scale bar). Bootstrap confidence level (500 replicates) and a confidence probability value based on the standard error test (22, 23) were calculated using MEGA and are included on the tree (top and bottom values, respectively), illustrating support for the division between the lineage 1 WN virus group (not including the India isolates) and the KUN virus group. The best estimated length of the segment (bold line) separating these groups, in units of expected nucleotide substitutions per site, is 0.06928 and is statistically significantly positive ($P < 0.01$) by the likelihood ratio test (fastDNAMl maximum likelihood program). An approximate 95% confidence interval for the true length of this segment is 0.03347, 0.10737. The isolate history of strains used in this tree and the alignment used for analysis are available upon request from the authors. GenBank accession numbers for the sequences included in the tree are as follows: WN-Romania 1996 H, AF130363; WN-Romania 1996, AF205879; WN-South Africa, AF205880; WN-Israel 1952, AF205881; WN-Egypt 1951, AF001568; WN-France 1965, AF001560; WN-Senegal 1979, AF001569; WN-Algeria 1968, AF001567; WN-New York 1999, AF196835; WN-Israel 1998, AF205882; WN-C.Afr.Rep. 1989, AF001558; WN-Italy 1998, AF205883; WN-Morocco 1996, AF205884; WN-Romania 1996 M, AF130362; WN-Kenya 1998, AF146082; WN-Senegal 1993, AF001570; WN-C.Afr.Rep. 1967, AF001566; WN-Ivory Coast 1981, AF001561; Kunjin 1994, AF196495; Kunjin 1966, AF196509; Kunjin 1973, AF196515; Kunjin 1960, D00246; Kunjin 1984b, AF196498; Kunjin 1991, AF196491; Kunjin 1984a, AF196519; WN-India 1955a, AF205885; WN-India 1955b, AF196525; WN-India 1980, AF196526; WN-India 1958, AF196524; WN-Madagascar 1978, AF001559; WN-Madagascar 1988, AF001574; WN-Kenya, AF001571; WN-Madagascar 1986, AF001564; WN-Uganda 1959, AF001562; WN-C.Afr.Rep. 1972a, AF001563; WN-C.Afr.Rep. 1983, AF001557; WN-C.Afr.Rep. 1972b, AF001565; WN-Nigeria, M12294; WN-Uganda, AF001573; WN-Senegal 1990, AF001556; JE SA 14, U04522.



Origin of the West Nile Virus Responsible for an Outbreak of Encephalitis in the Northeastern United States

R. S. Lanciotti,^{1*} J. T. Roehrig,¹ V. Deubel,² J. Smith,³ M. Parker,³ K. Steele,³ B. Crise,³ K. E. Volpe,¹ M. B. Crabtree,¹ J. H. Scherret,⁴ R. A. Hall,⁴ J. S. MacKenzie,⁴ C. B. Cropp,¹ B. Panigrahy,⁵ E. Ostlund,⁵ B. Schmitt,⁵ M. Malkinson,⁶ C. Banet,⁶ J. Weissman,⁶ N. Komar,¹ H. M. Savage,¹ W. Stone,⁷ T. McNamara,⁸ D. J. Gubler¹

In late summer 1999, an outbreak of human encephalitis occurred in the northeastern United States that was concurrent with extensive mortality in crows (*Corvus* species) as well as the deaths of several exotic birds at a zoological park in the same area. Complete genome sequencing of a flavivirus isolated from the brain of a dead Chilean flamingo (*Phoenicopterus chilensis*), together with partial sequence analysis of envelope glycoprotein (E-glycoprotein) genes amplified from several other species including mosquitoes and two fatal human cases, revealed that West Nile (WN) virus circulated in natural transmission cycles and was responsible for the human disease. Antigenic mapping with E-glycoprotein-specific monoclonal antibodies and E-glycoprotein phylogenetic analysis confirmed these viruses as WN. This North American WN virus was most closely related to a WN virus isolated from a dead goose in Israel in 1998.

Persistent impacts of West Nile virus on North American bird populations

T. Luke George^{a,1}, Ryan J. Harrigan^{b,1,2}, Joseph A. LaManna^{c,1}, David F. DeSante^d, James F. Saracco^d, and Thomas B. Smith^{b,e}

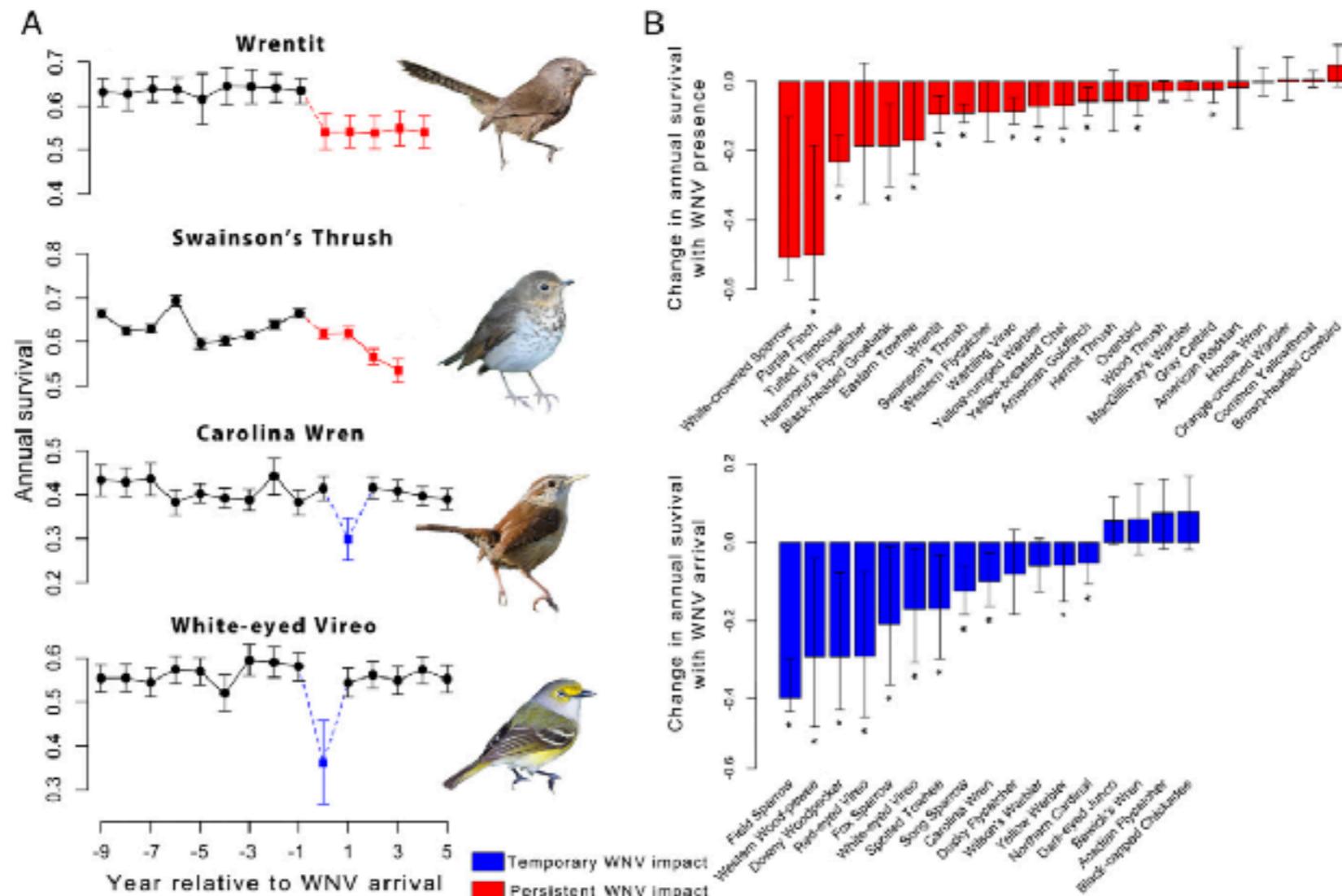


Fig. 1. The effects of WNV on landbird species across the continental United States. (*A*) Examples of temporary and persistent effects of WNV on model-averaged estimates of annual survival. In some species, such as the wrentit and Swainson's thrush, survival decreased (red) after the arrival of WNV, and remained low in subsequent years. In other species, such as the Carolina wren and white-eyed vireo, survival was reduced during the year of WNV arrival (blue), followed by recovery in subsequent years despite the disease persisting in the community (Materials and Methods). The year that WNV affected survival varied between species when the top-ranked model included a 1-y lag (Carolina wren). Time series were shorter for species where the majority of individuals were captured in the western United States where WNV arrived later (Swainson's thrush). Photos courtesy of (Top to Bottom) Harjeet Randhawa, Brian Plunkett, Nathan Corry, and Jacob Spendelow. (*B*) Change in annual survival because of WNV from top-ranked models for 40 landbird species (9 species did not have a WNV covariate in the top survival model). Species are shown as being affected by either the continued presence (red) or the arrival (blue) of WNV based on which variable was in the top-ranked model describing annual survival. Bars represent confidence intervals ($\pm 95\%$), and asterisks indicate a significant effect ($P < 0.05$) of WNV on survival.

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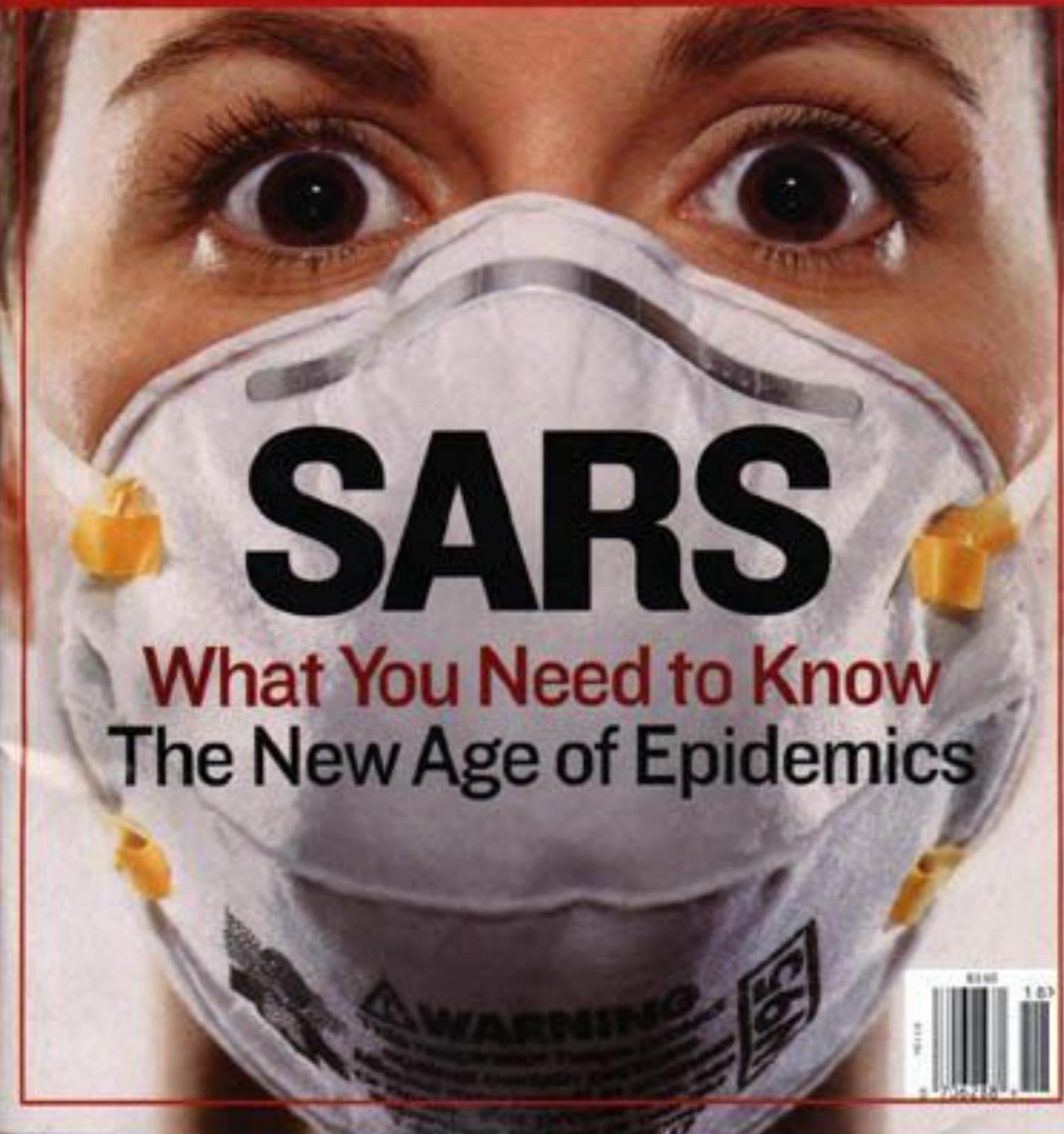
Newsweek

May 5, 2003

www.newsweek.com

SARS

What You Need to Know
The New Age of Epidemics

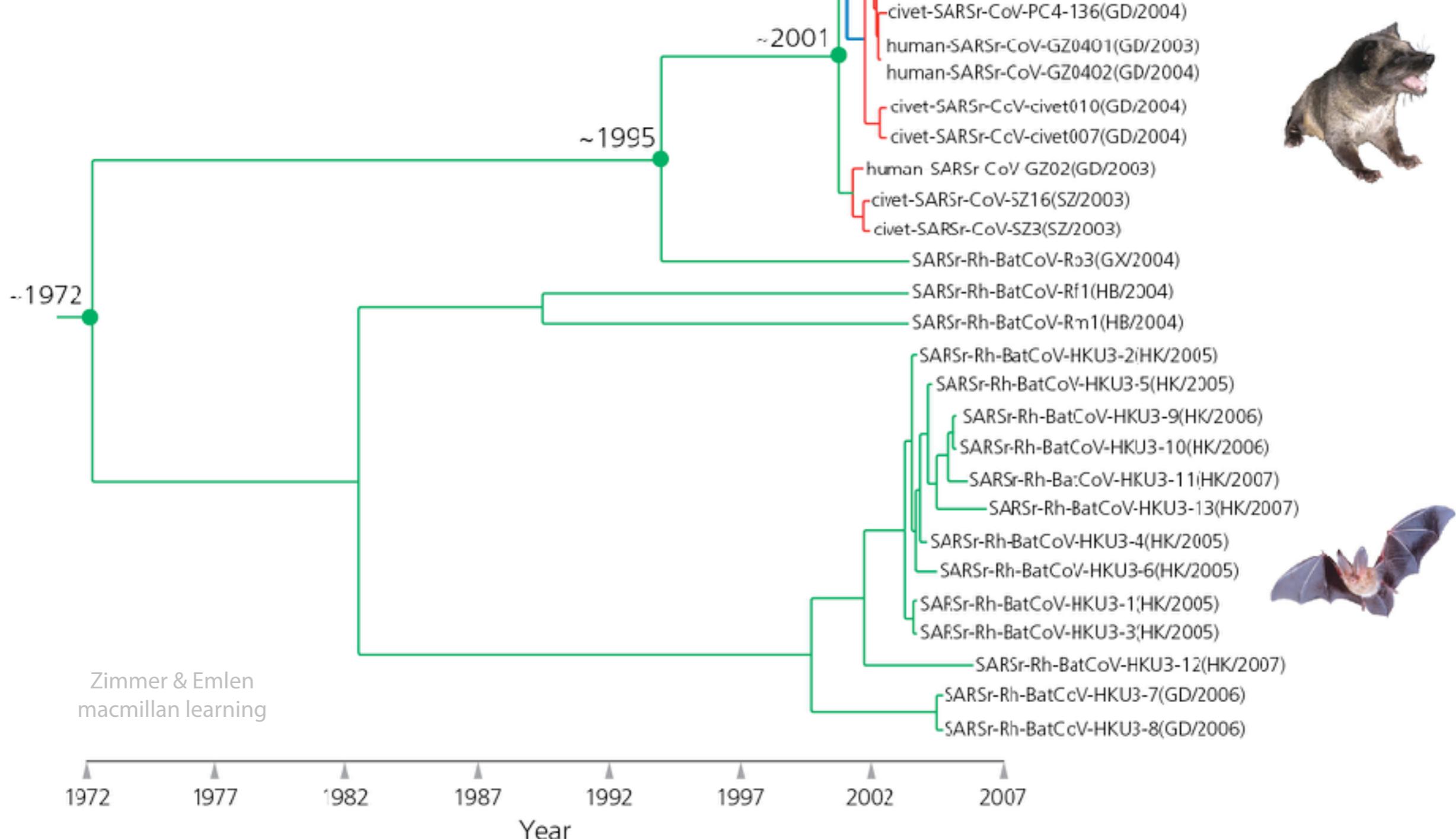


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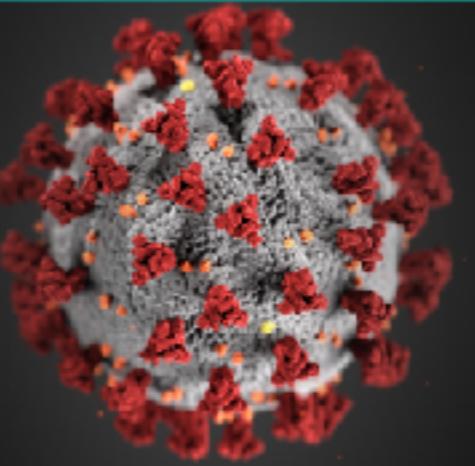
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Ecoepidemiology and Complete Genome Comparison of Different Strains of Severe Acute Respiratory Syndrome-Related *Rhinolophus* Bat Coronavirus in China Reveal Bats as a Reservoir for Acute, Self-Limiting Infection That Allows Recombination Events^{V†}

Susanna K. P. Lau,^{1,2,3,4,‡} Kenneth S. M. Li,^{4,‡} Yi Huang,⁴ Chung-Tong Shek,⁵ Herman Tse,^{1,2,3,4} Ming Wang,⁶ Garnet K. Y. Choi,⁴ Huifang Xu,⁶ Carol S. F. Lam,⁴ Rongtong Guo,⁶ Kwok-Hung Chan,⁴ Bo-Jian Zheng,⁴ Patrick C. Y. Woo,^{1,2,3,4,*} and Kwok-Yung Yuen^{1,2,3,4*}



CORONAVIRUS DISEASE
2019 (COVID-19)



www.cdc.gov/Covid19



THE CONVERSATION

PUBLIC HEALTH

Snakes Could Be the Original Source of the New Coronavirus Outbreak in China

A study of the virus's genetic sequence suggests similarities to that seen in snakes, but the origin must still be verified

By Hailao Guo, Guangxiang "George" Luo, Shou-Jiang Gao, The Conversation US on January 22, 2020



Credit: Getty Images

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nCoV's relationship to bat coronaviruses & recombination signals (no snakes) - no evidence the 2019-nCoV lineage is recombinant

Novel 2019 coronavirus | nCoV-2019 Evolutionary History



david.l.robertson

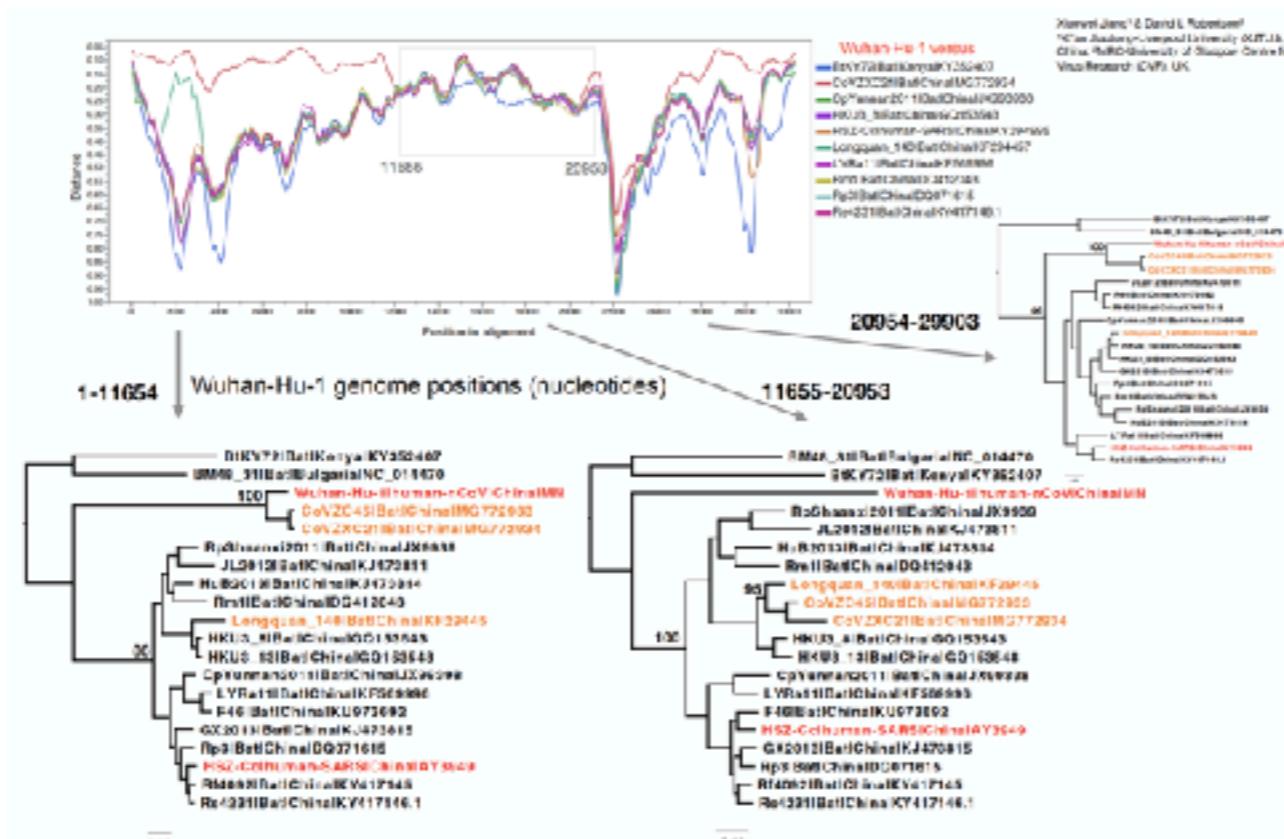
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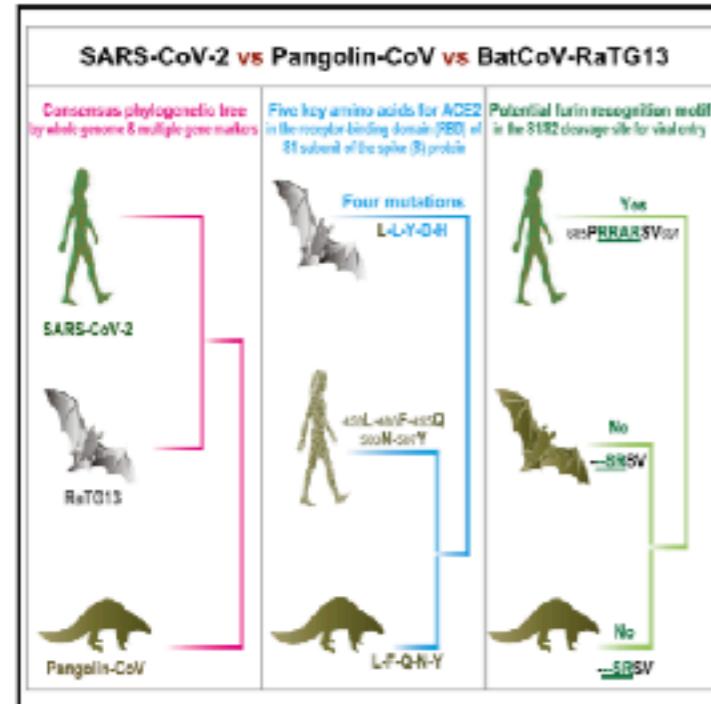
Jan 22

With Xiaowei Jiang at XJTLU we've carried out a preliminary evolutionary analysis to characterise the evolutionary origins of the Wuhan virus, nCoV. Focus of our analysis is on the Wuhan-Hu-1 virus (accession no. MN908947, released on GenBank by Shanghai Public Health Clinical Center and School of Public Health, Fudan University, Shanghai, China) as all nCoV cluster together so will share the same evolutionary ancestry. It's clear from phylogenetic analysis the new human virus is most closely related to bat coronaviruses in the Betacoronaviruses genera. While this is apparent from both the previously reported BLAST and full-genome phylogenetic analysis the closest related bat viruses (CoVZC45 and CoVZXC21) are in fact recombinants with shared breakpoints either side of ORF1b:



Top, diversity plot across the Wuhan-Hu-1 genome alignment in 800 nucleotide windows, incremented 50 nt (generated using RDP4). Complete genomes were aligned with MAFFT using data from VPF and GenBank. Phylogenetic trees from regions detected by the HyPhy software GARD to be between recombination locations are shown. Note, exact breakpoints location's are dependent on reference sets used. The trees were inferred using PhyML, with a 'BEET' tree search and HKY substitution model. The same options were used to perform bootstrapping (1000 replicates). Trees were visualised with FigTree.

Graphical Abstract



Highlights

- Pangolin-CoV is 91.02% identical to SARS-CoV-2 at the whole-genome level
- Pangolin-CoV is the second closest relative of SARS-CoV-2 behind RaTG13
- Five key amino acids in the RBD are consistent between Pangolin-CoV and SARS-CoV-2
- Only SARS-CoV-2 contains a potential cleavage site for furin proteases

Zhang et al., 2020, Current Biology 30, 1–6
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<https://doi.org/10.1016/j.cub.2020.03.002>

Report

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correspondence

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-2³ (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 deaths⁵.

SARS-CoV-2 is the seventh coronavirus known to infect humans; SARS-CoV, MERS-CoV 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.

Notable features of the SARS-CoV-2 genome

Our comparison of alpha- and betacoronaviruses identifies two notable genomic features of SARS-CoV-2: (i) on the basis of structural studies^{7–9} and biochemical experiments^{1,9,10}, SARS-CoV-2 appears to be optimized for binding to the human receptor ACE2; and (ii) the spike protein of SARS-CoV-2 has a functional polybasic (furin) cleavage site at the S1–S2 boundary through the insertion of 12 nucleotides⁸, which additionally led to the predicted acquisition of three O-linked glycans around the site.

1. Mutations in the receptor-binding domain of SARS-CoV-2. The receptor-binding domain (RBD) in the spike protein is the most variable part of the coronavirus genome¹². Six RBD amino acids have been shown to be critical for binding to ACE2 receptors and for determining the host range of SARS-CoV-like viruses⁷. With coordinates based on SARS-CoV, they are Y442, L472, N479, D480, T487 and Y4911, which correspond to L455, F486, Q493, S494, N501 and Y505 in SARS-CoV-2⁷. Five of these six residues differ between SARS-CoV-2 and SARS-CoV (Fig. 1a). On the basis of structural studies^{7–9} and biochemical experiments^{1,9,10}, SARS-CoV-2 seems to have an RBD that binds with high affinity to ACE2 from humans, ferrets, cats and other species with high receptor homology⁷.

While the analyses above suggest that SARS-CoV-2 may bind human ACE2 with high affinity, computational analyses predict that the interaction is not ideal⁷ and that the RBD sequence is different from those shown in SARS-CoV to be optimal for receptor binding^{7,11}. Thus, the high-affinity binding of the SARS-CoV-2 spike protein to human ACE2 is most likely the result of natural selection on a human or human-like ACE2 that permits another optimal binding solution to arise. This is strong evidence that SARS-CoV-2 is not the product of purposeful manipulation.

2. Polybasic furin cleavage site and O-linked glycans.

The second notable feature of SARS-CoV-2 is a polybasic cleavage site (RRAR) at the junction of S1 and S2, the two subunits of the spike⁸ (Fig. 1b). This allows effective cleavage by furin and other proteases and has a role in determining viral infectivity and host range¹². In addition, a leading proline is also inserted at this site in SARS-CoV-2; thus, the inserted sequence is PRRA (Fig. 1b). The turn created by the proline is predicted to result in the addition of O-linked glycans to S673, T678 and S686, which flank the cleavage site and are unique to SARS-CoV-2 (Fig. 1b). Polybasic cleavage sites have not been observed in related ‘lineage B’ betacoronaviruses, although other human betacoronaviruses, including HKU1 (lineage A), have those sites and predicted O-linked glycans¹³. Given the level of genetic variation in the spike, it is likely that SARS-CoV-2-like viruses with partial or full polybasic cleavage sites will be discovered in other species.

The functional consequence of the polybasic cleavage site in SARS-CoV-2 is unknown, and it will be important to determine its impact on transmissibility and pathogenesis in animal models. Experiments with SARS-CoV have shown that insertion of a furin cleavage site at the S1–S2 junction enhances cell–cell fusion without affecting viral entry¹⁴. In addition, efficient cleavage of the MERS-CoV spike enables MERS-like coronaviruses from bats to infect human cells¹⁵. In avian influenza viruses, rapid replication and transmission in highly dense chicken populations selects for the acquisition of polybasic cleavage sites in the hemagglutinin (HA) protein¹⁶, which serves a function similar to that of the coronavirus spike protein. Acquisition of polybasic cleavage sites in HA, by insertion or recombination, converts

low-pathogenicity avian influenza viruses into highly pathogenic forms¹⁶. The acquisition of polybasic cleavage sites by HA has also been observed after repeated passage in cell culture or through animals¹⁷.

The function of the predicted O-linked glycans is unclear, but they could create a ‘mucin-like domain’ that shields epitopes or key residues on the SARS-CoV-2 spike protein¹⁸. Several viruses utilize mucin-like domains as glycan shields involved in immunoevasion¹⁸. Although prediction of O-linked glycosylation is robust, experimental studies are needed to determine if these sites are used in SARS-CoV-2.

Theories of SARS-CoV-2 origins

It is improbable that SARS-CoV-2 emerged through laboratory manipulation of a related SARS-CoV-like coronavirus. As noted above, the RBD of SARS-CoV-2 is optimized for binding to human ACE2 with an efficient solution different from those previously predicted^{7,11}. Furthermore, if genetic manipulation had been performed, one of the several reverse-genetic systems available for betacoronaviruses would probably have been used¹⁹. However, the genetic data irrefutably show that SARS-CoV-2 is not derived from any previously used virus backbone²⁰. Instead, we propose two scenarios that can plausibly explain the origin of SARS-CoV-2: (i) natural selection in an animal host before zoonotic transfer; and (ii) natural selection in humans following zoonotic transfer. We also discuss whether selection during passage could have given rise to SARS-CoV-2.

1. Natural selection in an animal host before zoonotic transfer.

As many early cases of COVID-19 were linked to the Huanan market in Wuhan^{1,2}, it is possible that an animal source was present at this location. Given the similarity of SARS-CoV-2 to bat SARS-CoV-like coronaviruses², it is likely that bats serve as reservoir hosts for its progenitor. Although RaTG13, sampled from a *Rhinolophus affinis* bat¹, is ~96% identical overall to SARS-CoV-2, its spike diverges in the RBD, which suggests that it may not bind efficiently to human ACE2⁷ (Fig. 1a).

Malayan pangolins (*Manis javanica*) illegally imported into Guangdong province contain coronaviruses similar to SARS-CoV-2²¹. Although the RaTG13 bat virus remains the closest to SARS-CoV-2 across the

Coronavirus: Revenge of the Pangolins?

China has banned the trade of wildlife, suspecting that exotic animals infected humans. What will that really do?

By Wufei Yu

Mr. Yu is a contributor to Outside Magazine.

March 5, 2020



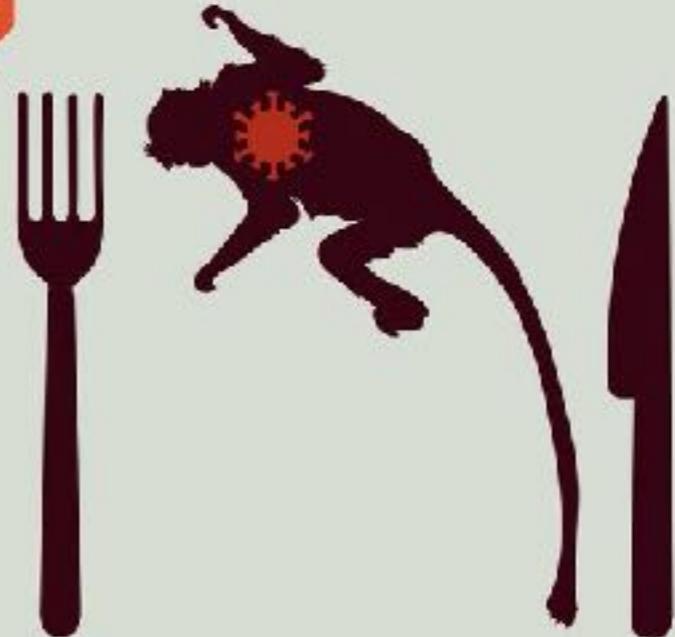
Let's

STOP



WILDLIFE TRADE

STOP



WILDLIFE CONSUMPTION

STOP



DESTROYING NATURE

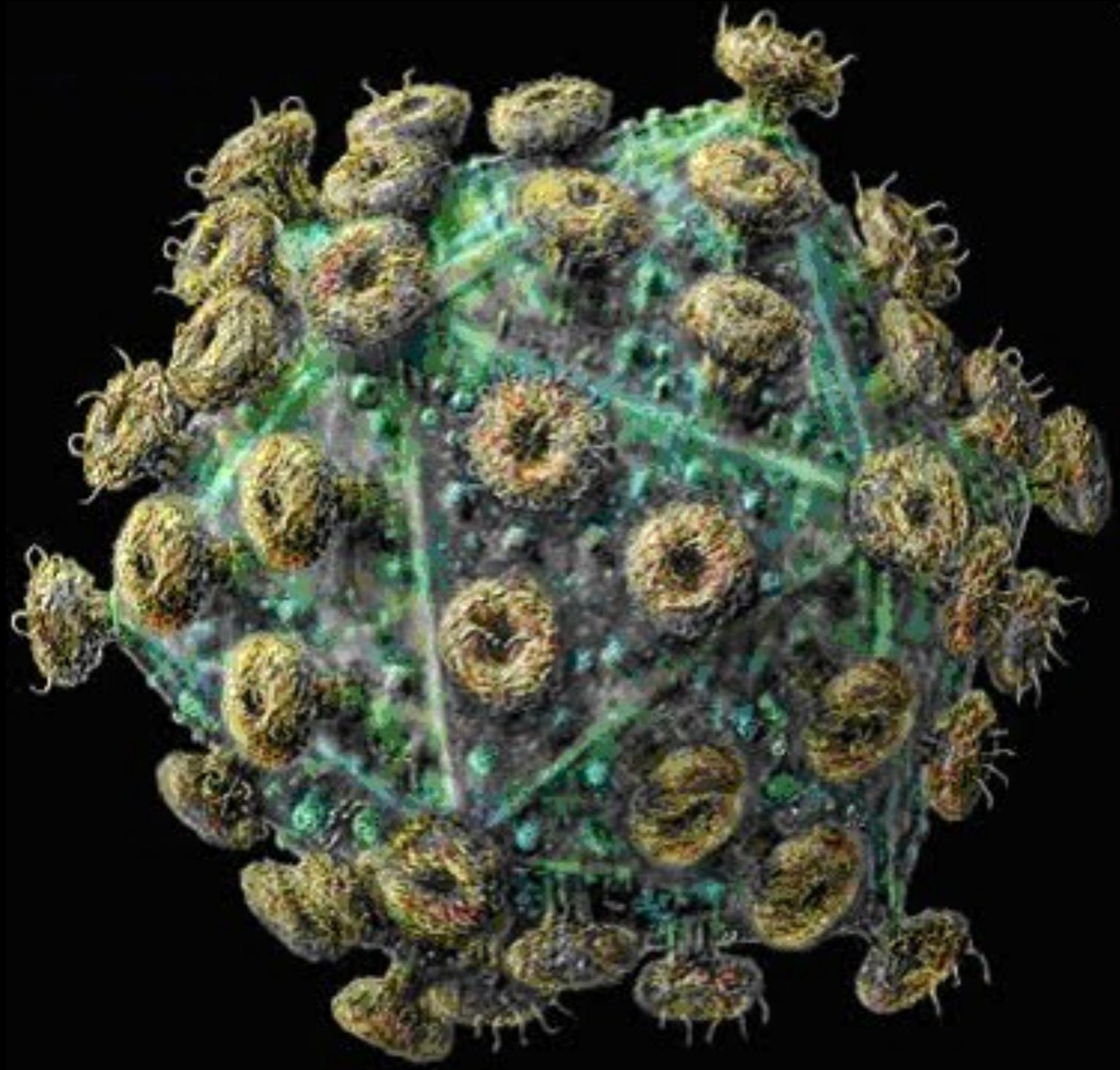
TO STOP PANDEMICS



Wildlife
Conservation
Society



GLOBAL
WILDLIFE
CONSERVATION



<https://co.pinterest.com/pin/512214157611834174/>

varios orígenes de tipos de virus HIV humanos a partir de primates



Cold Spring Harbor Perspectives in Medicine
www.perspectivesinmedicine.org

Origins of HIV and the AIDS Pandemic

Paul M. Sharp¹ and Beatrice H. Hahn²

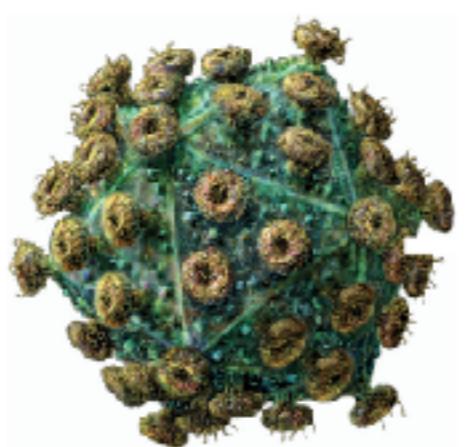
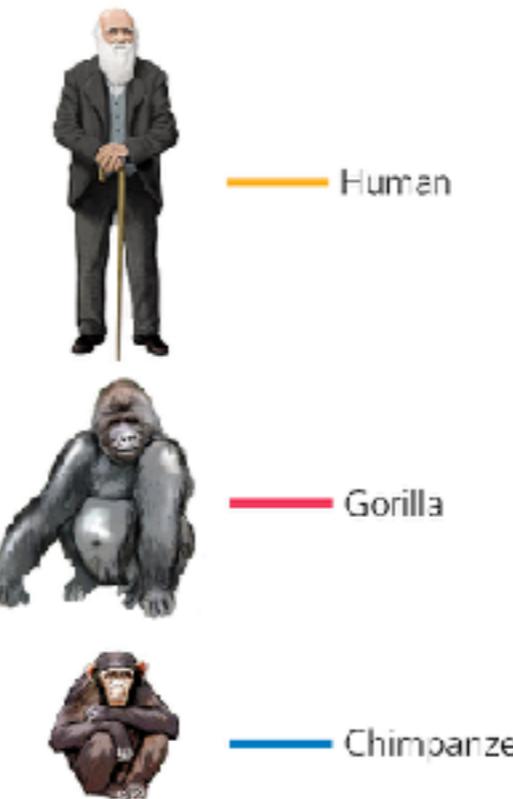
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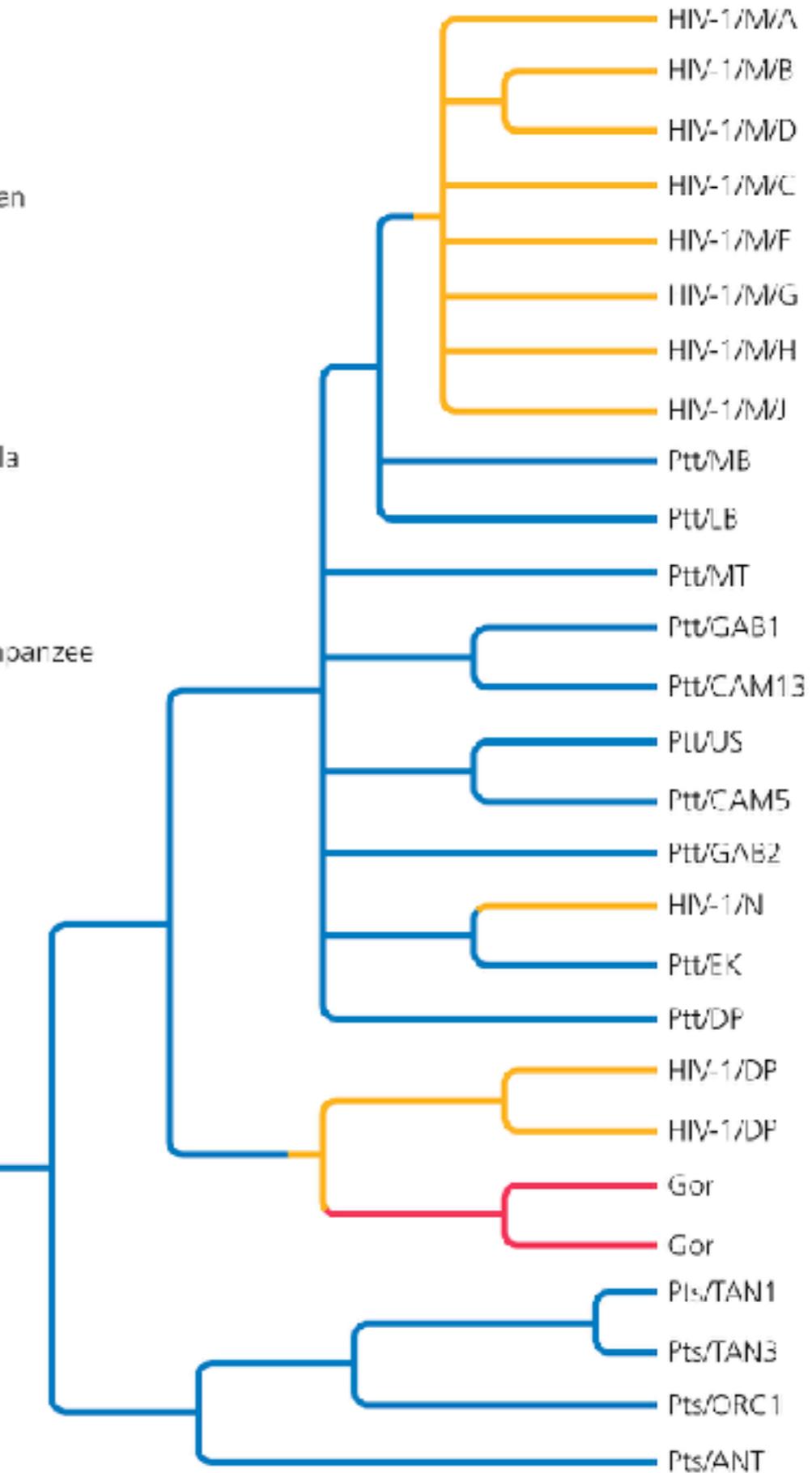
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Acquired immunodeficiency syndrome (AIDS) of humans is caused by two lentiviruses, human immunodeficiency viruses types 1 and 2 (HIV-1 and HIV-2). Here, we describe the origins and evolution of these viruses, and the circumstances that led to the AIDS pandemic. Both HIVs are the result of multiple cross-species transmissions of simian immunodeficiency viruses (SIVs) naturally infecting African primates. Most of these transfers resulted in viruses that spread in humans to only a limited extent. However, one transmission event, involving SIVcpz from chimpanzees in southeastern Cameroon, gave rise to HIV-1 group M—the principal cause of the AIDS pandemic. We discuss how host restriction factors have shaped the emergence of new SIV zoonoses by imposing adaptive hurdles to cross-species transmission and/or secondary spread. We also show that AIDS has likely afflicted chimpanzees long before the emergence of HIV. Tracing the genetic changes that occurred as SIVs crossed from monkeys to apes and from apes to humans provides a new framework to examine the requirements of successful host switches and to gauge future zoonotic risk.

HOST OF VIRUS



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Origin of the HIV-1 group O epidemic in western lowland gorillas

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Contributed by Beatrice H. Hahn, February 2, 2015 (sent for review December 16, 2014; reviewed by Catherine A. Brennan and Tony L. Goldberg)

HIV-1, the cause of AIDS, is composed of four phylogenetic lineages, groups M, N, O, and P, each of which resulted from an independent cross-species transmission event of simian immunodeficiency viruses (SIVs) infecting African apes. Although groups M and N have been traced to geographically distinct chimpanzee communities in southern Cameroon, the reservoirs of groups O and P remain unknown. Here, we screened fecal samples from western lowland ($n = 2,611$), eastern lowland ($n = 103$), and mountain ($n = 218$) gorillas for gorilla SIV (SIVgor) antibodies and nucleic acids. Despite testing wild troops throughout southern Cameroon ($n = 14$), northern Gabon ($n = 16$), the Democratic Republic of Congo ($n = 2$), and Uganda ($n = 1$), SIVgor was identified at only four sites in southern Cameroon, with prevalences ranging from 0.8–22%. Amplification of partial and full-length SIVgor sequences revealed extensive genetic diversity, but all SIVgor strains were derived from a single lineage within the chimpanzee SIV (SIVcpz) radiation. Two fully sequenced gorilla viruses from southwestern Cameroon were very closely related to, and likely represent the source population of, HIV-1 group P. Most of the genome of a third SIVgor strain, from central Cameroon, was very closely related to HIV-1 group O, again pointing to gorillas as the immediate source. Functional analyses identified the cytidine deaminase APOBEC3G as a barrier for chimpanzee-to-gorilla, but not gorilla-to-human, virus transmission. These data indicate that HIV-1 group O, which spreads epidemically in west central Africa and is estimated to have infected around 100,000 people, originated by cross-species transmission from western lowland gorillas.

AIDS | HIV-1 | gorilla | SIVgor | zoonotic transmission

AIDS is caused by HIV-1 and HIV-2, which are derived from a clade of lentiviruses [simian immunodeficiency viruses (SIVs)] found naturally in more than 40 species of nonhuman primates in sub-Saharan Africa (1, 2). These SIVs mostly fall into host-specific clades, but they have occasionally jumped species and spread successfully in new hosts. Of particular interest, chimpanzees (*Pan troglodytes*) acquired two distinct lineages of SIV from two different monkey species; all known strains of chimpanzee SIV (SIVcpz) are derived from a hybrid formed by recombination between these two viruses (3). The spread of this virus in chimpanzees appears to have occurred comparatively recently, because only two closely related subspecies in Central Africa are infected, whereas two other subspecies are not (4–8). Subsequently, strains of SIVcpz from one chimpanzee subspecies (*Pan troglodytes troglodytes*) have been subject to further transmission both to humans, leading to HIV-1, and to western

gorillas (*Gorilla gorilla*), giving rise to gorilla SIV (SIVgor) (4, 9). The limited number of strains of SIVgor characterized so far form a single clade, but HIV-1 strains fall into four phylogenetically distinct groups, each of which must reflect a separate cross-species transmission from apes (1). These four zoonotic events have had very different outcomes. One gave rise to group M, the cause of the AIDS pandemic, which has infected more than 40 million people and spread across Africa and throughout the rest of the world. At the other extreme, group N and P viruses have only been found in small numbers of individuals from Cameroon: group N in fewer than 20 individuals (10) and group P in only two individuals (11, 12). Group O, although not nearly as prevalent as group M, has nonetheless caused a substantial epidemic. Although largely restricted to west central

Significance

Understanding emerging disease origins is important to gauge future human infection risks. This is particularly true for the various forms of the AIDS virus, HIV-1, which were transmitted to humans on four independent occasions. Previous studies identified chimpanzees in southern Cameroon as the source of the pandemic M group, as well as the geographically more restricted N group. Here, we show that the remaining two groups also emerged in southern Cameroon but had their origins in western lowland gorillas. Although group P has only been detected in two individuals, group O has spread extensively throughout west central Africa. Thus, both chimpanzees and gorillas harbor viruses that are capable of crossing the species barrier to humans and causing major disease outbreaks.

MICROBIOLOGY

Author contributions: M.D., A.A., P.M.S., B.H.H., E.D., E.M.N., and M.P. designed research; M.D., A.A., A.E., V.B., F.L., L.E., M.O., M.L., and V.A.S. performed research; V.B., F.L., N.T., F.H.L., C.B., N.F.M., M.M.R., M.G., and E.M.N. contributed new reagents/analytic tools; M.D., A.A., G.H.L., A.C., M.O., M.L., V.A.S., and M.P. analyzed data; and M.D., A.A., P.M.S., B.H.H., and M.P. wrote the paper.

Reviewers: C.A.B., Abbott Diagnostics; and T.L.G., University of Wisconsin.

The authors declare no conflict of interest.

Freely available online through the PNAS open access option.

Data deposition: The sequences reported in this paper have been deposited in the GenBank database (accession nos. KP004989–KP004991 and KP004992–KP004999).

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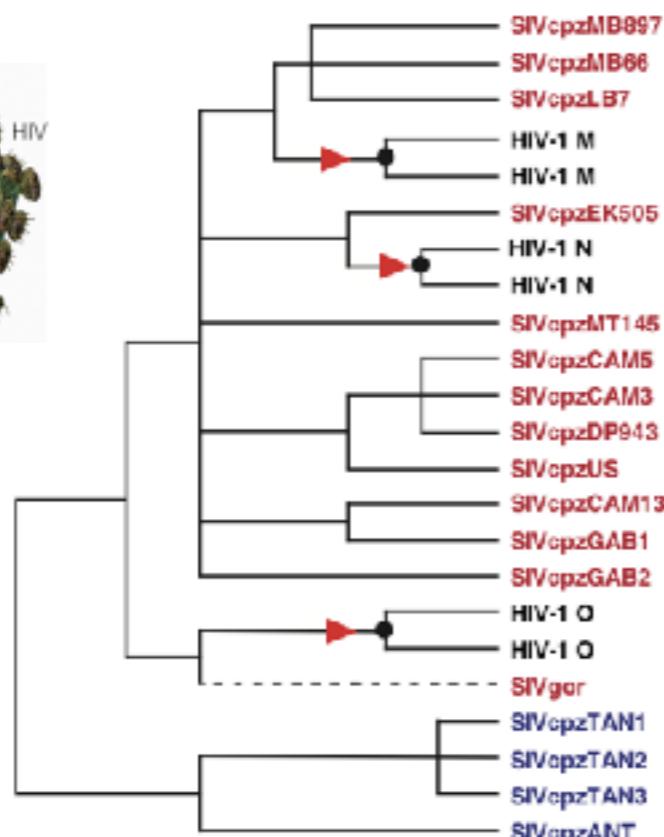
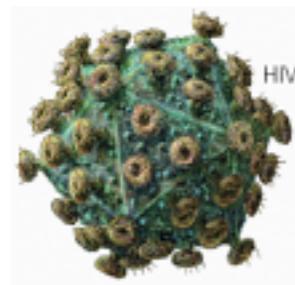
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selección natural y evolución convergente del virus HIV

Adaptation of HIV-1 to Its Human Host

Louise V. Wain,¹ Elizabeth Bailes,¹ Frederic Bibollet-Ruche,² Julie M. Decker,² Brandon F. Keele,² Fran Van Heuverswyn,³ Yingying Li,² Jun Takehisa,² Eitel Mpoudi Ngole,⁴ George M. Shaw,² Martine Peeters,³ Beatrice H. Hahn,² and Paul M. Sharp¹



"as all three lineages of HIV-1 evolved from chimp-virus ancestors, each lineage acquired the same mutation encoding the same new amino acid in the same position in the same protein"

(Zimmer & Emlen 2013)

p17 Gag	26	30	34
HIV-1 group M ancestor	K	K	Y
HIV-1 group N ancestor	-	-	-
HIV-1 group O ancestor	-	-	-
SIVcpzPtt (MB66)	-	-	M
SIVcpzPtt (MB897)	-	-	M
SIVcpzPtt (LB7)	R	-	M
SIVcpzPtt (MT145)	-	-	M
SIVcpzPtt (EK505)	-	-	M
SIVcpzPtt (CAM13)	R	-	M
SIVcpzPtt (CAM3)	-	-	M
SIVcpzPtt (CAM5)	-	-	M
SIVcpzPtt (DP943)	-	-	M
SIVcpzPtt (GAB1)	R	-	M
SIVcpzPtt (GAB2)	R	-	M
SIVcpzPts (ANT)	-	-	I
SIVcpzPts (TAN1)	R	-	L
SIVcpzPts (TAN2)	R	-	L
SIVcpzPts (TAN3)	R	-	L
chimpanzee HIV-1 JC16	-	-	M
chimpanzee HIV-1 NC7	-	-	M

FIG. 2.—Species-specific adaptive changes in the matrix protein (Gag p17). Sequences from a region in the N-terminal basic domain of HIV-1/SIVcpz matrix proteins reveal a site (boxed) that differs between the HIV-1 group ancestors (R, Arg) and chimpanzee viruses (M, Met/L, Leu). Dashes indicate amino acid identity to the sequence at the top. Clones JC16 and NC7 were isolated from two chimpanzees experimentally infected with HIV-1 (Mwaengo and Novembre 1998).

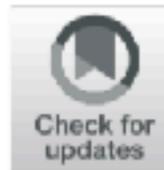




RESEARCH

Open Access

The evolution of a bat population with white-nose syndrome (WNS) reveals a shift from an epizootic to an enzootic phase



Craig L. Frank^{1*} , April D. Davis² and Carl Herzog³

Abstract

Background: White-nose Syndrome (WNS) is a mycosis caused by a cutaneous infection with the fungus *Pseudogymnoascus destructans* (*Pd*). It produces hibernation mortality rates of 75–98% in 4 bats: *Myotis lucifugus*, *M. septentrionalis*, *M. sodalis*, and *Perimyotis subflavus*. These high mortality rates were observed during the first several years after the arrival of *P. destructans* at a hibernation site. Mortality is caused by a 60% decrease in torpor bout duration, which results in a premature depletion of depot fat prior to spring.

Results: Little is known about the long-term effects of *Pd* on torpor and mortality, thus we conducted a 9-year study on *M. lucifugus* at 5 of the hibernation sites where *Pd* first appeared in North America during the winter of 2007–08. The *M. lucifugus* hibernating at one of these sites one year after the arrival of *Pd* (2008–09) had: a) a mean torpor bout duration of 7.6 d, b) no depot fat reserves by March, and c) an apparent over-winter mortality rate of 88%. The *M. lucifugus* hibernating at this same site 6–9 years after the arrival of *Pd*, in contrast, had: a) a mean torpor bout duration of 14.7 d, b) depot fat remaining in March, and c) an apparent mortality rate of 50%. The number of *M. lucifugus* hibernating at 2 of these sites has consistently increased since 2010 and is now more than 3.0-fold higher than the number remaining after the winter of 2008–09.

Conclusions: These findings indicate that this population of *M. lucifugus* has evolved mechanisms to hibernate well in the presence of *Pd*, thus reducing over-winter mortality.

Keywords: White-nose syndrome, *Myotis lucifugus*, *Pseudogymnoascus destructans*, Hibernation, Torpor, Mycosis

evolución molecular y selección dentro de pacientes

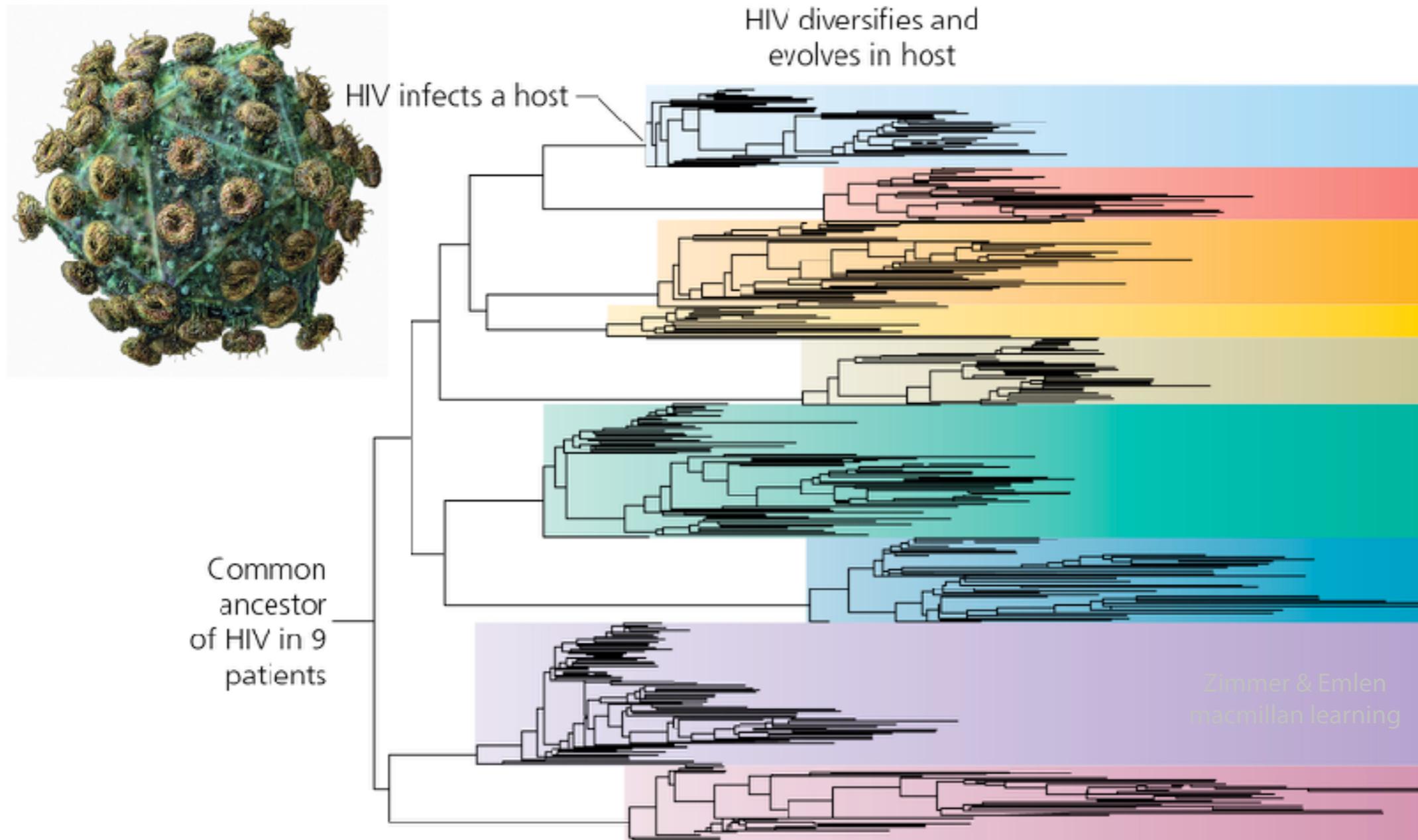


Figure 4 | Contrasting patterns of intra- and inter-host evolution of HIV. The tree was constructed using the NEIGHBOUR-JOINING METHOD on envelope gene-sequence data that was taken from nine HIV-infected patients⁴⁸ (a total of 1,195 sequences, 822 base pairs in length), with those viruses sampled from each patient depicted by a different colour. In each case, intra-host HIV evolution is characterized by continual immune-driven selection, such that there is a successive selective replacement of strains through time, with relatively little genetic diversity at any time point. By contrast, there is little evidence for positive selection at the population level (bold lines connecting patients), so that multiple lineages are able to coexist at any time point.

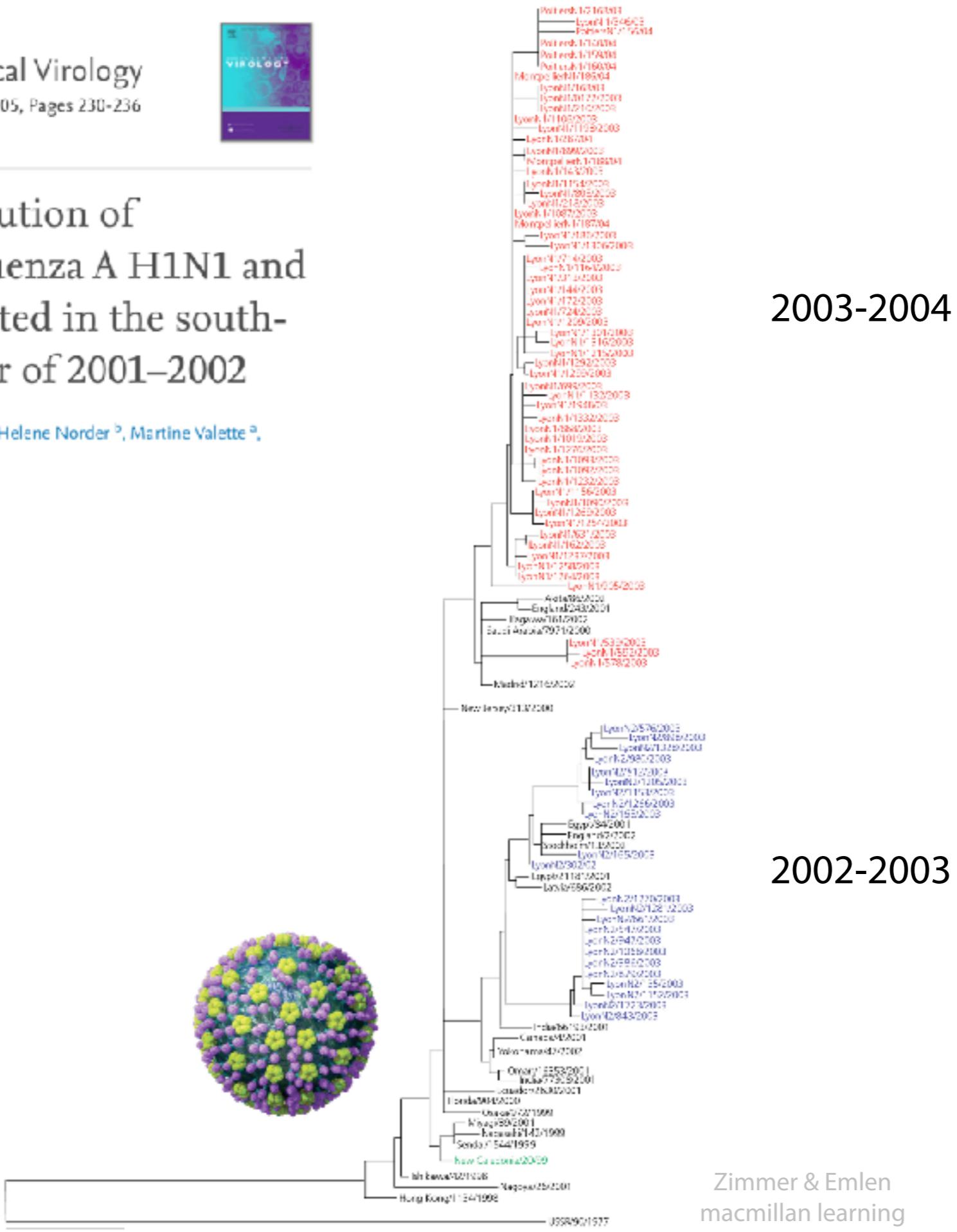
THE CAUSES AND CONSEQUENCES OF HIV EVOLUTION

Andrew Rambaut^a, David Peacock^b, Keith A. Crandall^c and Edward C. Holmes^d



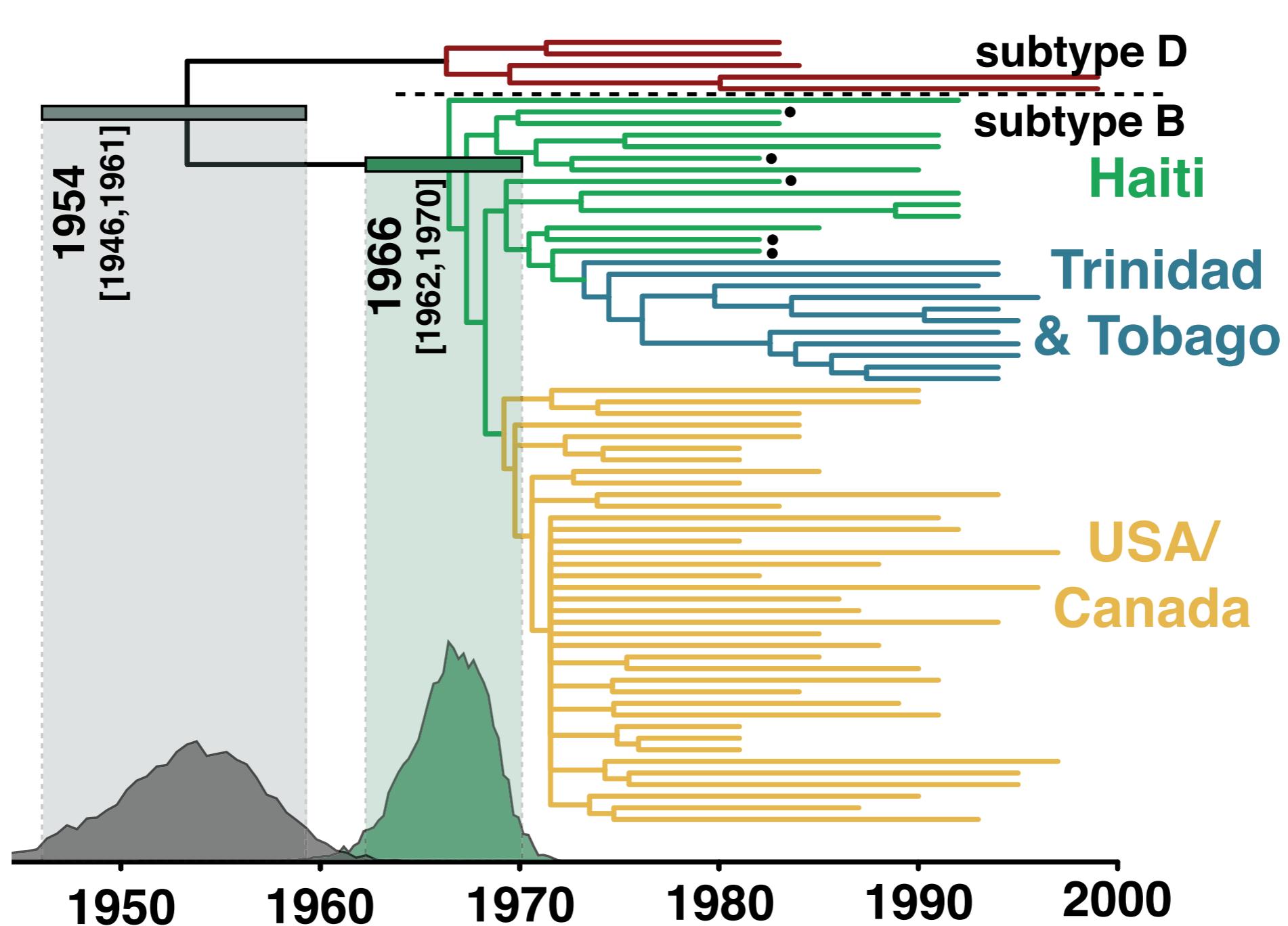
Divergent genetic evolution of hemagglutinin in influenza A H1N1 and A H1N2 subtypes isolated in the south-France since the winter of 2001–2002

Shaker Al Farees², Gaëlle Cartet³, Olivier Ferraris³, Hélène Norder³, Martine Valette³, Bruno Lina^{2,3,4}



Zimmer & Emlen
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virus, filogenias y la temporalidad de epidemias: HIV-1 M subtipo B en USA



The emergence of HIV/AIDS in the Americas
and beyond

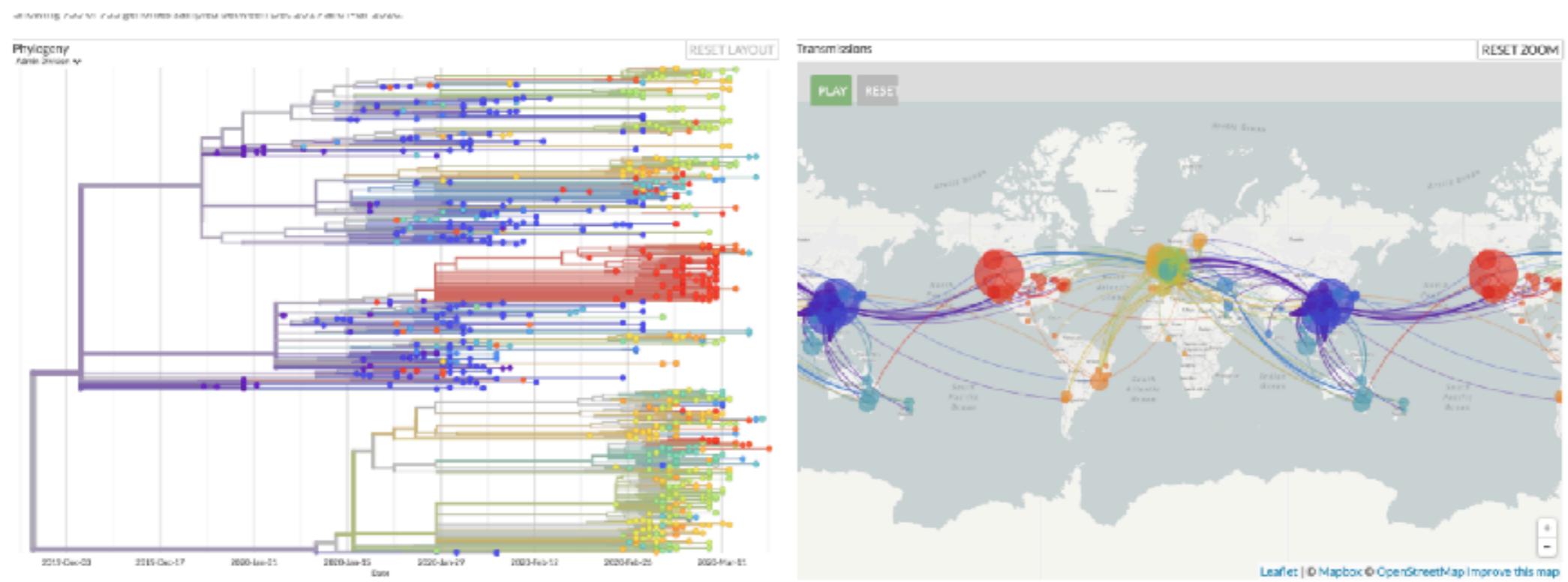
M. Thomas P. Gilbert*,†, Andrew Rambaut‡, Gabriela Masiuk*, Thomas J. Spira§, Arthur E. Pitchenik†,
and Michael Worobey*†

<https://github.com/cdanielcadena/evolucion>

Evolución UniAndes 2020-10

Profesor: C. Daniel Cadena (ccadena@uniandes.edu.co)
Asistente Graduada: María Alejandra Meneses (ma.meneses10@uniandes.edu.co)
Materiales para curso virtual de Evolución, Universidad de los Andes
Horario: martes, miércoles y jueves, 9:30-10:20 am
Aula Virtual en SicuaPlus

La Teoría Evolutiva Aplicada a Comprender una Pandemia: el caso de COVID-19



Distribución geográfica y filogenia basada en genomas del corononavirus SARS-CoV-2, causante de la pandemia de COVID-19. Imagen tomada el 23 de marzo de 2020 del proyecto [Nexstrain](#).

Objetivos de aprendizaje

- Comprender cómo los análisis filogenéticos pueden emplearse para monitorear la expansión de una enfermedad infecciosa emergente.
- Usando como estudio de caso la pandemia de COVID-19, conocer cómo conceptos fundamentales en evolución pueden aplicarse a un problema de salud pública con importantes implicaciones para la sociedad.

Competencias a desarrollar

- Habilidad para hacer inferencias biogeográficas a partir de árboles filogenéticos.
- Capacidad de divulgar información científica para un público amplio mediante una infografía.

Instrucciones para los estudiantes

Martes 24 de marzo

Vivimos una coyuntura única en la historia reciente de la humanidad. Esta coyuntura supone enormes desafíos para nuestra sociedad, pero al mismo tiempo representa una oportunidad de aplicar nuestros conocimientos y adquirir conocimientos nuevos en un contexto de gran importancia científica y social. Vamos a aprovechar esa oportunidad para estudiar la aplicación de herramientas que ya hemos trabajado en el curso (i.e. árboles filogenéticos) y para aprender sobre el papel que conceptos fundamentales en evolución juegan en el origen y propagación de un agente infeccioso. Dichos conceptos incluyen selección natural, adaptación, evolución molecular, especiación y coevolución, entre otros. Comenzaremos la fase virtual de nuestro curso con una clase dictada por el profesor a través del aula virtual de SicuaPlus acerca de aplicaciones del análisis filogenético para estudios de evolución de virus y otros patógenos que afectan la vida silvestre y a los humanos.