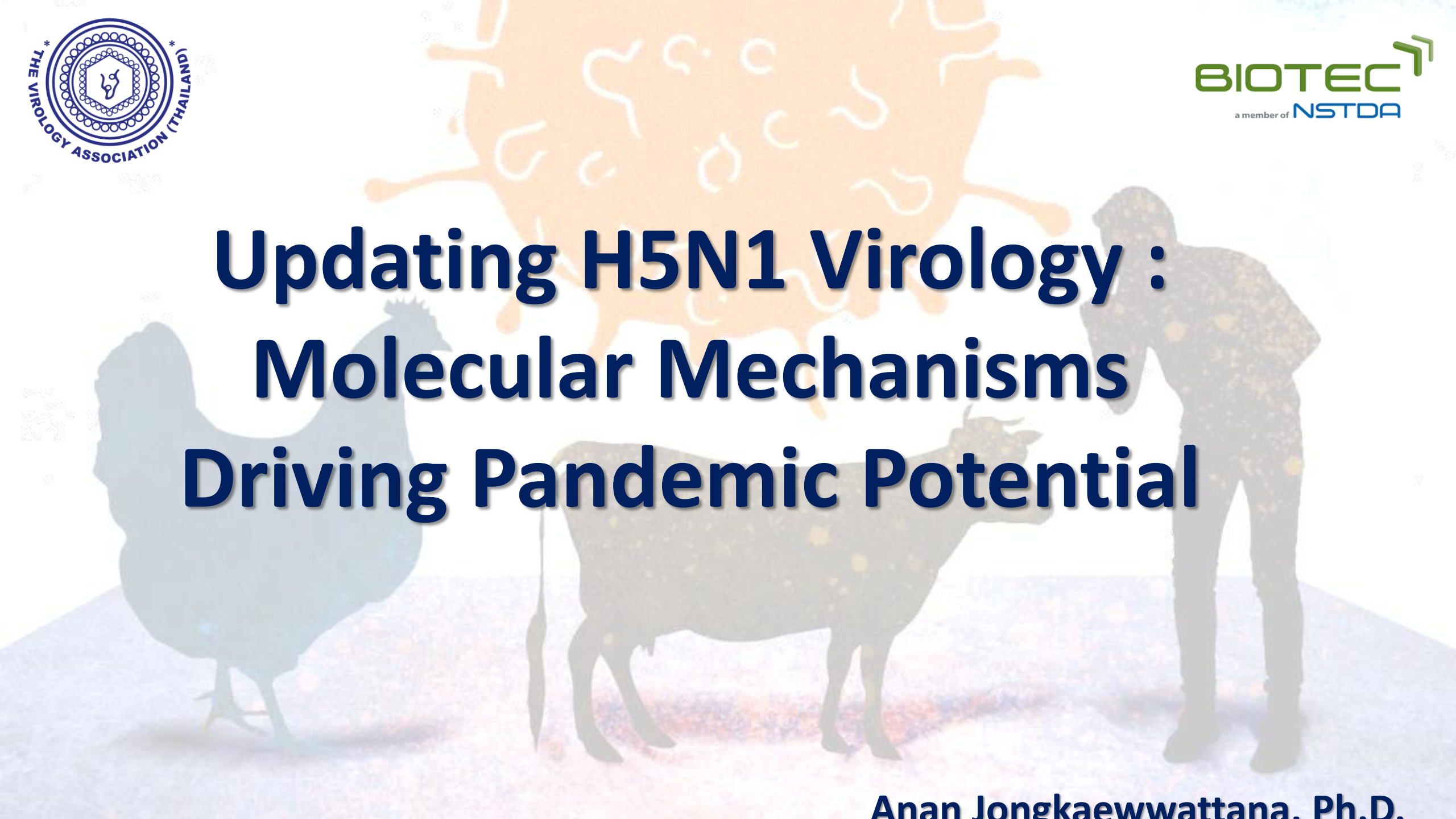


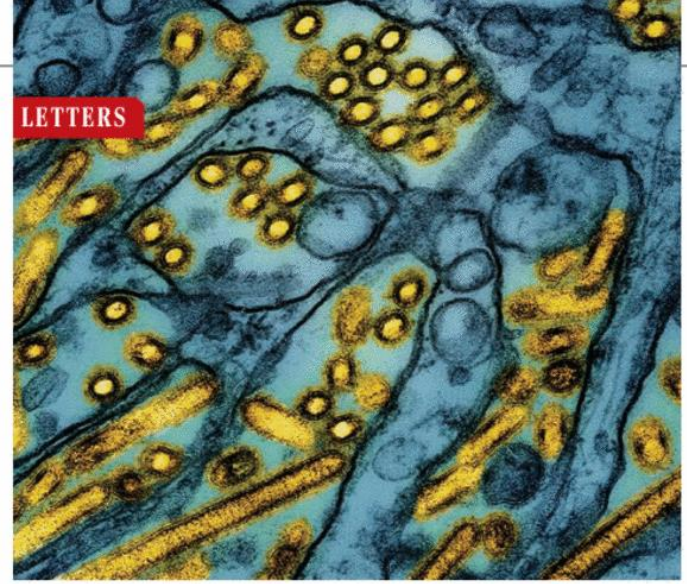
Updating H5N1 Virology : Molecular Mechanisms Driving Pandemic Potential

Anan Jongkaewwattana, Ph.D.

News

The world should prepare now for a potential H5N1 flu pandemic, experts warn

• 7th March 2025



An electron microscope image shows avian influenza A H5N1 virus particles (yellow) in epithelial cells (blue).

PHOTO: CDC/NIAID/VIAAP

Edited by **Jennifer Sills**

Prepare now for a potential H5N1 pandemic

The H5N1 virus has crossed species and adapted to mammalian hosts, including dairy cattle, causing widespread exposure and sporadic human illness (*1*). Although most cases have been mild, H5N1 can cause severe disease (*2*). Given H5N1's potential to spread, urgent action is needed to address pandemic preparedness gaps.

Rapid influenza vaccine availability is highly constrained by currently approved technologies, such as protein-based vaccines (*3*, *4*). Vaccine availability is also slowed by the time required to conduct immunogenicity and efficacy assessments and lot release and potency assays (*5*). Furthermore, many regulatory agencies lack the resources and capacity needed to quickly but robustly evaluate pandemic vaccines (*5*). To streamline vaccine development, assessment, production, and access, industry, governments, and regulators should enhance collaboration on new technologies, such as mRNA-based vaccines and vaccines using novel antigens; align regulatory pathways and requirements; and modernize immunogenicity assessment and lot release tools. To ensure equitable access, a global access framework should be established, including an entity that can provide financing and advanced vaccine purchases for low- and middle-income countries.

Immunization programs are complex and demand advance planning. Success requires defined roles, responsibilities, and financing as well as effective information and supply chain management. Strategies should build on experiences from seasonal influenza, COVID-19, and other outbreaks; use existing infrastructure; and engage those who will implement the programs. Immunization and communications planning must be integrated and engage affected communities, and planning must transcend political divisions. Global, federal, state, and local authorities need to clearly understand their responsibilities and the circumstances under which plans activate. Consideration of societal and economic risks from both a pandemic and potential mitigations should be integrated into decision-making. Proactive analysis is also required to prepare for impacts on supply chains for vaccines and source materials as well as effects on health care and other sectors.

To effectively address these gaps, pandemic preparedness initiatives should be urgently resourced and implemented (*6*). First, an effort to develop rapidly scalable pandemic influenza vaccines [building on models such as Operation Warp Speed (*7*)] should engage industry, governments, regulators, and the scientific community, with equitable access supported by a funded global framework. Such an initiative would, ideally, also include development of and access to improved therapeutics, diagnostics, personal protective equipment, and other needed medical countermeasures. Second, a comprehensive outreach and communications program, supported by behavioral science, should work to better understand and respond to concerns about vaccines and rebuild trust in public health. Finally, pandemic response plans should undergo transparent in-depth testing, during which countries should share plans and playbooks and form global collaborations that incorporate different disease scenarios and immunization strategies. Similar initiatives should address ongoing agricultural outbreaks (*8*). Enhancing readiness now can save lives and reduce societal and economic disruption if H5N1 or another outbreak becomes a pandemic.

Jesse L. Goodman[1]*, Norman W. Baylor[2], Rebecca Katz[3], Lawrence O. Gostin[4], Rick A. Bright[5], Nicole Lurie[6], Bruce G. Gellin[7]

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*Corresponding author.
Email: jesse.goodman@georgetown.edu
Opinions are the authors' and do not necessarily reflect the views of their institutions.

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3. Center for Infectious Disease Research and Policy, "Influenza vaccines R&D roadmap" (Regents of the University of Minnesota, 2023); https://ivr.cidrap.umn.edu/sites/default/files/IVR_Feb_2023.pdf.
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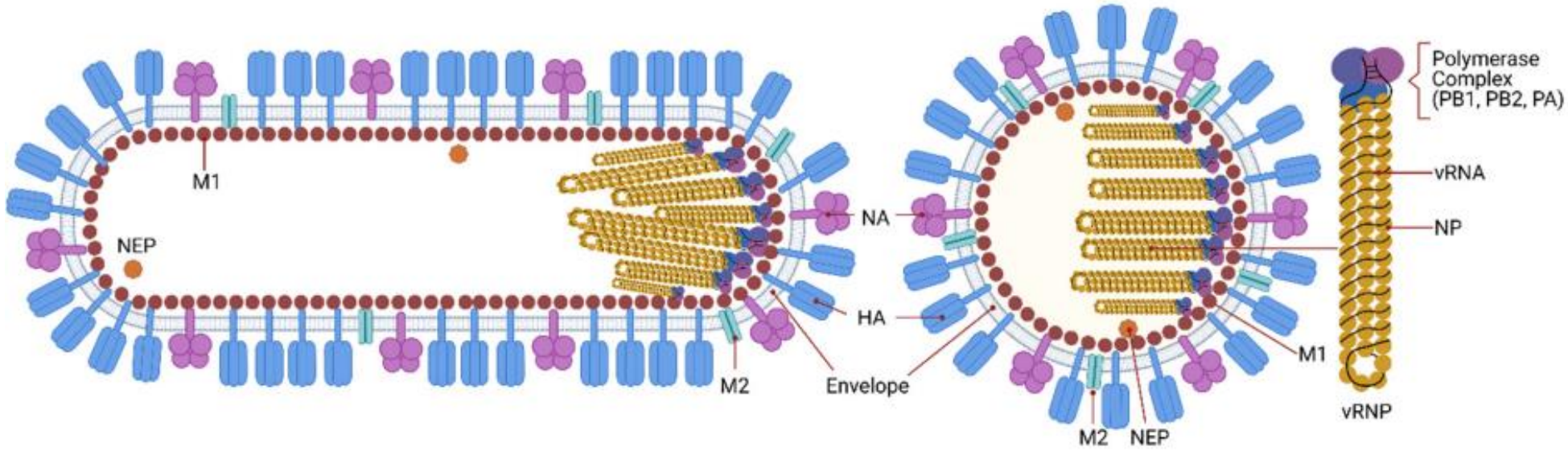
8. National Academies of Sciences, Engineering, and Medicine, *Potential Research Priorities to Inform U.S. Readiness and Response to Avian Influenza A (H5N1): Proceedings of a Workshop—In Brief*, E. P. Carlin, S. Singaravelu, L. Brown, Eds. (National Academies Press, 2024).

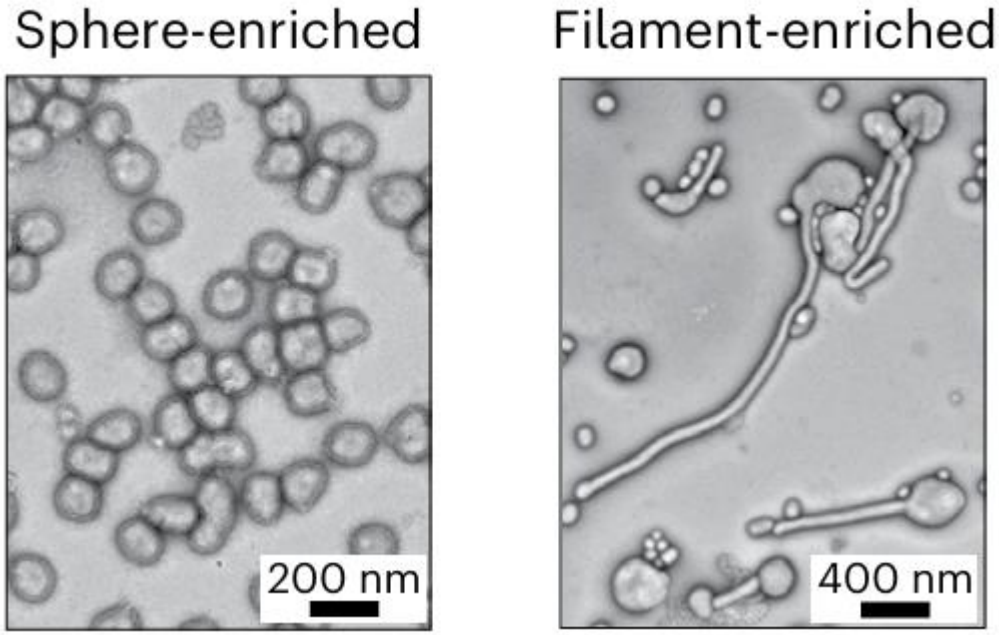
COMPETING INTERESTS

J.L.G. serves on the Board and Science Committee of GSK and as a volunteer member of the board of the nonprofit United States Pharmacopeia. R.A.B. receives nonfinancial support from the Coalition for Epidemic Preparedness Innovations (CEPI) for serving on its scientific advisory committee, receives fees from CEPI and Cidara, and has patents for virus-like particle vaccine design issued to Novavax with no financial gain.

10.1126/science.adw3278

The causative agent of influenza





nature microbiology

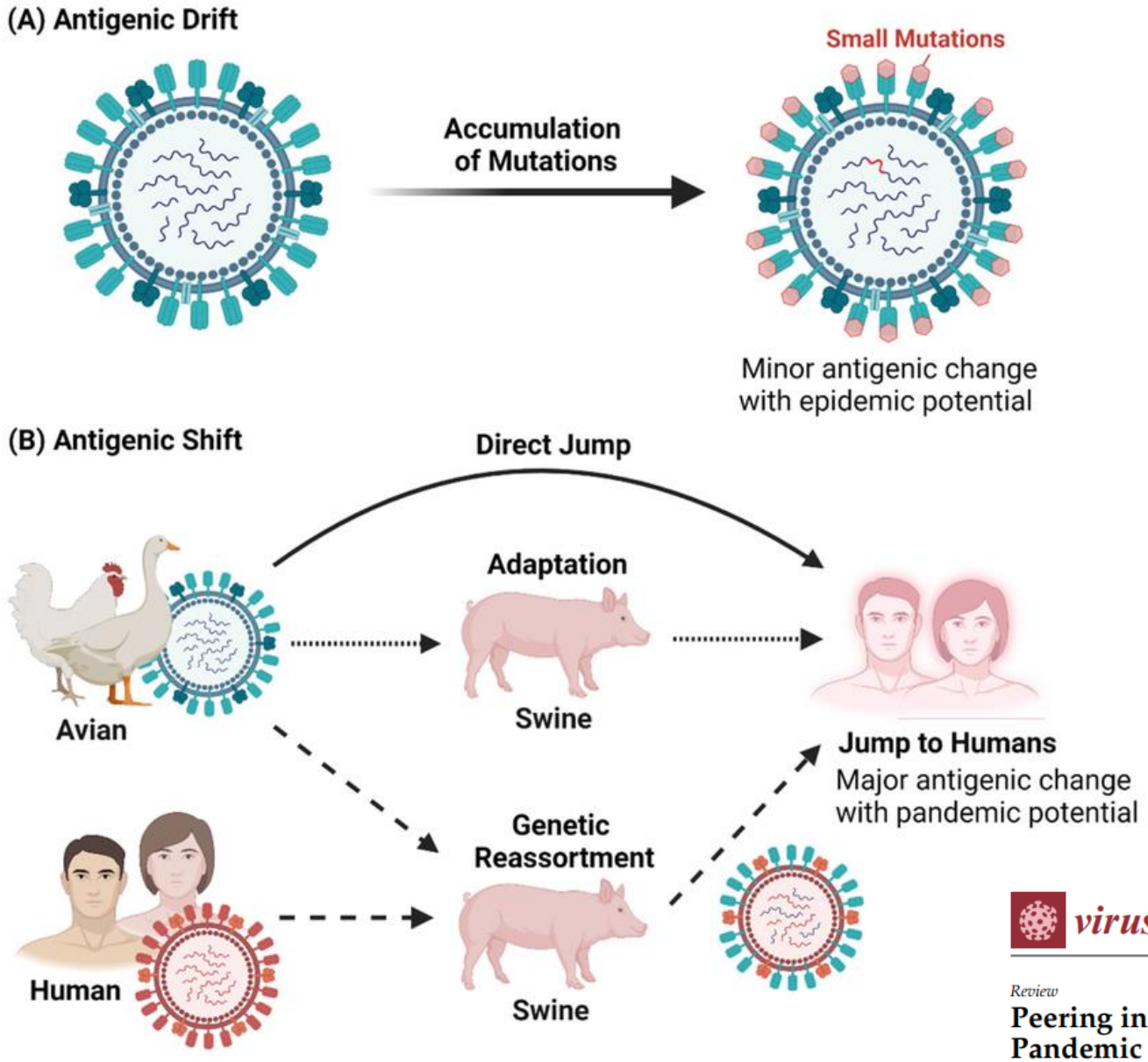
Article

https://doi.org/10.1038/s41564-025-01925-9

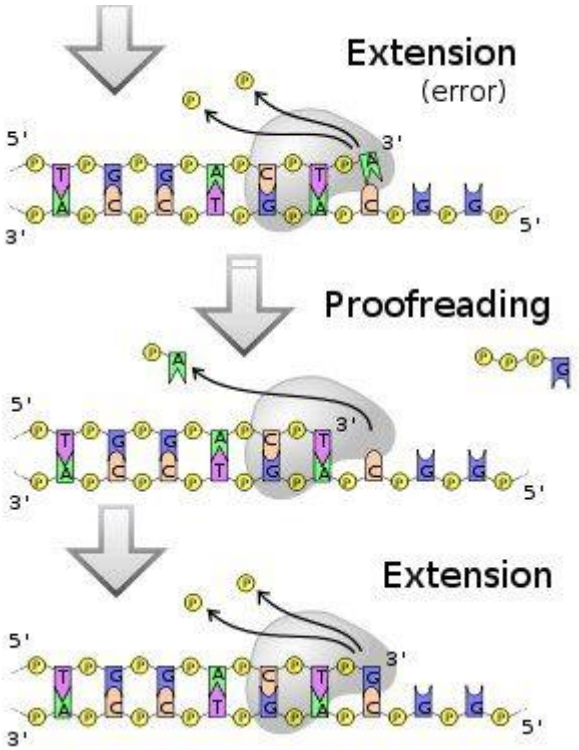
Influenza A virus rapidly adapts particle shape to environmental pressures

In summary, pleomorphy in IAV is a conserved, tunable feature that provides an evolutionary advantage under immune and entry challenges. This plasticity enables IAV to rapidly respond to immune pressures or new hosts, facilitating both persistence of adapted strains and new adaptation after spillover.

Antigenic Drift vs. Antigenic Shift: Mechanisms of Change in Influenza

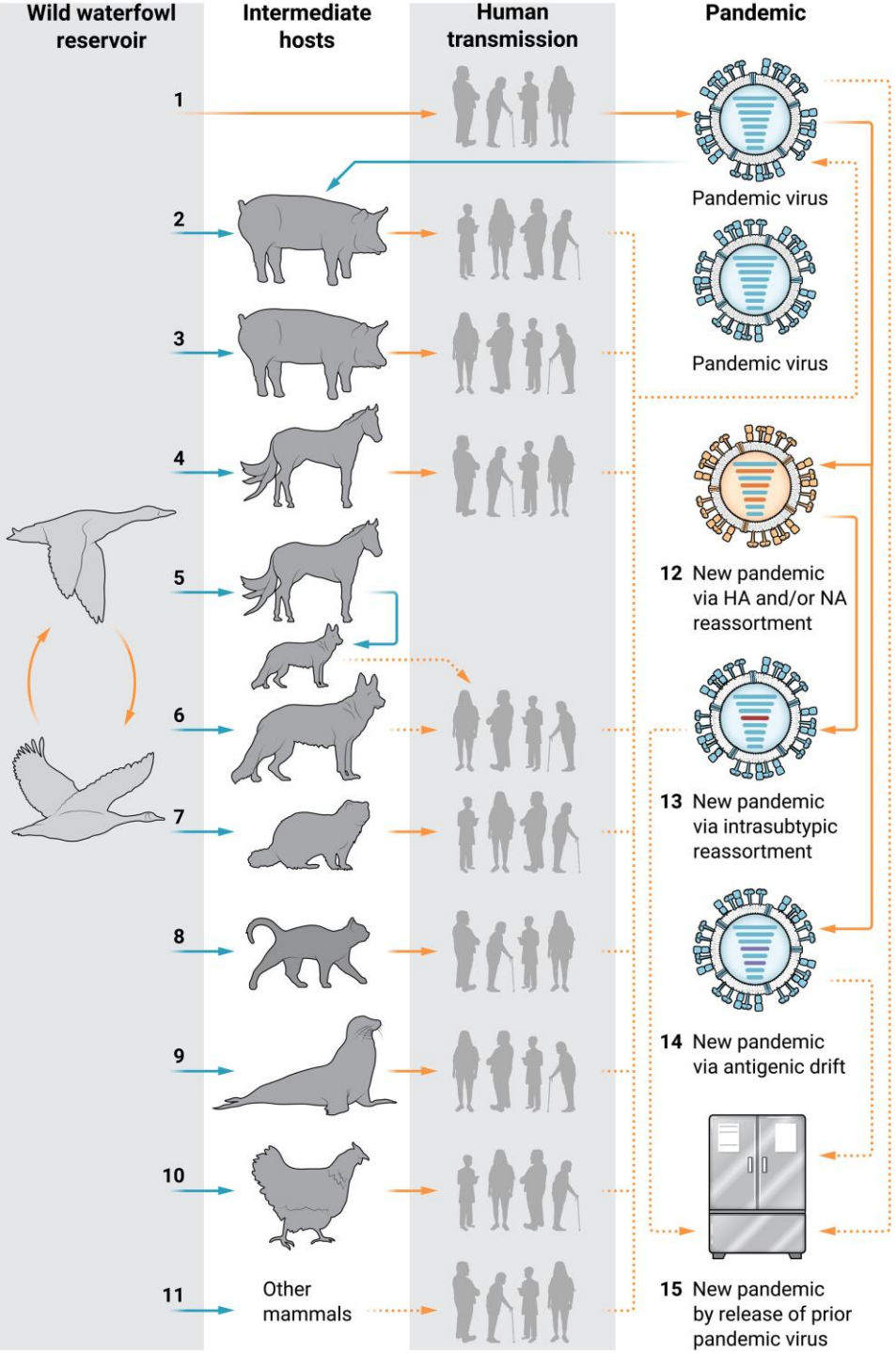


Flu has no proofreading



viruses
MDPI
Review

Peering into Avian Influenza A(H5N8) for a Framework towards Pandemic Preparedness



SCIENCE TRANSLATIONAL MEDICINE | VIEWPOINT

ZOONOTIC DISEASES

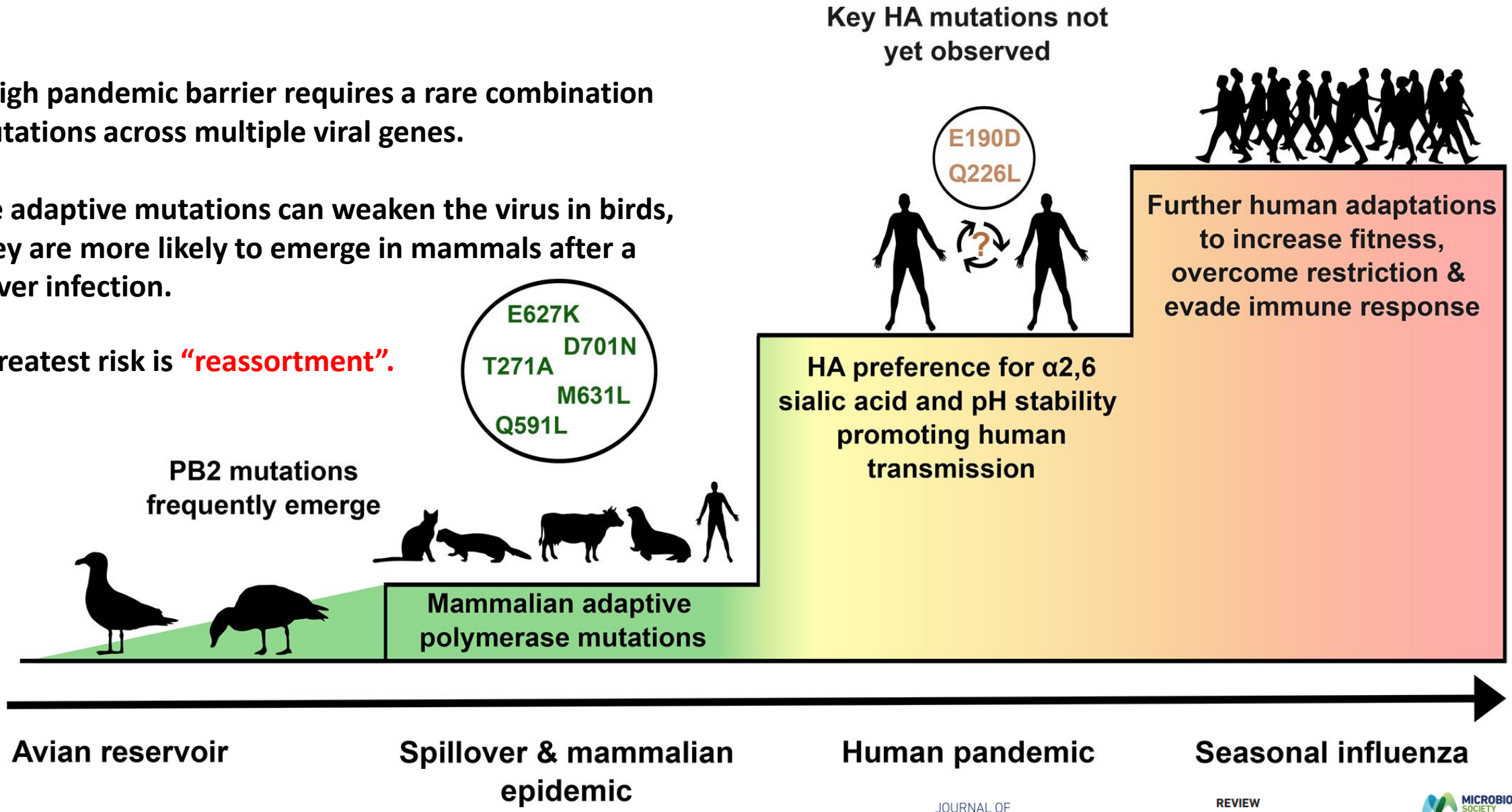
Many potential pathways to future pandemic influenza

Pandemic prediction is extremely difficult

- The 1918 pandemic occurred when a waterfowl IAV adapted directly to humans (pathway 1)
- The 2009 pandemic resulted from reassortment between avian, swine, and human IAVs (pathway 2)
- The 1957 and 1968 pandemics occurred when human IAVs reassorted with avian IAVs (pathway 12)
- Seasonal human IAV evolution through reassortment (pathway 13) or antigenic drift (pathway 14)
- Potential release of prior pandemic viruses from laboratories (pathway 15)

Why aren’t pandemics more common?

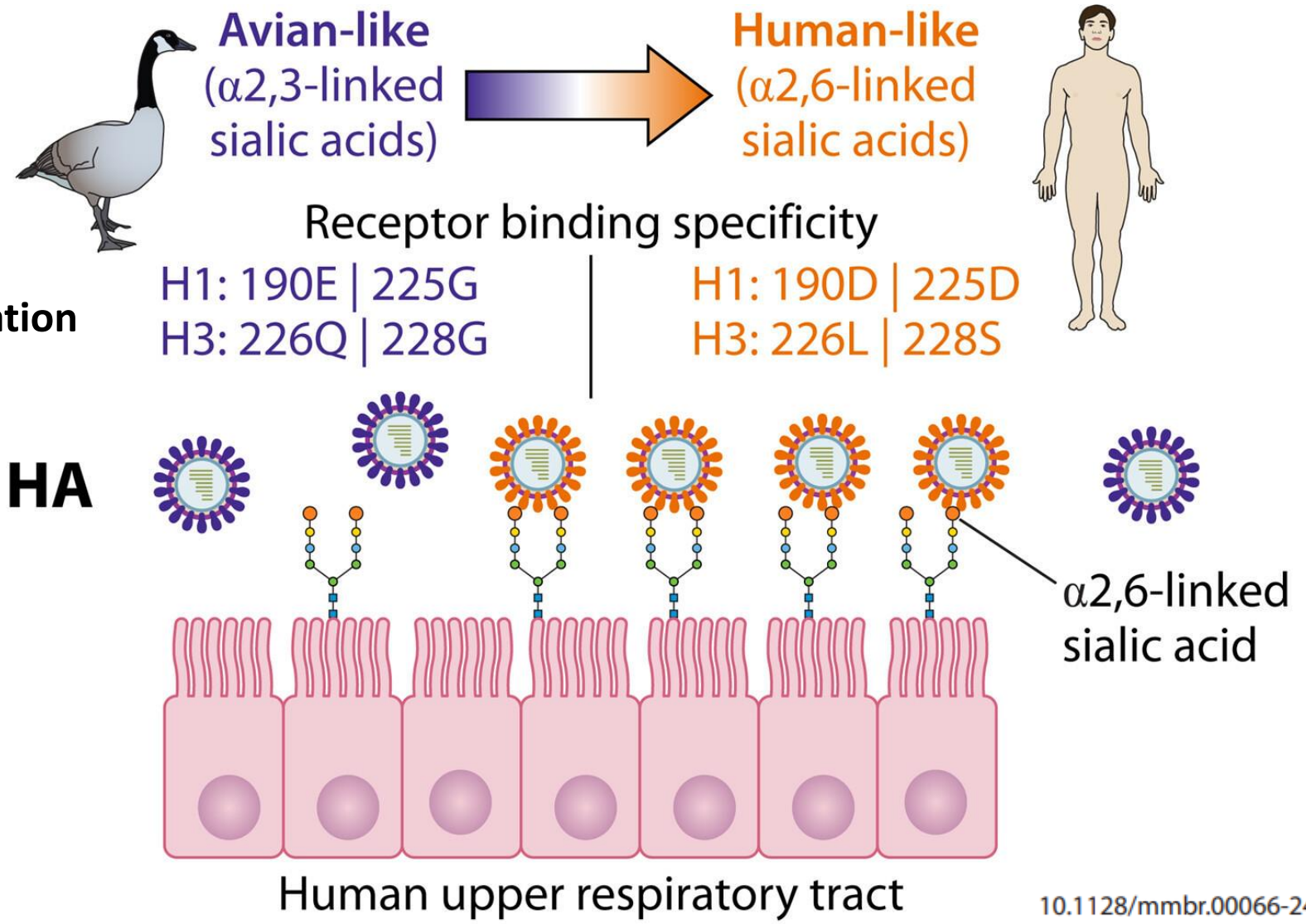
- The high pandemic barrier requires a rare combination of mutations across multiple viral genes.
- These adaptive mutations can weaken the virus in birds, so they are more likely to emerge in mammals after a spillover infection.
- The greatest risk is “reassortment”.



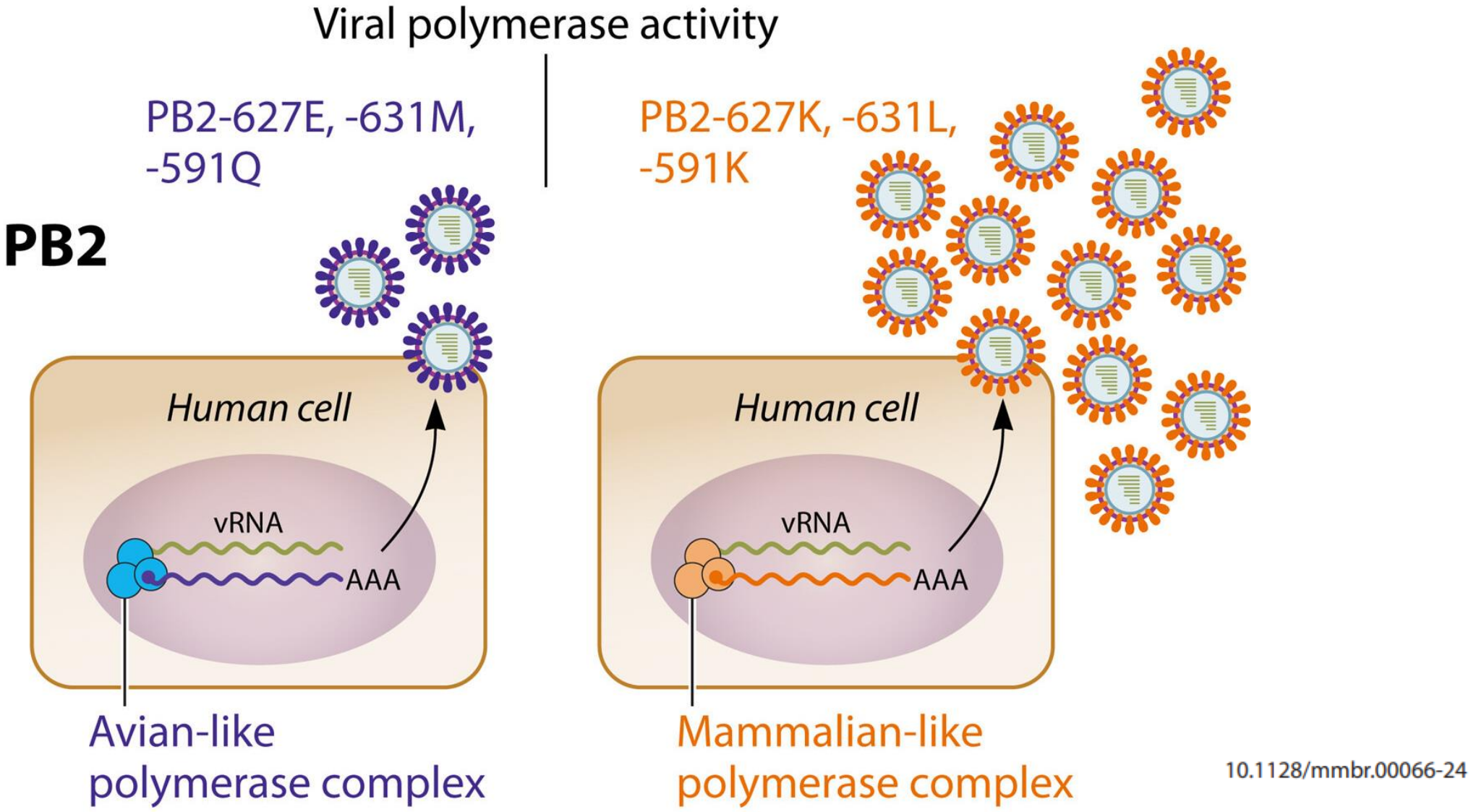
JOURNAL OF GENERAL VIROLOGY
REVIEW
Capelastegui and Goldhill, *Journal of General Virology* 2025;106:002109
DOI 10.1099/jgv.0.002109
MICROBIOLOGY SOCIETY
OPEN ACCESS

HA mutations affecting receptor binding and stability drive avian flu's adaptation to humans

Key Mutations for Human Adaptation

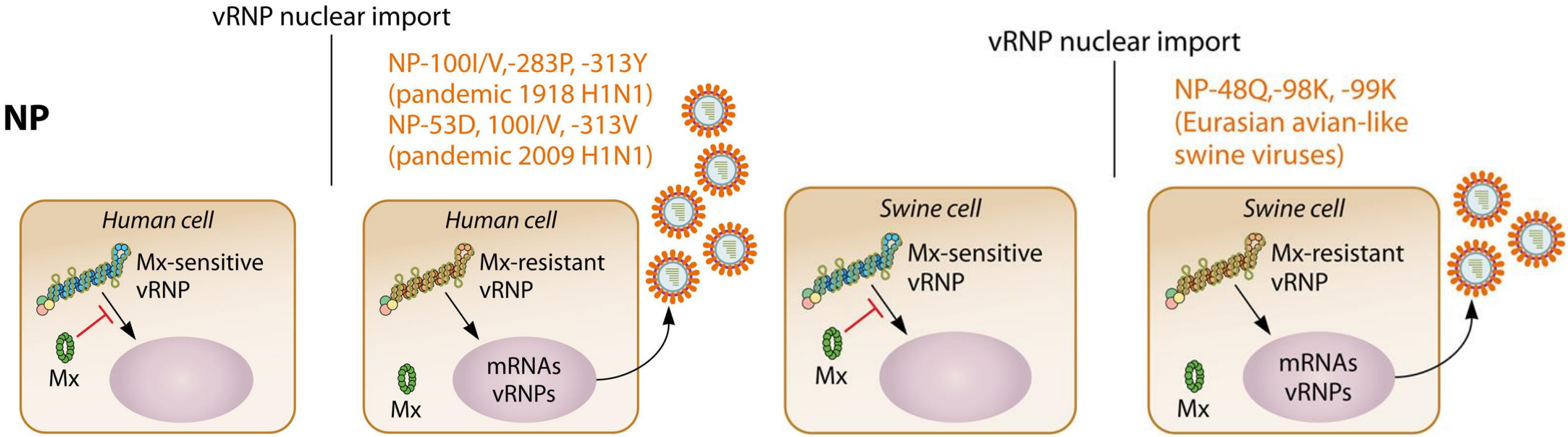


PB2 mutations boost mammalian replication and enable airborne spread



10.1128/mmbr.00066-24

NP mutations help the virus adapt to mammals by overcoming host-specific defenses



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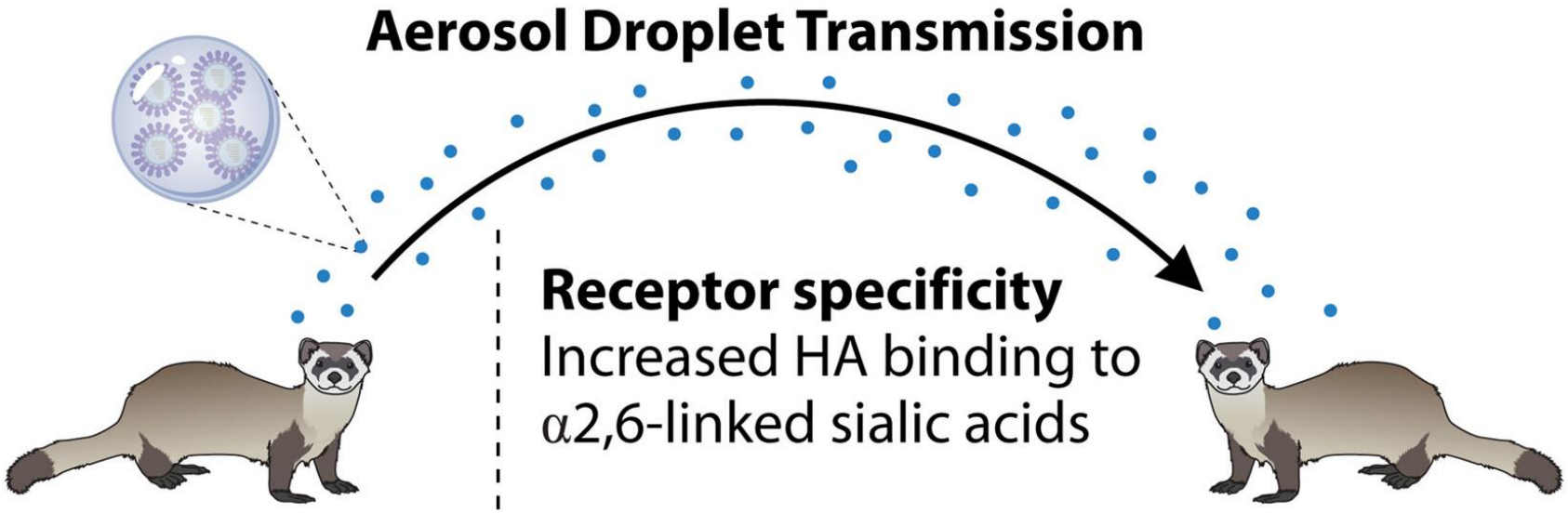
Common themes among mammalian-transmissible avian viruses



Receptor specificity
Increased HA binding to α2,6-linked sialic acids

HA glycosylation
Loss of glycosylation at HA-158-160

HA stability
Restoration of HA stability after adaptation to α2,6-linked sialic acids

Replicative ability
Increased replication in mammalian cells

10.1128/mmbr.00066-24

History of H5N1

•1996-1997: The Beginning

- A dangerous form of A(H5N1) virus first appeared in domestic birds in South China.

•2003-2005: The Second Wave

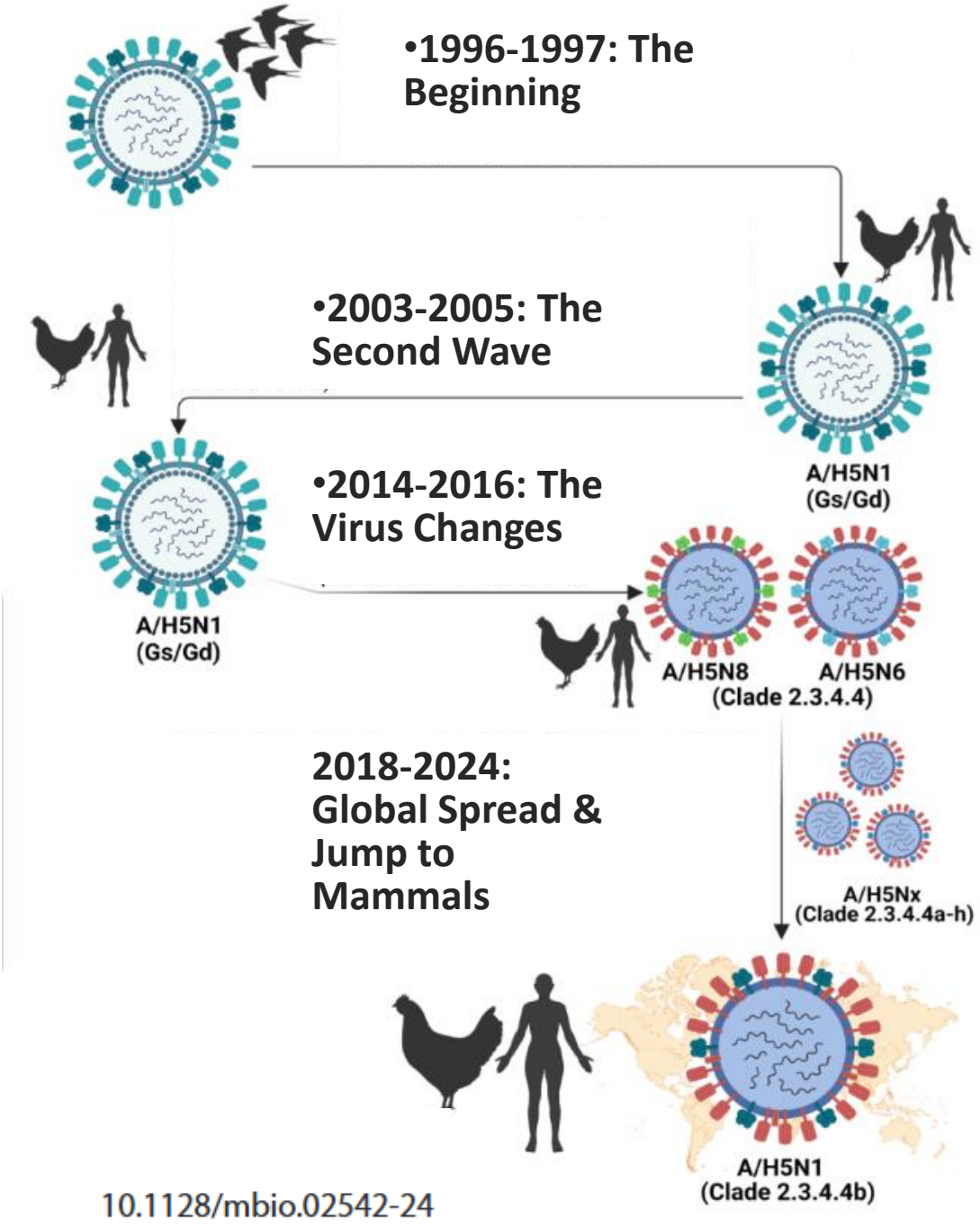
- The virus re-emerged in Asia and was carried by migratory birds to Europe and Africa

•2014-2016: The Virus Changes

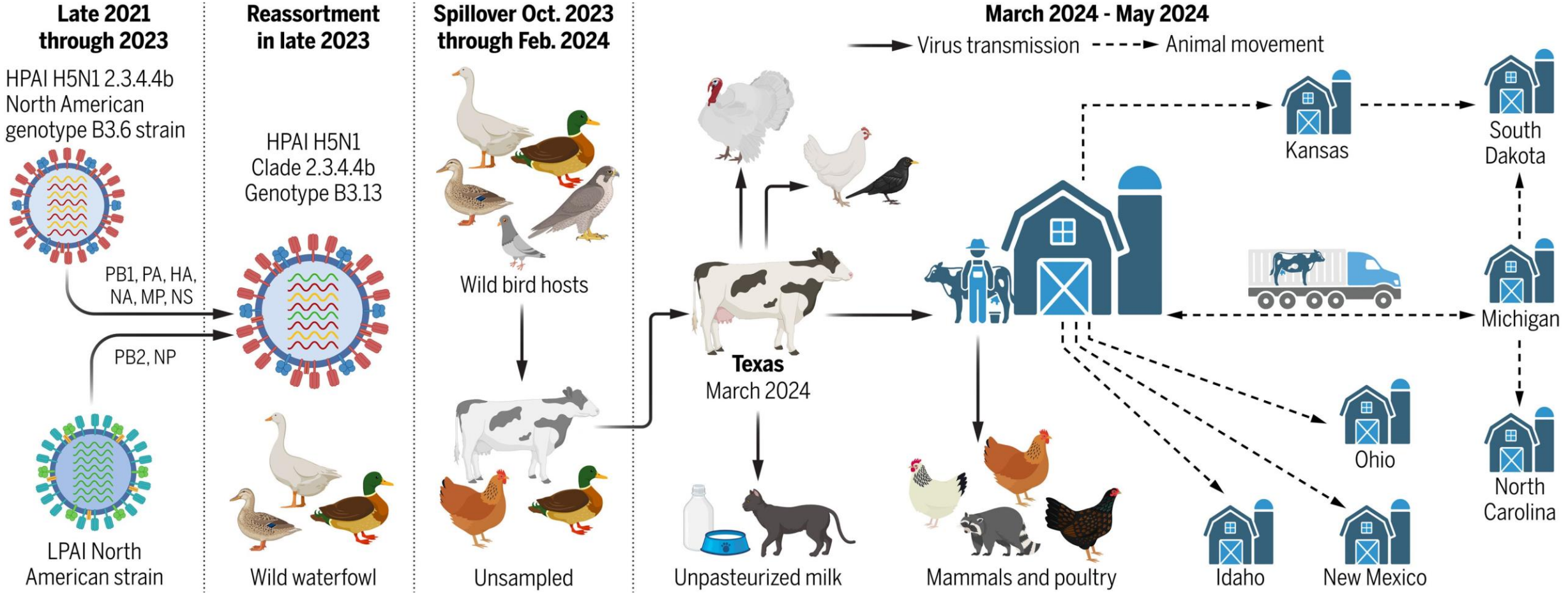
- The virus continued to evolve, mixing with other flu viruses to create new reassortant strains, notably A/H5N6 and A/H5N8, which belonged to a new genetic group **(Clade 2.3.4.4)**.

•2018-2024: Global Spread & Jump to Mammals

- A novel and highly successful version of the A/H5N1 virus (Clade 2.3.4.4b) emerged and spread across Asia, Africa, Europe, and North America.
- Most significantly, this new version has not only infected birds and humans but has also been detected in **several mammalian species**, marking a critical expansion of its host range.



Spillover and spread of HPAI H5N1 genotype B3.13 into dairy cattle



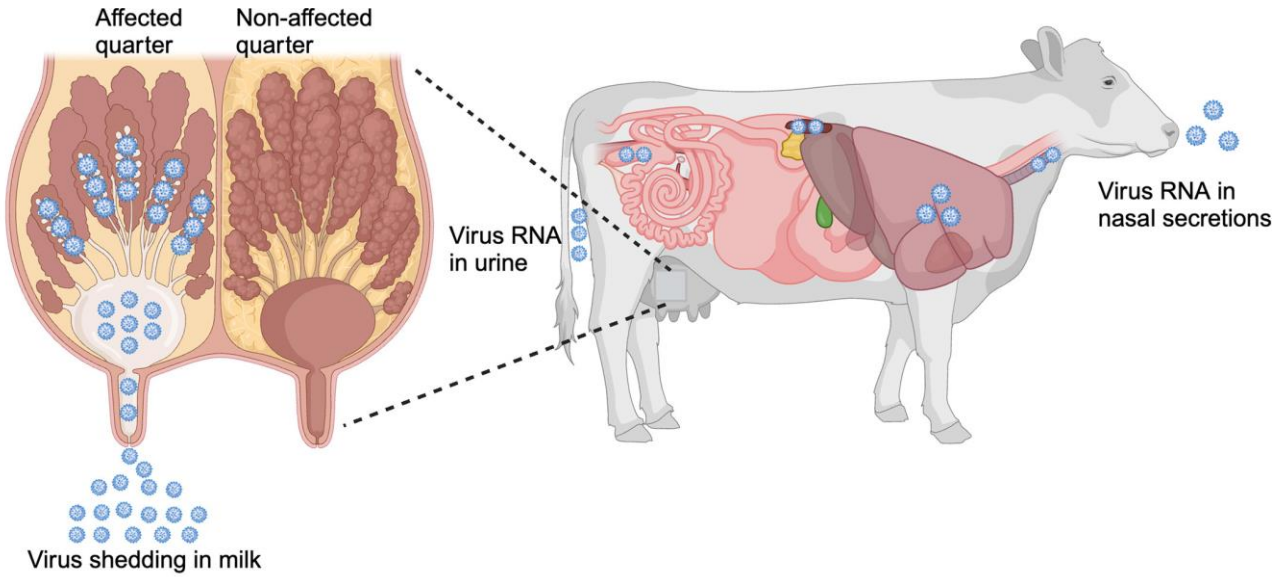
RESEARCH

RESEARCH ARTICLE SUMMARY

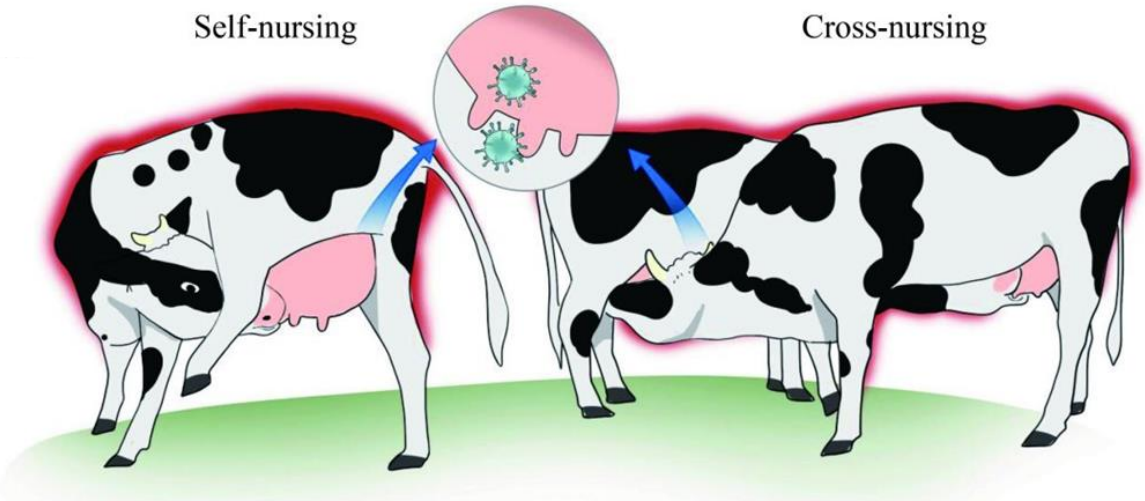
AVIAN INFLUENZA

Emergence and interstate spread of highly pathogenic avian influenza A(H5N1) in dairy cattle in the United States

Solving the Puzzle: Oral-to-Mammary Transmission of H5N1 in Cattle



The high level of sialic acid receptors in oral tissues allows influenza virus infect cattle through contaminated feed or water



Virus replicating in the mouth of cattle could be transmitted to its own mammary glands through self-nursing or to the mammary glands of others through cross-nursing

H5N1 virus adapting and evolving as it spread from cow to cow

Gene	Coding-region change	Functional type	Cattle with variant (no.)	Mean allele frequency	Consensus sequence	Low-frequency variants
HA	E91K	Mammal adapt.	1 (1)	0.05	0	1
HA	S137F	Mammal adapt.	1 (1)	0.376 ★	0	1
HA	Q154R	Pathogenicity	5 (9)	0.013	0	6
HA	N209T	Mammal adapt.	1 (3)	0.012	0	1
HA	Q234K/R	Virulence	8 (32)	0.039	0	9
HA	G240R	Mammal adapt.	1 (1)	0.025	0	1
HA	S336N	Virulence	18 (245)	0.892 ★	15	2
HA	P337L	Virulence	8 (21)	0.715 ★	5	2
MP	R77K	Virulence	1 (7)	0.006	0	1
NA	T438A/I	Antiviral resist.	3 (9)	0.627 ★	2	1
NA	R430K	Mammal adapt.	1 (82)	0.130	0	1
NS	D125N/G	Virulence	27 (20)	0.873 ★	24	3
NS	E229K	Virulence	21 (85)	0.999 ★	18	0
PB2	R389K/G	Mammal adapt.	2 (5)	0.012	0	2
PB2	E627K	Virulence/adapt.	1 (12)	0.329 ★	0	1
PB2	D701N	Virulence	2 (3)	0.015	0	2

RESEARCH

RESEARCH ARTICLE SUMMARY

AVIAN INFLUENZA

Emergence and interstate spread of highly pathogenic avian influenza A(H5N1) in dairy cattle in the United States

Cell

CellPress

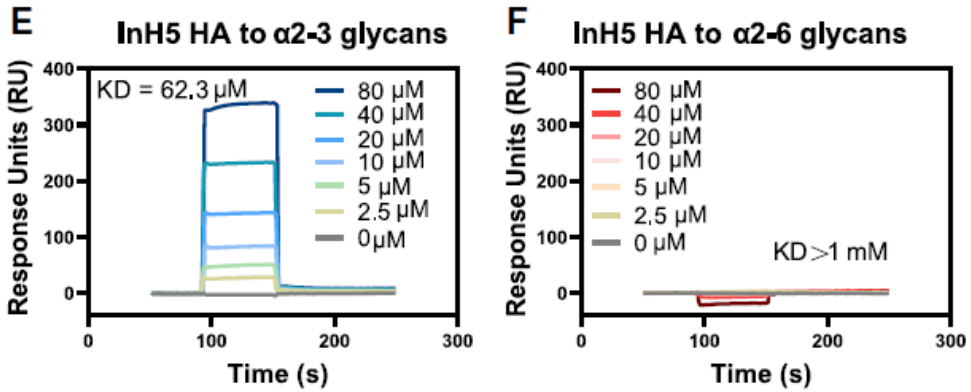
Article

Receptor binding, structure, and tissue tropism of cattle-infecting H5N1 avian influenza virus hemagglutinin

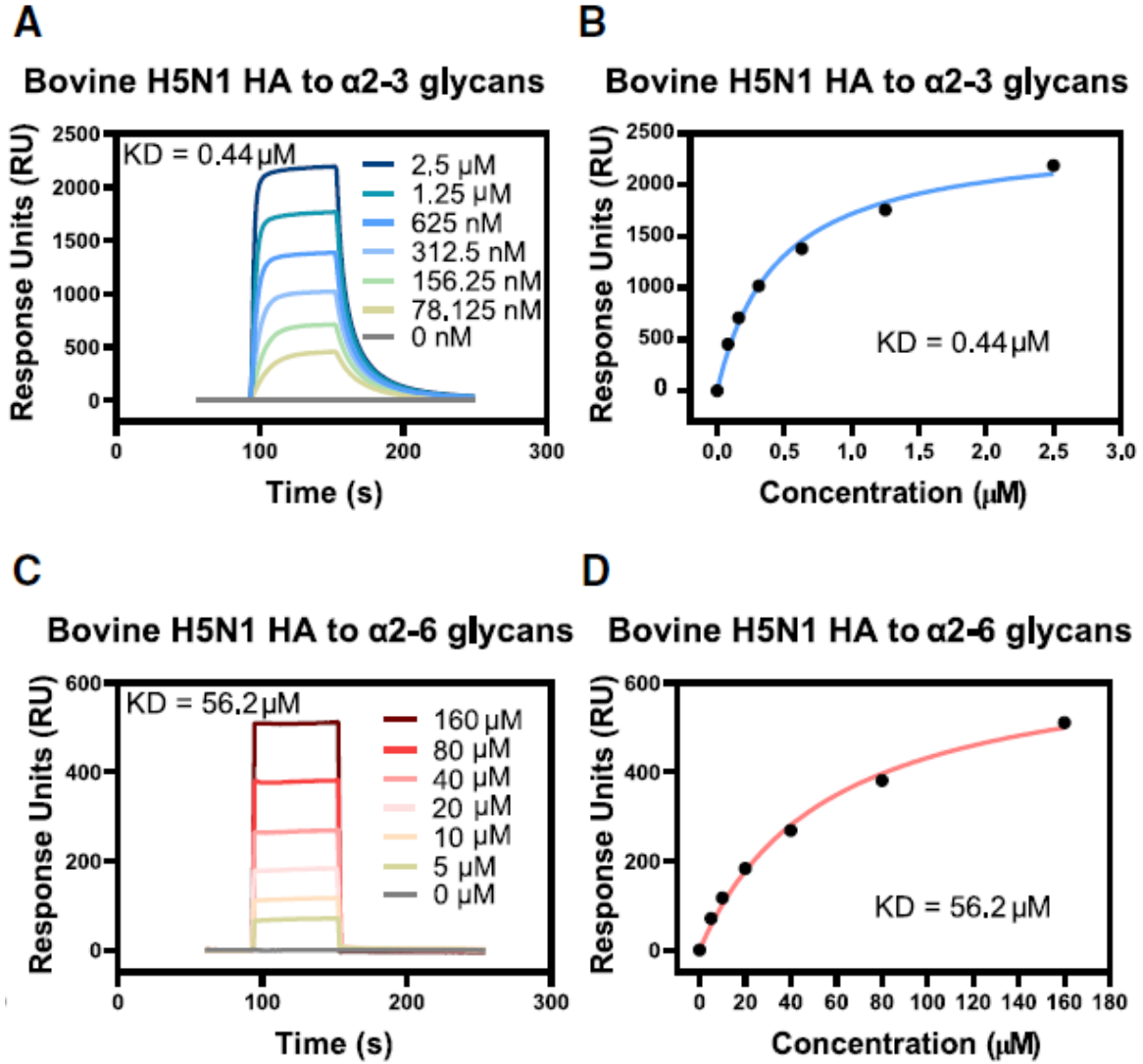
Cell *188*, 919–929, February 20, 2025

Receptor Binding Properties

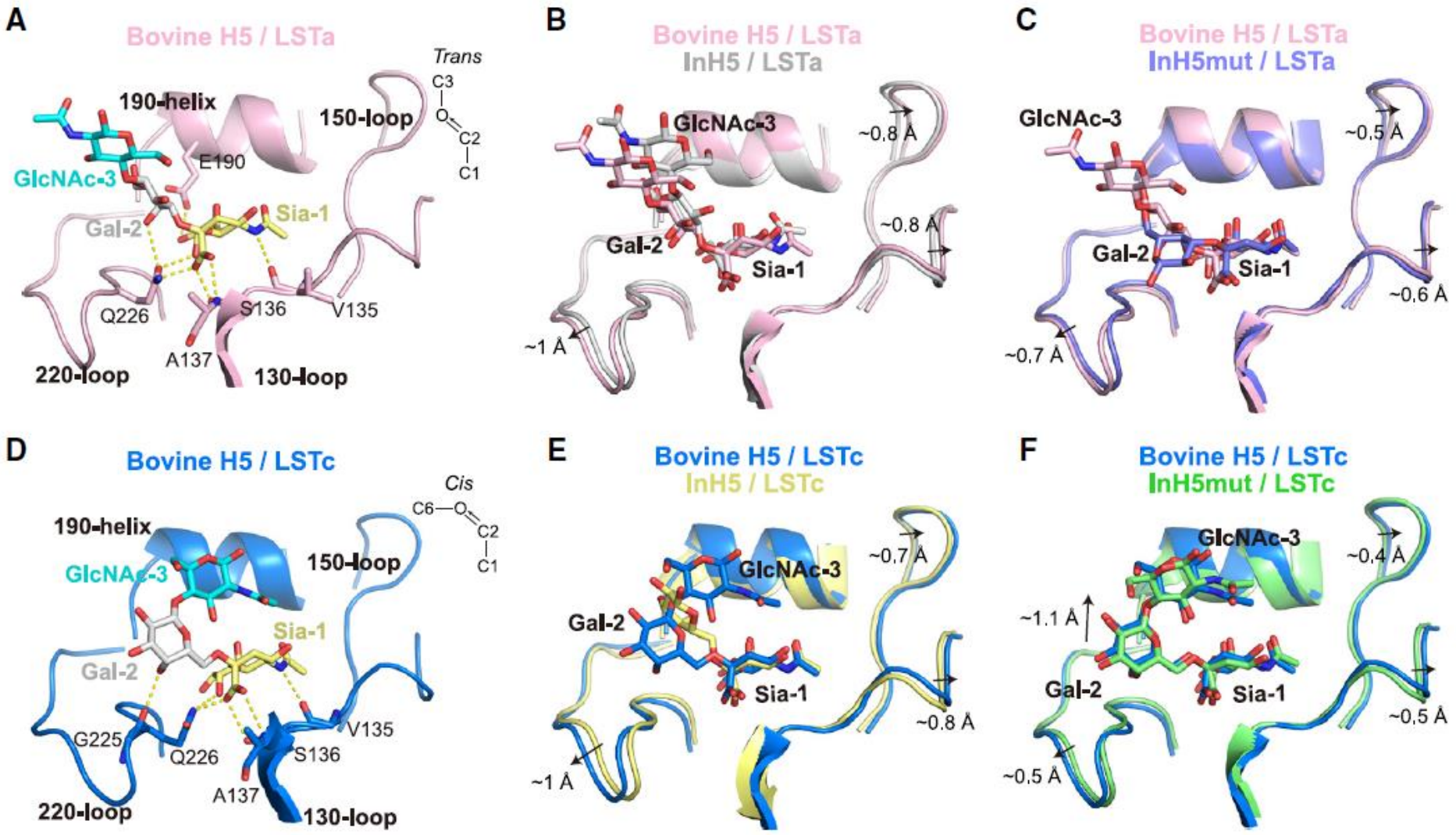
- Preference for Avian Receptors with Slight Human Receptor Affinity



Previous H5N1 strain (A/Indonesia/5/2005) showed binding only to avian receptors with no detectable binding to human receptors



Structural Adaptations in Receptor Binding Site



- Cryo-EM structures revealed an enlarged receptor binding site (RBS) compared to previous H5N1 strains

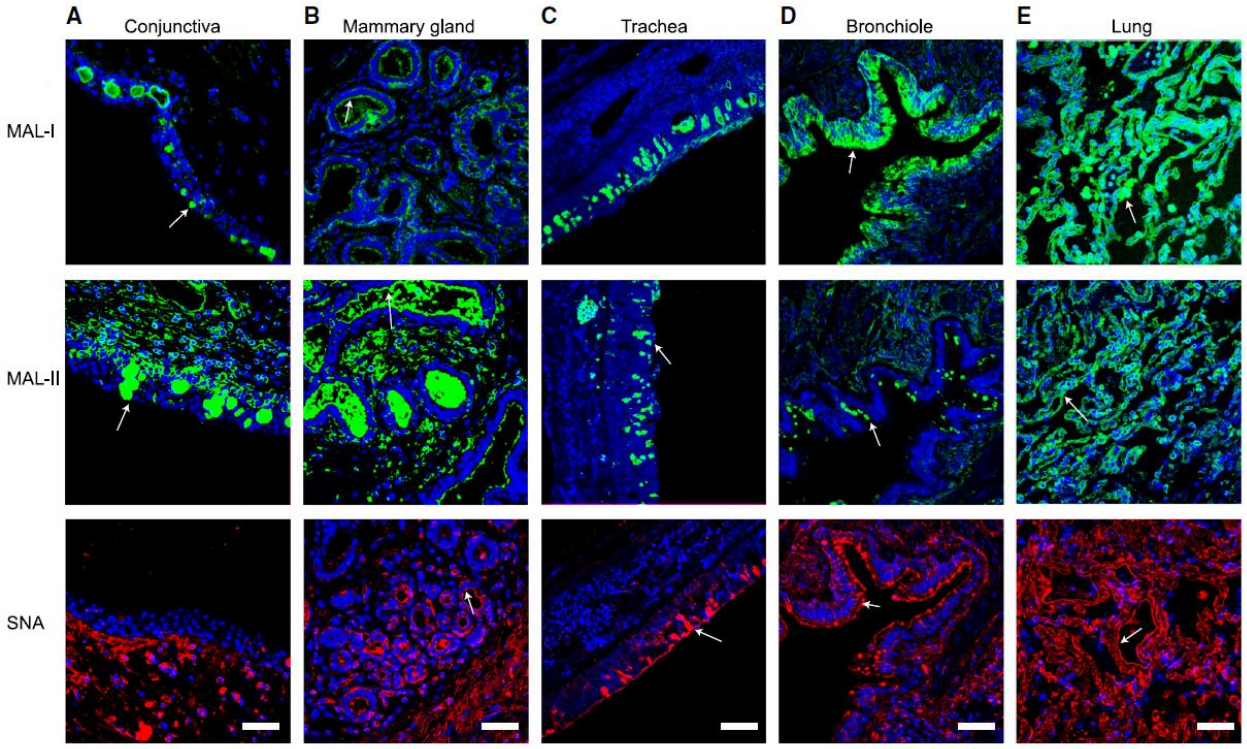
Enhance the affinity of bovine H5 for both human and avian receptors?

Human infection risk

Binding to Human Tissues

Immunohistochemical staining showed bovine H5N1 HA binds effectively to:

- Human conjunctiva (stratified columnar epithelium)
- Human mammary gland (alveolar epithelium)
- Human trachea (pseudostratified ciliated columnar epithelium)
- Human bronchioles (epithelial cells)
- Human lung (alveolar cells)



Conjunctiva, mammary gland, and lung tissues are potential sites for bovine H5N1 infection in humans

Article Wisconsin

A human isolate of bovine H5N1 is transmissible and lethal in animal models

https://doi.org/10.1038/s41586-024-08254-7
Received: 18 July 2024
Accepted: 18 October 2024
Published online: 28 October 2024

Chunyang Gu[1,6], Tadashi Maemura[1,6], Lizheng Guan[1,6], Amie J. Eisfeld[1,6], Asim Biswas[1,6], Maki Kiso[2,6], Ryuta Uraki[2], Mutsumi Ito[2], Sanja Trifkovic[1], Tong Wang[1], Lavanya Babujee[1], Robert Presler Jr[1], Randall Dahn[1], Yasuo Suzuki[3], Peter J. Halfmann[1], Seiya Yamayoshi[2], Gabriele Neumann[1] & Yoshihiro Kawaoka[1,2,4,5]

Article CDC

Transmission of a human isolate of clade 2.3.4.4b A(H5N1) virus in ferrets

https://doi.org/10.1038/s41586-024-08246-7
Received: 10 July 2024
Accepted: 17 October 2024
Published online: 28 October 2024

Joanna A. Pulit-Penaloza[1], Jessica A. Belser[1], Nicole Brock[1], Troy J. Kieran[1], Xiangjie Sun[1], Claudia Pappas[1], Hui Zeng[1], Paul Carney[1], Jessie Chang[1], Brandon Bradley-Ferrell[1], James Stevens[1], Juan A. De La Cruz[1], Yasuko Hatta[1], Han Di[1], C. Todd Davis[1], Terrence M. Tumpey[1] & Taronna R. Maines[1]

Key Points of Agreement

Severe Pathogenicity in Animal Models

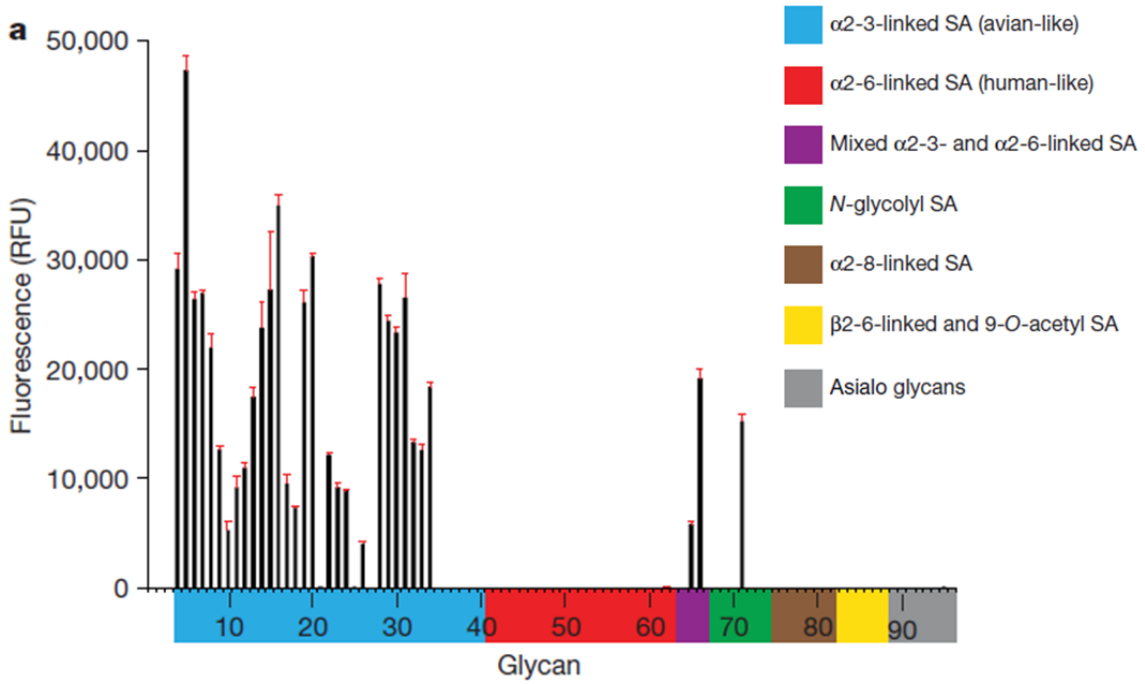
- TX/37 is highly lethal in ferrets, with 100% mortality
- Systemic spread to multiple organs beyond the respiratory tract
- Rapid disease progression in ferrets, with death occurring within 2-5 days

Respiratory Droplet Transmission

- TX/37 can transmit between ferrets via respiratory droplets
- Similar transmission rates (CDC study: 4/6 or 67% of pairs; Wisconsin study: 17-33% of pairs)
- Ferrets infected through respiratory transmission developed fatal disease

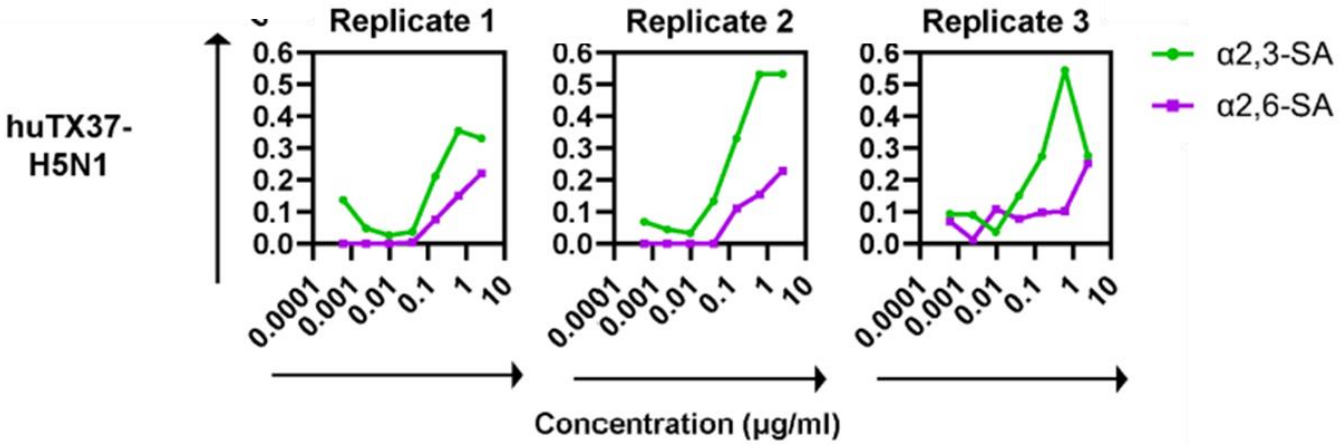
Systemic Infection

- Virus spread to multiple organs including brain, liver, and blood
- High viral loads in respiratory tissues and evidence of viremia



CDC

- TX/37 maintains strict avian-like (α2,3-linked sialic acid) receptor binding with no detectable binding to human-type (α2,6-linked) receptors
- Glycan microarray analysis



Wisconsin

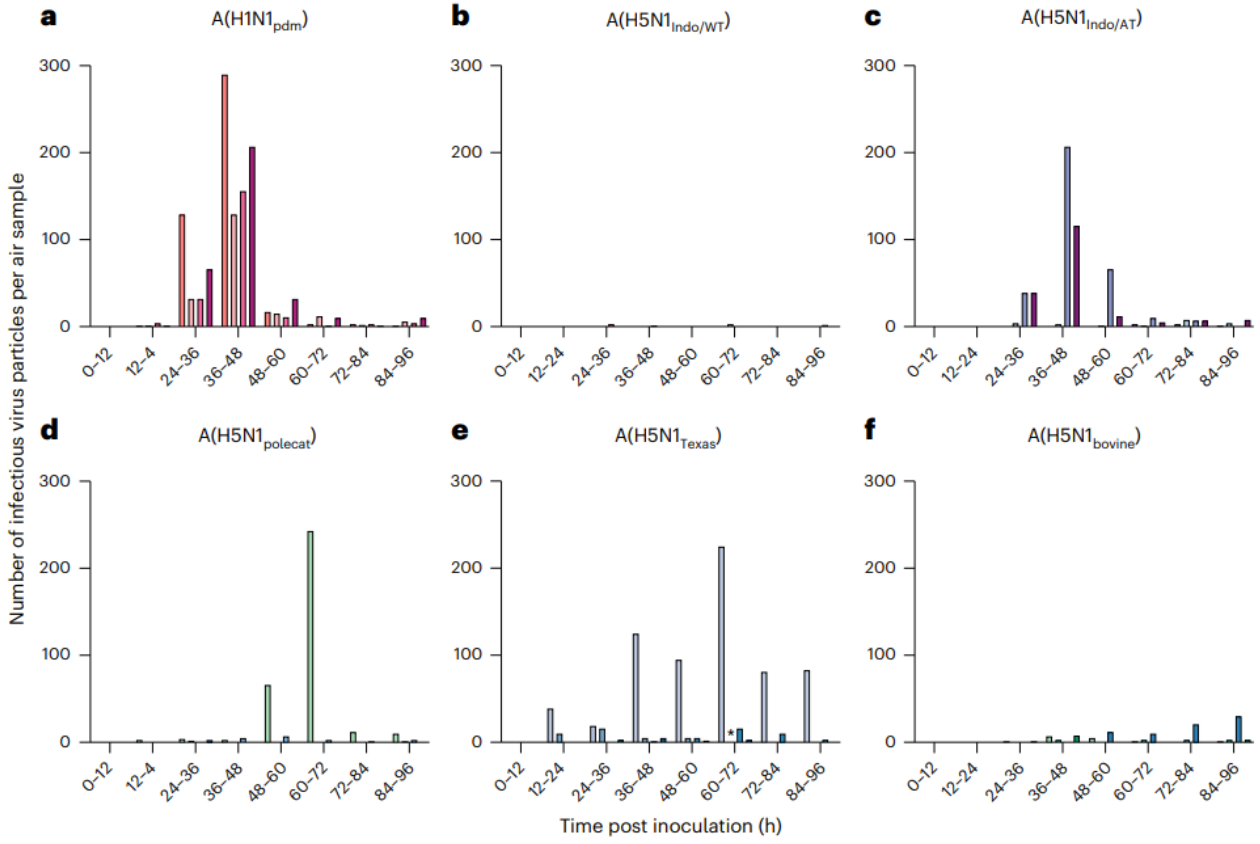
- TX/37 shows preferential binding to α2,3-linked sialic acids but also binds to α2,6-linked sialic acids
- Solid-phase binding assay with sialylglycopolymers

nature microbiology

Brief Communication

https://doi.org/10.1038/s41564-024-01885-6

Influenza A(H5N1) shedding in air corresponds to transmissibility in mammals



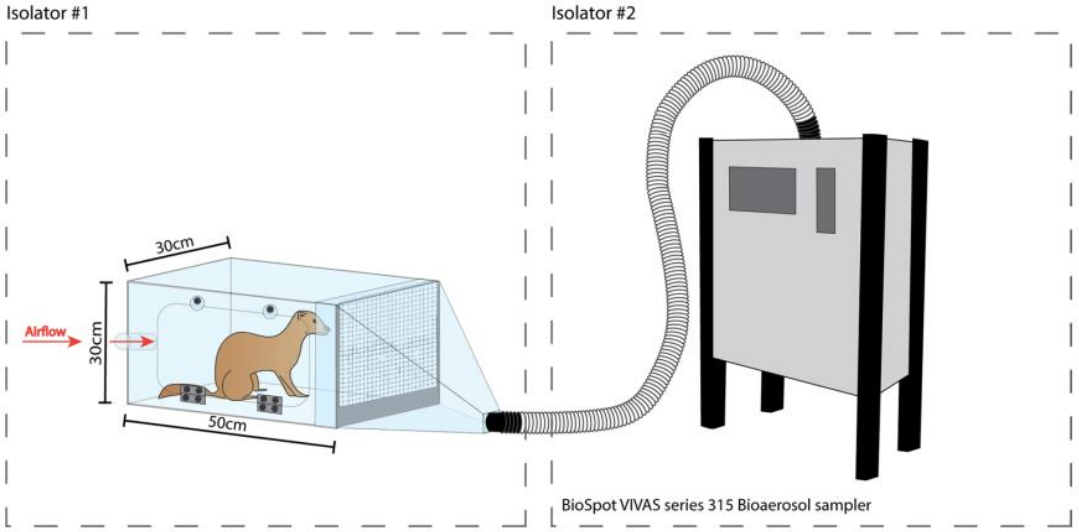


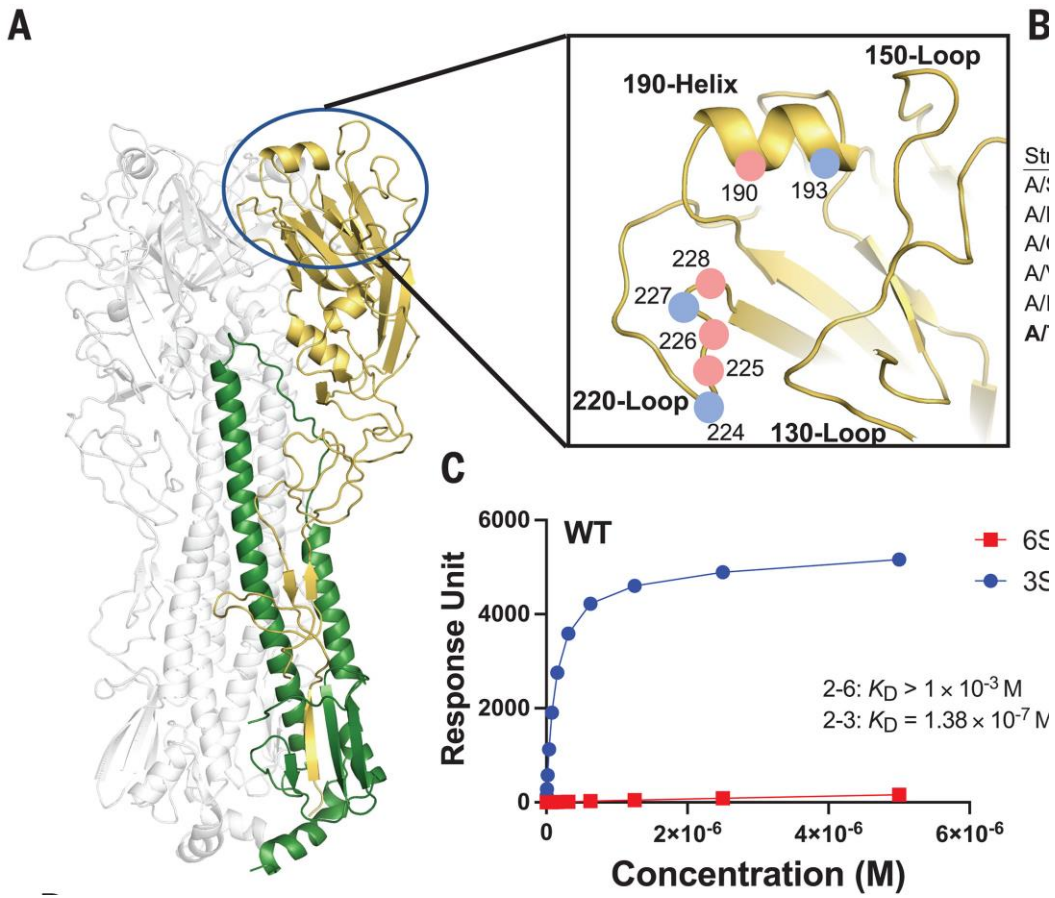
Table 1 | Transmission and virus shedding in experimentally infected animals

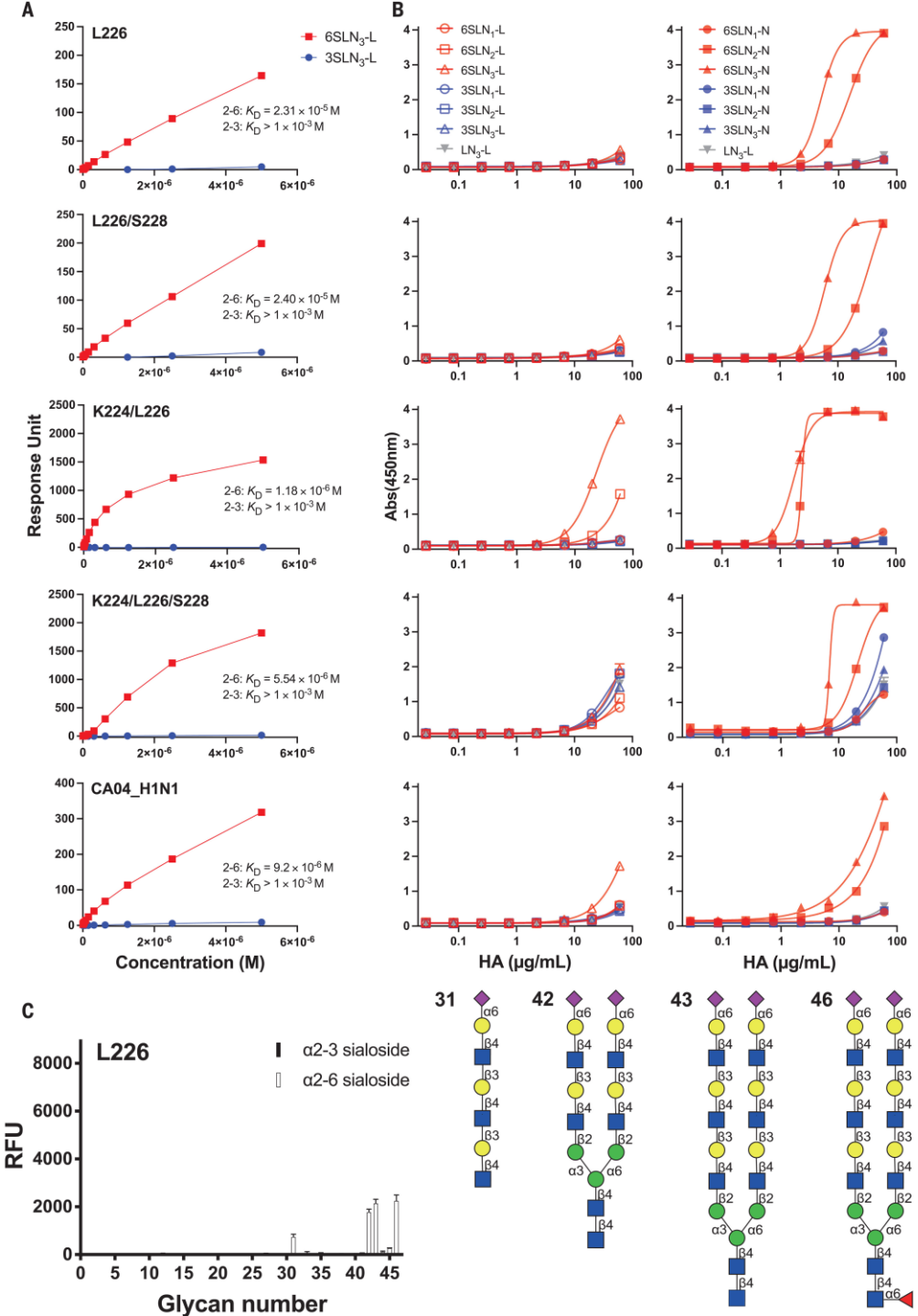
Influenza virus	Transmission efficiency between ferrets as demonstrated by virus isolation from recipient	Infectious virus collected from air (this study)
A(H1N1pdm)	4/4 (ref. 30)	4/4
A(H5N1$_{Indo/WT}$)	0/4 (ref. 7)	0/4
A(H5N1$_{Indo/AT}$)	3/4 (ref. 11)	2/4
A(H5N1$_{polecat}$)	1/4[$]	1/4
A(H5N1$_{Texas}$)	10/30 (refs. 14,18)	1/4
A(H5N1$_{bovine}$)	0/4* (ref. 19)	0/4

*Seroconversion was demonstrated in one animal.

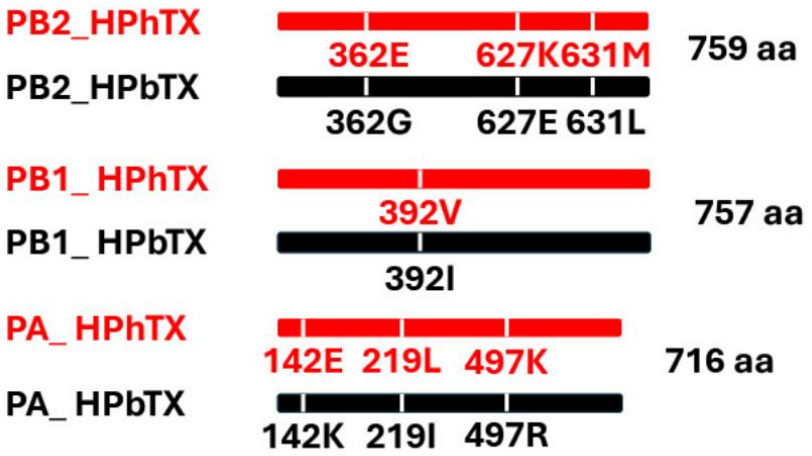
RESEARCH ARTICLE | INFLUENZA

A single mutation in bovine influenza H5N1 hemagglutinin switches specificity to human receptors





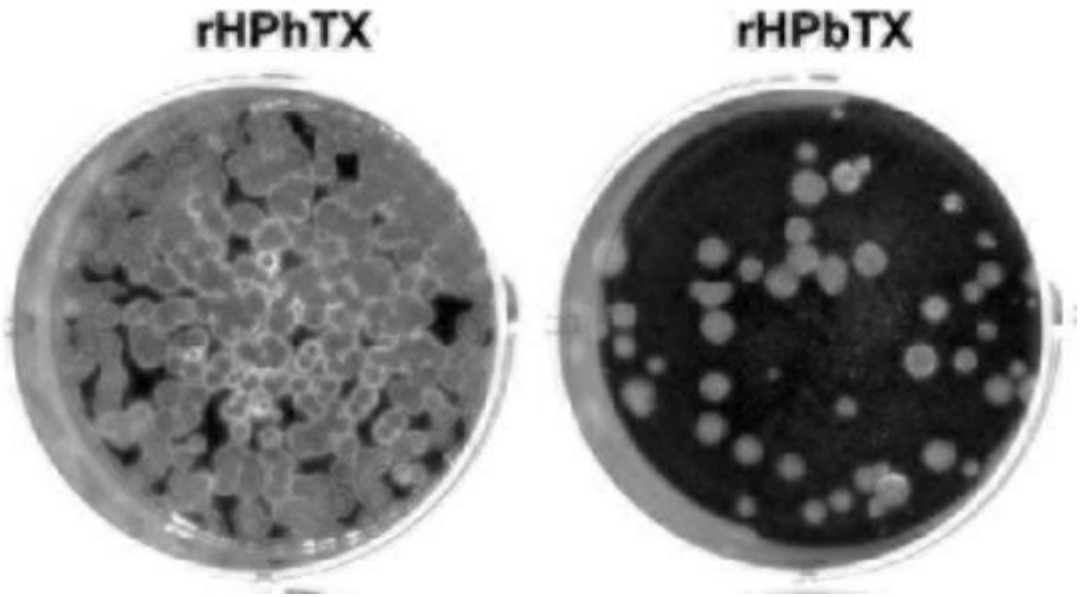
Identification of Amino Acid Residues Responsible for Differential Replication and Pathogenicity of Avian Influenza Virus H5N1 Isolated from Human and Cattle in Texas, US

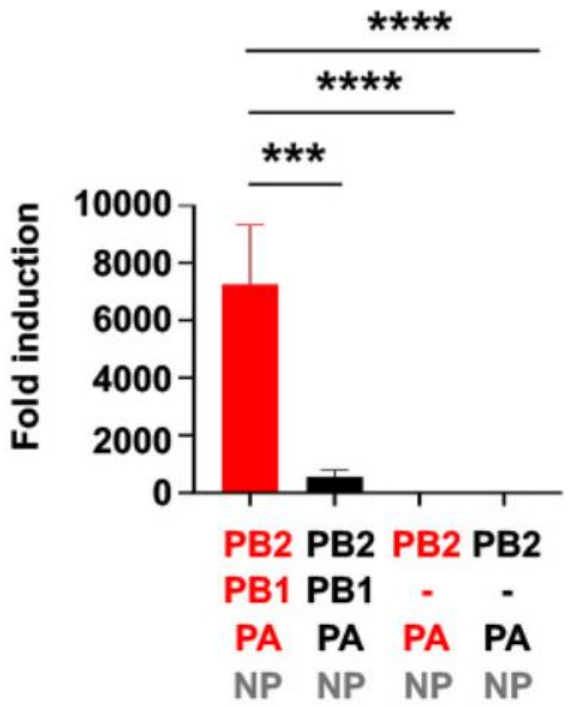


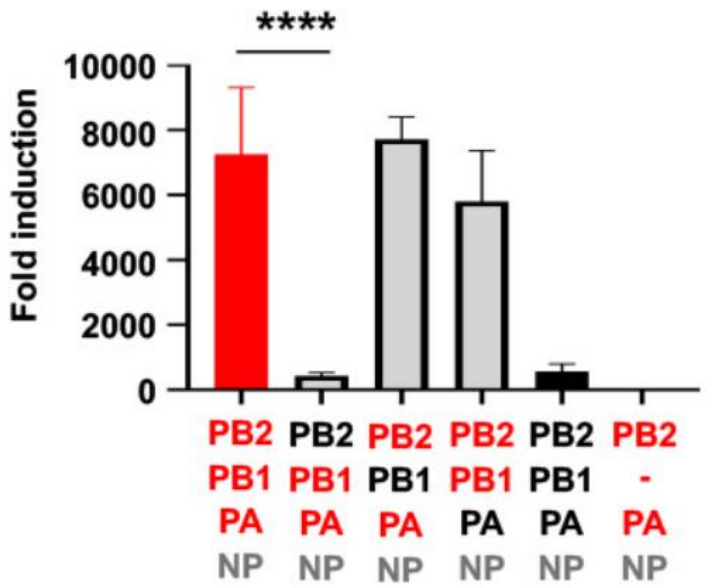
HPhTX: Influenza A/Texas/37/2024 H5N1

HPbTX: Influenza A/bovine/Texas/24-029328-02/2024 H5N1

PB2 protein is the primary determinant of this increased activity.







Health

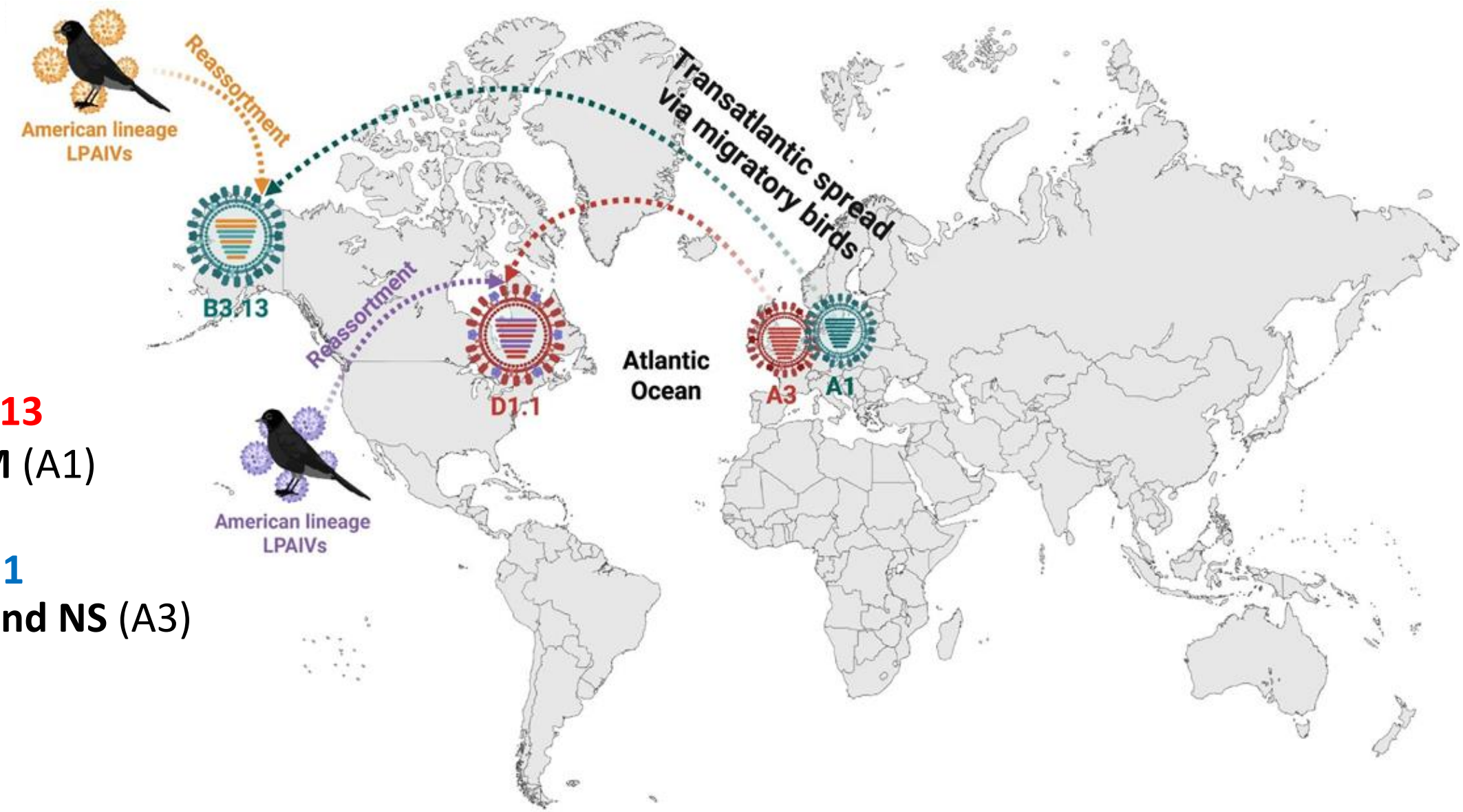
New version of bird flu infects Nevada dairy worker

This version of the virus is circulating broadly in wild birds and is different from the virus that has been causing dairy cow outbreaks since early 2024, the CDC said.

Yesterday at 3:22 p.m. PT

Listen to article 4 min

Dairy cows eat in a feeding barn in Nicasio, California. (David Paul Morris/Bloomberg News)

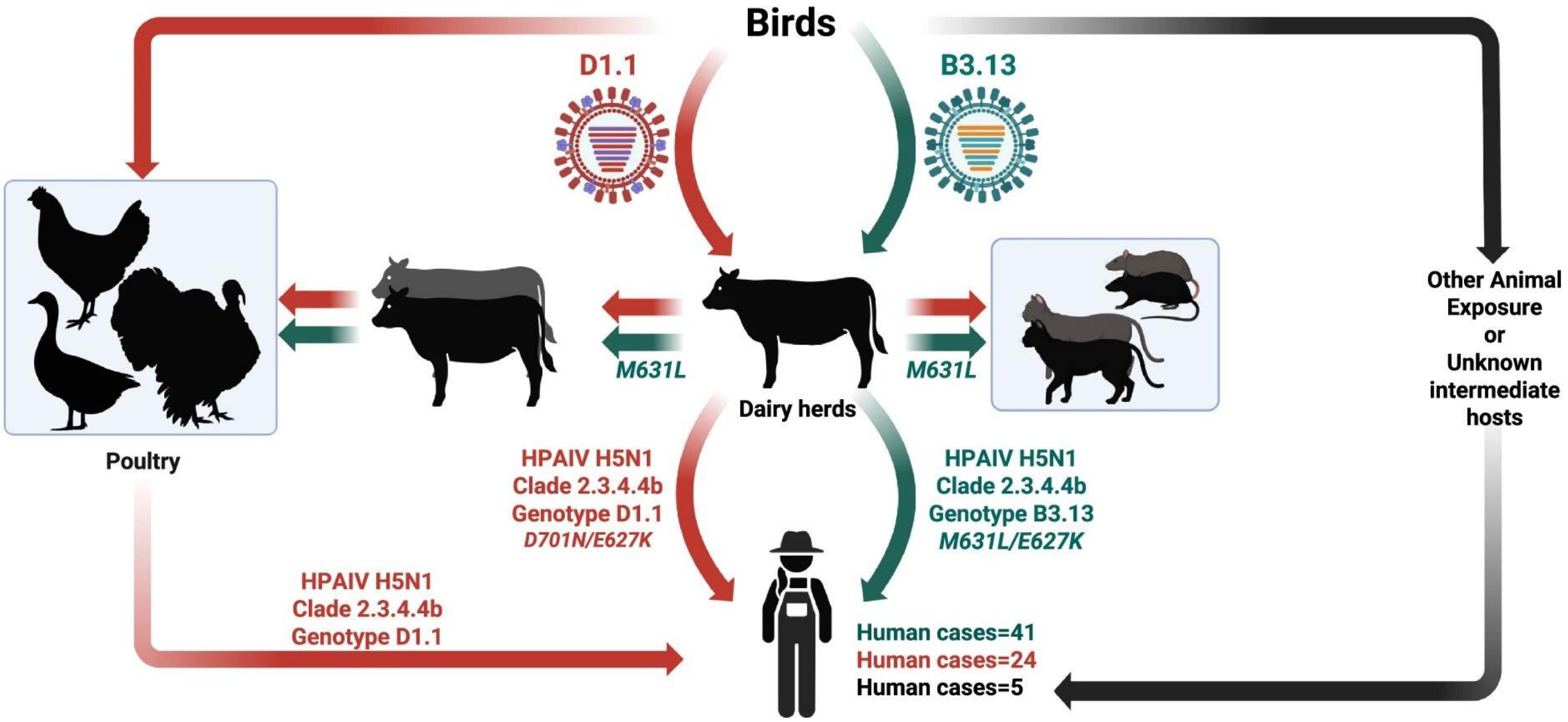


Genotype B3.13

PA, HA, NA, M (A1)

Genotype D1.1

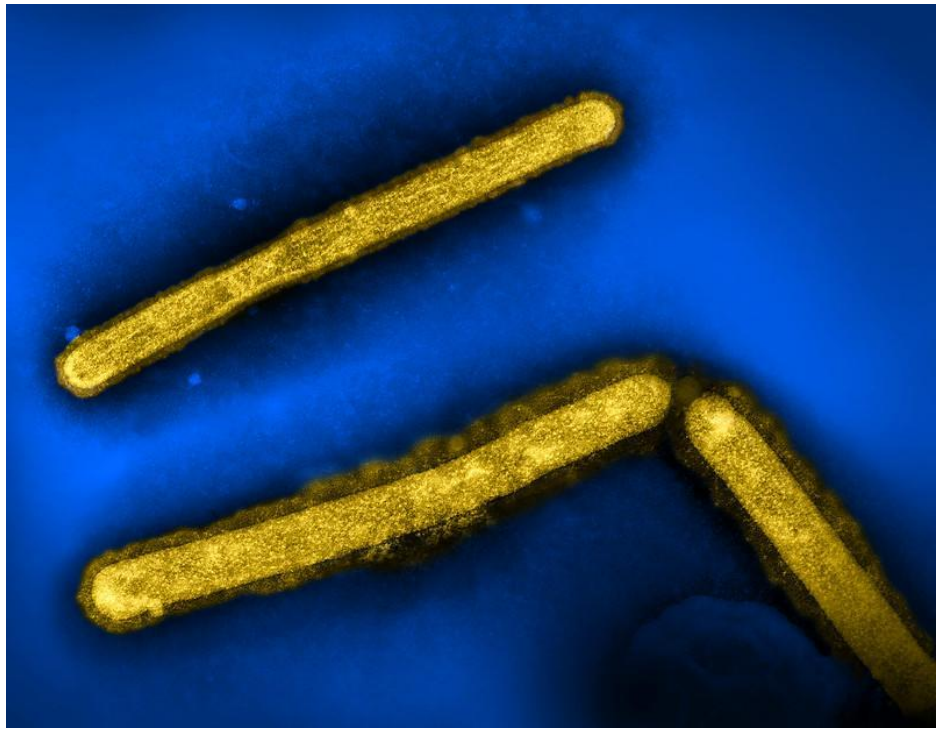
PB1, HA, M, and NS (A3)


Birds
D1.1
B3.13
Poultry
M631L
M631L
Dairy herds
Other Animal Exposure or Unknown intermediate hosts
HPAIV H5N1
Clade 2.3.4.4b
Genotype D1.1
D701N/E627K
HPAIV H5N1
Clade 2.3.4.4b
Genotype B3.13
M631L/E627K
HPAIV H5N1
Clade 2.3.4.4b
Genotype D1.1
Human cases=41
Human cases=24
Human cases=5

Mexico's fatal H5N1 case involved D1.1 genotype, which has been tied to serious illness

Lisa Schnirring, *April 18, 2025*
Topics: Avian Influenza (Bird Flu)

The child was transferred to a tertiary care hospital and died on April 8 due to respiratory complications. Along with the initial unsubtypable influenza A virus, tests also identified parainfluenza 3. The H5N1 finding was confirmed by polymerase chain reaction (PCR) testing on April 1, and genetic sequencing revealed that the virus belonged to the 2.3.4.4b clade and the D1.1 genotype, the same one linked to serious infections in the United States and British Columbia, Canada.



Comparison of B3.13 (cattle genotype) and D1.1 (dominant genotype in birds in the USA)

B3.13 exhibits mammalian adaptability, while D1.1 retains avian adaptability

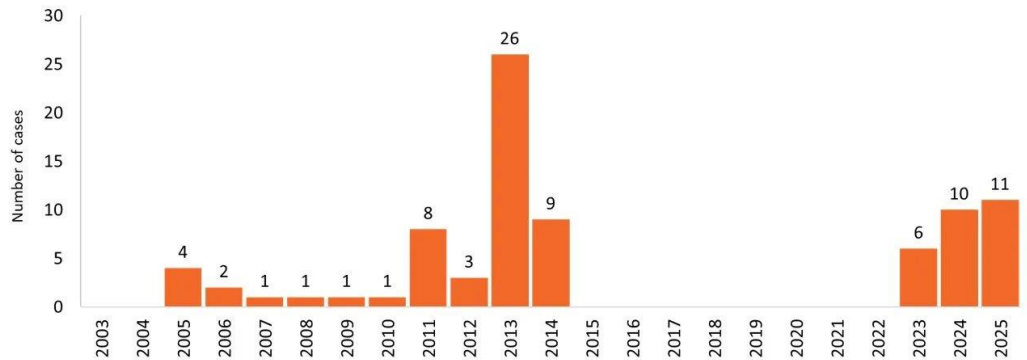
	Mutation numbering	B3.13 (2,839)	D1.1(300)	H5NX 2.3.4.4b (Human, 65)
Receptor binding site (RBS)	HA-133	/	/	L133R (1.5%)
	HA-135	V135M (26.3%), V135A (0.14%), V135E (0.04%)	/	V135M (15.4%)
	HA-137	/	/	A137T (1.5%)
	HA-185	S185P (0.04%)	/	S185P (3.1%)
	HA-189	/	/	E189A (1.5%)
	HA-192	T192A (0.39%)	/	T192I (24.6%), T192R (1.5%)
	HA-193	N193T (0.04%)	/	N193K (3.1%)
	HA-199	/	T199N (0.66%), T199A (0.33%)	T199I (38.5%)
	HA-217	/	I217L (0.66%)	/
	HA-221	S221P (0.07%)	S221P (0.33%)	/
	HA-222	Q222R (0.04%), Q222L (0.04%)	/	/
	HA-226	/	/	Q226L (1.5%)
Cleavage site	HA-323	S323N (52.9%)	S323N (2.3%)	S323N (30.8%)
	HA-324	P324L (0.88%)	/	/
	HA-325	L325P (1.9%)	/	L325P (3.1%)
	HA-326	/	R326I (0.66%)	R326K (1.5%)
	HA-328	/	/	K328R (15.4%), K328T (3.1%)
	HA-330	R330K (0.11%)	R330I (0.66%)	/
	HA-331	K331R (0.32%)	/	/

Protein	Mutation	B3.13(2,839)	D1.1(300)	H5NX 2.3.4.4b (Human, 65)
Neuraminidase (NA) active site	NA-70	N70S (80.5%)	/	N70S (33.8%)
	NA-110	S110Y (0.11%)	/	S110E (33.8%)
	NA-136	/	/	Q136H (3.1%)
	NA-197		P197S (0.33%)	
	NA-198	/	/	D198N (32.3%)
	NA-222	I222S (0.04%), I222T (0.04%)	/	/
	NA-247	S247N (0.04%)	S247N (1.67%)	/
	NA-436	I436S (0.07%)	/	I436L (33.8%)
Polymerase acidic (PA)	PA-24	Y24H (0.14%)	/	/
	PA-38	I38V (0.11%), I38M (0.04%)	/	I38M (1.5%)
	PA-105	F105S (0.07%)	/	/
	PA-122	V122I (0.04%)	/	/
	PA-137	K137R (0.07%)	/	/
Matrix 2 (M2)	M2-13	N13S (3.87%)	N13T (2%), N13S (0.33%)	N13S (16.9%)
	M2-16	E16G (0.39%)	E16G (0.33%), E16D (1%)	E16G (1.5%))
	M2-27	V27I (0.53%), V27A (0.04%)	V27A (0.66%)	V27T (1.5%)
	M2-28	I28T (2.9%), I28N (0.04%)	I28V (1%)	/
	M2-30	A30S (0.07%)	/	/
	M2-31	/	S31N (5%)	S31N (3.1%)

Current Research in Microbial Sciences 8 (2025) 100377

Table 1: Details of Avian Influenza A (H5N1) cases reported in Cambodia between 1 January- 1 July 2025

Indicator	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10	Case 11
Date of notification	10-Jan-25	26-Feb-25	23-Mar-25	28-May-25	14-Jun-25	17-Jun-25	21-Jun-25	23-Jun-25	25-Jun-25	29-Jun-25	1-Jul-25
Province	Kampong Cham	Prey Veng	Kratie	Kampong Speu	Takeo	Takeo	Svay Rieng	Siem Reap	Siem Reap	Siem Reap	Siem Reap
Sex	M	M	M	M	F	M	M	F	F	M	F
Age (years)	18-65	<5	<5	5-<18	18-65	<5	18-65	18-65	18-65	5-<18	18-65
Date symptom onset	1-Jan-25	17-Feb-25	18-Mar-25	18-May-25	4-Jun-25	7-Jun-25	14-Jun-25	18-Jun-25	Asymptomatic	Asymptomatic	23-Jun-2025
Date hospitalization	7-Jan-25	20-Feb-25	21-Mar-25	22-May-25	7-Jun-25	9-Jun-25	18-Jun-25	21-Jun-25	Not hospitalized	Not hospitalized	29-Jun-2025
Date sample collection	9 Jan-25	24-Feb-25	22-Mar-25	26-May-25	11-Jun-25	13-Jun-25	19-Jun-25	22-Jun-25	23-Jun-25	27-Jun-25	29-Jun-2025
Date of laboratory confirmation	10-Jan-25	25-Feb-25	22-Mar-25	27-May-25	12-Jun-25	16-Jun-25	20-Jun-25	23-Jun-25	24-Jun-25	28-Jun-25	30-Jun-2025
Date discharge	Not applicable	Not applicable	Not applicable	Not applicable	24-Jun-25	Not applicable	Not applicable	Still hospitalized as of 2 July	Not applicable	Not applicable	Still hospitalized
Exposure	Sick poultry	Sick poultry	chickens	Sick poultry	Sick poultry	Sick poultry	handling and culling chickens	Dead chickens	Sick poultry	Sick poultry	Sick poultry
Status	Died on 10 January 2025	Died on 25 February 2025	Died on 23 March 2025	Died on 27 May 2025	recovered and discharged	Died on 14 June 2025	Died on 19 June 2025	Stable as of 2 July 2025	Stable	Stable	Severe and hospitalized
Close contacts	16	69	21	15	77	9	20	3	3	3	3



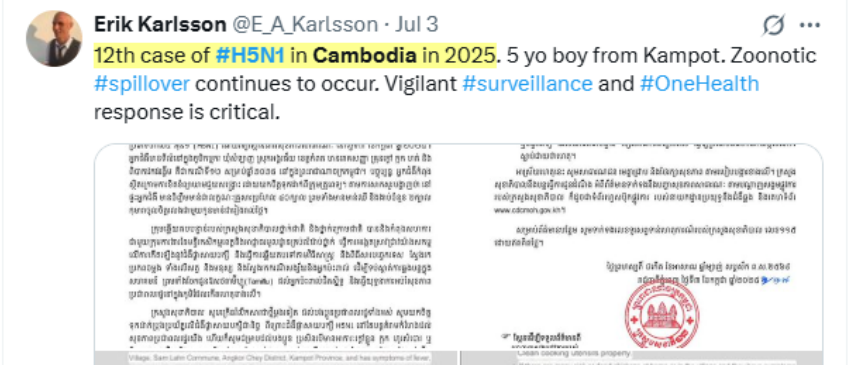
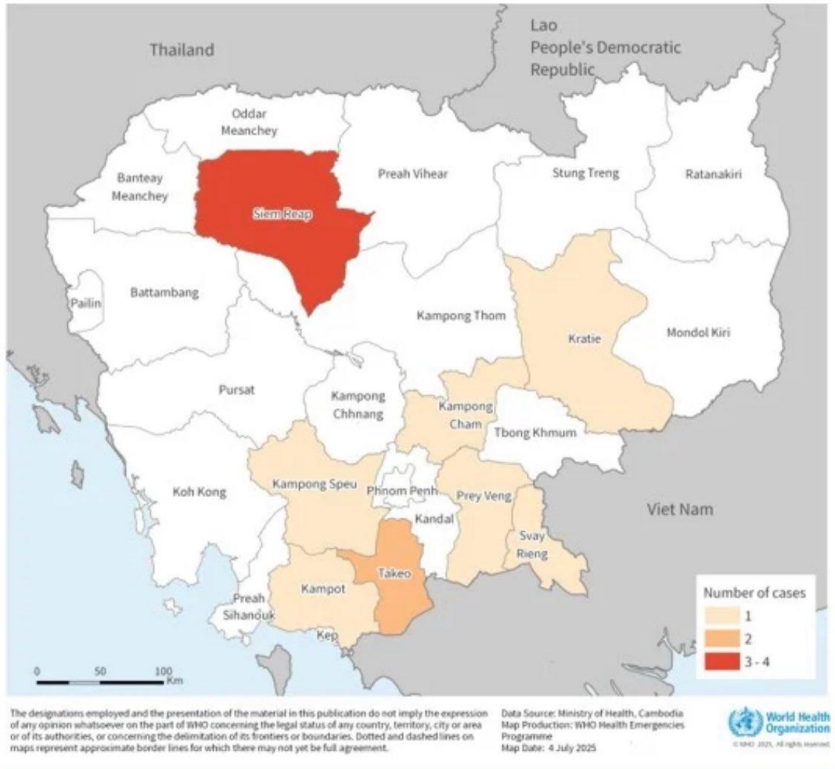
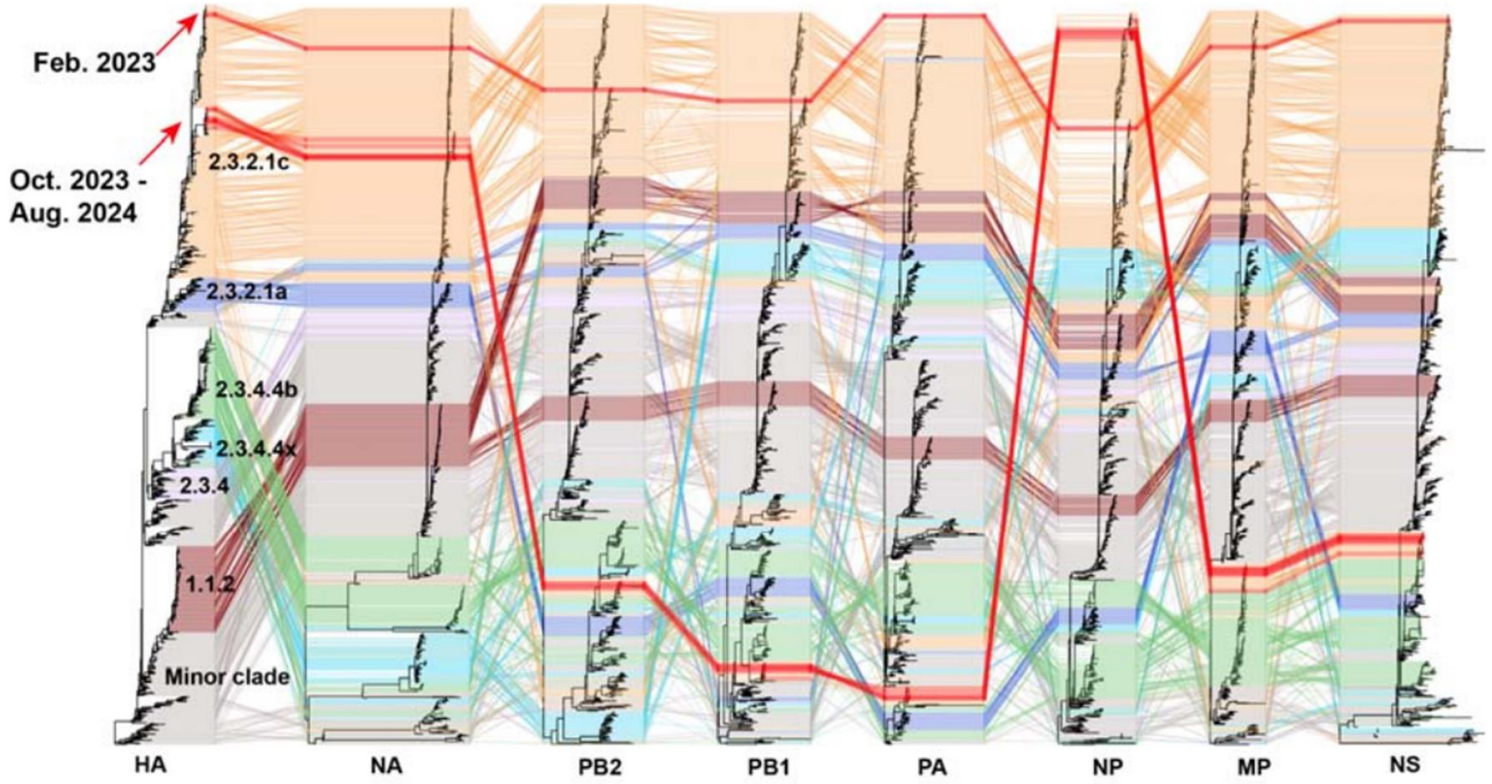


Figure 2: Geographic distribution of Avian Influenza A (H5N1) cases reported in Cambodia from 1 January – 1 July 2025.



Emergence of a Novel Reassortant Clade 2.3.2.1c Avian Influenza A/H5N1 Virus Associated with Human Cases in Cambodia

Jurre Y Siegers, Ruopeng Xie, Alexander M P Byrne,
Kimberly M Edwards, Shu Hu, Sokhoun Yann, Sarath Sin, Songha Tok,



Why Cambodia's novel H5N1 reassortant virus needs close monitoring

Premium

All available data suggest human infections caused by the reassortant virus are attributed to direct poultry-to-human transmission, with no evidence of human-to-human spread

Published – November 09, 2024 11:00 pm IST

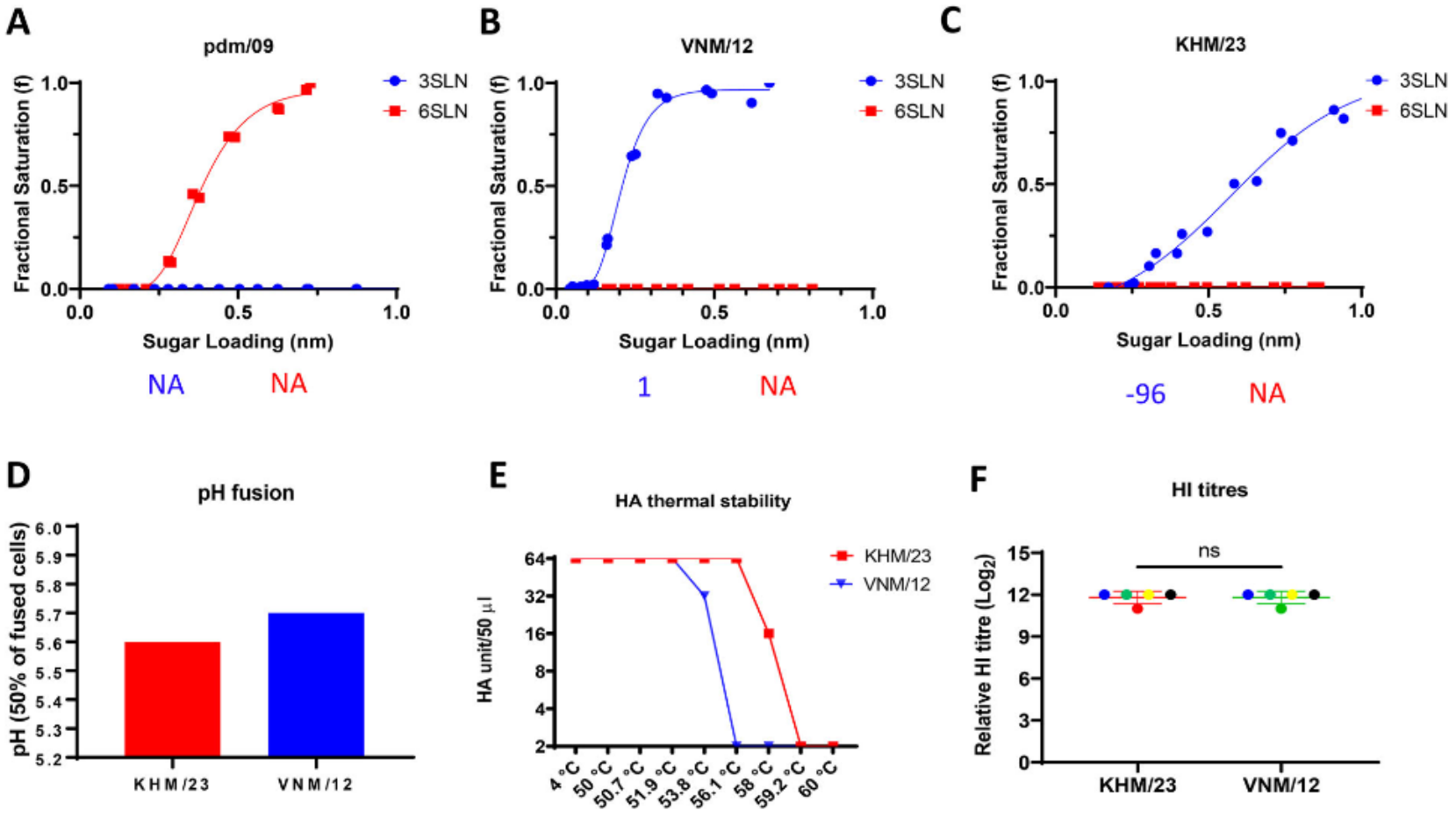
READ LATER PRINT

2023: 2.3.2.1c (all 8 segments)

2024: Reassortant between
2.3.2.1c (HA, NA, NP)
and
2.3.4.4b (PB2, PB1, PA, M, NS)

Emerging Microbes & Infections
2023, VOL. 12, 2244091 (4 pages)
https://doi.org/10.1080/22221751.2023.2244091

Characterization of the haemagglutinin properties of the H5N1 avian influenza virus that caused human infections in Cambodia



- Exhibited similar receptor binding and antigenicity to earlier clade 2.3.2.1c strain
- Did not bind to human-like receptors
- Increased thermal stability and reduced pH of fusion (enhance environmental persistence and transmissibility)
- Antigenicity tests showed no significant drift between KHM/23 and early clade 2.3.2.1c strains.

Thank you

anan.jon@biotec.or.th