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Molecular characteristics of endemic animal viruses: Insights from ecology, evolution, and case studies

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ABSTRACT

Endemic animal viruses represent a critical but often underappreciated dimension of global virology and One Health. Persisting within specific wildlife reservoirs, these viruses typically cause little pathology in their natural hosts, yet they harbor molecular traits that enable adaptation, immune evasion, and, under the right ecological circumstances, cross-species transmission. Advances in high-throughput sequencing and molecular characterization have revealed the genomic plasticity, receptor-binding versatility, and immune-modulatory strategies that underpin viral persistence and pathogenic potential. This critical review synthesizes current knowledge of the molecular characteristics of endemic viruses, focusing on three representative families with significant implications for livestock, wildlife, and human health: *Orthomyxoviridae* (avian influenza viruses in waterfowl), *Paramyxoviridae* (henipaviruses in bats), and *Herpesviridae* (malignant catarrhal fever viruses in wild ruminants). We employed a structured literature search to identify key molecular determinants, such as polymerase adaptations, glycoprotein receptor usage, and latency mechanisms, that contribute to ecological fitness and spillover potential. The review integrates these molecular findings with ecological perspectives, emphasizing how factors such as habitat encroachment and wildlife–livestock interfaces amplify risks. By bridging molecular virology with ecology, we advocate for a comprehensive One Health approach to understanding endemic animal viruses.

Keywords*:* Endemic animal viruses; Molecular virology; Paramyxoviridae; Orthomyxoviridae; Herpesviridae.

ABSTRAK

KARAKTERISTIK MOLEKULER VIRUS HEWAN ENDEMIK: PERSPEKTIF DARI EKOLOGI, EVOLUSI, DAN STUDI KASUS. Virus hewan endemik merupakan dimensi penting namun seringkali kurang mendapat perhatian dalam virologi global dan pendekatan *One Health*. Kemampuan *survive* dalam reservoir satwa liar tertentu, virus-virus ini umumnya tidak menyebabkan patologi signifikan pada inang alaminya, namun memiliki sifat molekuler yang memungkinkan adaptasi, pengalihan sistem imun, dan, dalam kondisi ekologi tertentu, transmisi lintas spesies. Kemajuan dalam teknologi sekuensing dan karakterisasi molekuler telah mengungkap plastisitas genom, fleksibilitas pengikatan reseptor, serta strategi modulasi imun yang mendasari persistensi dan potensi patogenik virus. Tinjauan kritis ini mensintesis pengetahuan terkini mengenai karakteristik molekuler virus endemik, dengan fokus pada tiga famili representatif yang memiliki implikasi signifikan bagi kesehatan ternak, satwa liar, dan manusia: *Orthomyxoviridae* (virus influenza burung pada unggas air), *Paramyxoviridae* (henipavirus pada kelelawar), dan *Herpesviridae* (virus demam ganas pada ruminansia liar). *Review* ini dilakukan melalui penelusuran literatur terstruktur untuk mengidentifikasi

determinan molekuler utama, seperti adaptasi *polimerase*, penggunaan glikoprotein reseptor, dan mekanisme laten yang berkontribusi terhadap kebugaran ekologi dan potensi penularan. Ulasan ini mengintegrasikan temuan molekuler tersebut dengan perspektif ekologi, menyoroti bagaimana faktor-faktor seperti perambahan habitat dan interaksi satwa liar dan ternak meningkatkan risiko. Melalui penjembatani virologi molekuler dan ekologi pendekatan *One Health* yang komprehensif dalam memahami virus hewan endemik dapat dipahami.

Kata Kunci: Virus hewan endemik; Virologi Moleculer; *Paramyxoviridae*; *Orthomyxoviridae*; *Herpesviridae*.

INTRODUCTION

Endemic animal viruses have coevolved with their natural hosts over long evolutionary timescales, often establishing stable, asymptomatic associations that ensure persistence within specific ecological niches (Cunningham *et al*., 2017; Kaján *et al*., 2020). Unlike epidemic or pandemic pathogens, these viruses circulate continuously at relatively low prevalence, contributing to the ecological equilibrium of host populations. Their molecular characteristics underpin their persistence, shaping replication strategies, immune evasion, and adaptability to fluctuating environments (Holmes, 2009; Han *et al*., 2023). The study of endemic viruses provides unique insights into the mechanisms of viral evolution and cross-species transmission.

This co-evolutionary dynamic is often governed by the *Red Queen* hypothesis, where the virus and host engage in a continuous molecular arms race. In reservoir hosts, this typically results in an evolutionary détente; viruses evolve mechanisms to subvert or evade host immunity without causing significant damage that would limit their own transmission potential (Woolhouse *et al*., 2012). For instance, the high mutation rates seen in RNA viruses allow for the constant generation of variants capable of escaping neutralizing antibodies, while DNA viruses often acquire host-homologous genes to modulate cytokine responses. Understanding these baseline molecular interactions in the natural reservoir is crucial, as they define the starting point for any potential adaptation to a new species.

Furthermore, the mechanisms of persistence differ fundamentally based on viral genome architecture. For RNA viruses, persistence is often maintained at the population level through rapid replication and transmission among susceptible cohorts (viral quasispecies), whereas DNA viruses, such as herpesviruses, utilize latency to persist at the individual level (Domingo *et al*., 2012). These distinct life history strategies impose different selective pressures on viral molecular machinery. Latency requires a sophisticated set of gene products to silence lytic replication and maintain the viral episome, creating a "molecular stealth" mode. In contrast, chronic RNA virus infections must constantly navigate the host's innate immune system, leading to the selection of potent interferon antagonists that, while optimized for the reservoir, may act as virulence factors in spillover hosts.

Many emerging infectious diseases (EIDs) of humans and livestock, including influenza, henipavirus infections, and malignant catarrhal fever, can be traced back to viruses that were historically endemic in wildlife reservoirs (Morse *et al*., 2012). This transition from benign circulation in reservoir hosts to pathogenic outbreaks in spillover hosts highlights the central role of molecular determinants, such as receptor-binding proteins and replication enzymes, in shaping host range and pathogenic potential (Woolhouse and Gowtage-Sequeria, 2005).

The barrier to cross-species transmission is largely defined by "molecular compatibility" between viral

surface proteins and host cell receptors. This lock-and-key interaction is the primary gatekeeper of spillover; however, successful emergence requires more than just entry (Parrish et al., 2008). Intracellular host restriction factors, such as APOBEC3 cytidine deaminases, tetherins, and IFITM proteins, serve as a second line of defense, forcing viruses to adapt their replication machinery. Consequently, the molecular history of an emerging virus often reveals a stepwise accumulation of mutations: first to gain entry, and subsequently to antagonize these intracellular restriction factors. Viruses that possess "generalist" molecular features, such as the conserved receptor usage seen in some paramyxoviruses, possess a distinct advantage in navigating these barriers compared to specialists.

Intermediate hosts often play a critical role in bridging the molecular gap between wildlife reservoirs and humans. In these "mixing vessel" species, such as swine for influenza or horses for Hendra virus, viruses undergo a period of rapid adaptation or recombination (Smith *et al*., 2009; Drexler *et al*., 2012; Escudero-Pérez *et al*., 2023). This ecological stepping stone allows the virus to optimize its molecular determinants for mammalian replication before encountering humans. For example, the adaptation of avian influenza polymerases to mammalian temperatures often occurs in intermediate hosts, enhancing replicative fitness. Therefore, analyzing the molecular characteristics of viruses in these transitional hosts provides essential clues about the specific genetic changes required to transform an endemic wildlife virus into a potential pandemic threat.

Recent technological advances, particularly in next-generation sequencing, structural biology, and molecular virology, have accelerated our understanding of viral genome diversity and host–virus interactions (Shi *et al*., 2016). These tools reveal not only the extraordinary adaptability of viral genomes but also the conserved strategies employed to balance persistence and transmission (Lipkin and Firth, 2013). The integration of molecular data with ecological observations now allows us to build a more complete picture of how viral traits influence disease emergence and ecosystem health. In this review, we synthesize current knowledge on the molecular features of endemic animal viruses with emphasis on three case studies: avian influenza viruses in wild waterfowl (Orthomyxoviridae), henipaviruses in bats (Paramyxoviridae), and malignant catarrhal fever viruses in wild ruminants (Herpesviridae).

Unbiased metagenomic sequencing has revolutionized our view of the "virosphere," revealing that the vast majority of viruses exist without causing discernable disease in their hosts. This "dark matter" of virology challenges the traditional pathogen-centric view and suggests that pathogenicity is the exception rather than the rule (Letko *et al*., 2020). By characterizing the genomes of these non-pathogenic viruses, researchers can identify the "molecular background" against which pathogenic traits evolve. Furthermore, advances in phylodynamics allow us to reconstruct the timing and spatial origins of viral dispersal, linking molecular evolution directly to ecological events such as migration or habitat fragmentation. This capability enables the tracking of specific mutations as they move through wildlife populations in real-time.

Complementing genomics, breakthroughs in structural biology, such as cryo-electron microscopy (cryo-EM), have unveiled the atomic-level details of viral glycoproteins and polymerase complexes (Tong *et al*., 2013). These structures provide a physical map of the virus-host interface, identifying the precise amino acid residues responsible for receptor specificity and immune evasion. By mapping evolutionary changes onto these 3D structures, we can predict the functional consequences of mutations found in surveillance data. This review leverages these structural insights to explain why

specific viral families, despite their ancient origins, remain potent sources of emerging disease, bridging the gap between abstract genomic data and concrete biological function.

METHODS

To conduct this critical review, a comprehensive literature search was performed using the Scopus, PubMed, and Web of Science databases. The search strategy targeted peer-reviewed articles published primarily between 2000 and 2024, with the inclusion of seminal papers from earlier dates to provide historical context.

Search strategy and selection criteria

Keywords used included combinations of "endemic animal viruses," "viral reservoirs," "molecular mechanisms of spillover," "viral persistence," "avian influenza ecology," "bat paramyxoviruses," and "malignant catarrhal fever." Boolean operators were employed to refine the search (e.g., "viral evolution" AND "reservoir host" AND "molecular determinants").

The inclusion criteria focused on:

1. Molecular Focus: Studies elucidating specific viral genomic features (e.g., receptor binding, polymerase fidelity, latency genes).
2. Ecological Context: Papers linking molecular traits to ecological factors (e.g., host density, migration, habitat overlap).
3. Target Families: Literature specifically addressing *Orthomyxoviridae*, *Paramyxoviridae*, and *Herpesviridae* to allow for comparative analysis across different genome types (segmented RNA, non-segmented RNA, and DNA).

Data were extracted and synthesized to identify convergent and divergent evolutionary strategies. The review was structured to critically evaluate how these molecular mechanisms function within the "One Health" framework.

RESULTS AND DISCUSSION

Comparative molecular features of endemic viruses

The literature analysis reveals distinct molecular strategies employed by different virus families to maintain endemicity and facilitate spillover (Table 1).

Orthomyxoviridae: Genomic Plasticity

Orthomyxoviridae, particularly avian influenza viruses (AIV) in waterfowl, exemplify genomic plasticity. The review identified the segmented RNA genome as a critical molecular determinant, allowing for high rates of antigenic drift and shift ((Webster *et al*., 1992). This mechanism sustains viral diversity within aquatic ecosystems, ensuring the maintenance of a global influenza gene pool (Olsen *et al*., 2006). Ecologically, this results in a stable reservoir system that paradoxically serves as the origin for pandemic strains upon reassortment in intermediate hosts (Smith *et al*., 2009; Taubenberger and Kash, 2010).

Paramyxoviridae: Receptor Versatility

Paramyxoviridae in bats, in contrast, demonstrate molecular conservation coupled with functional versatility. Structural biology studies confirm that their glycoproteins target conserved ephrin molecules (Ephrin-B2/B3) shared across multiple mammalian taxa (Halpin *et al*., 2007). The results indicate a fine balance between polymerase fidelity (for genomic stability) and adaptability (Drexler *et al*., 2012). These features interact with bat immunology, specifically dampened STING-dependent interferon pathways (Baker *et al*., 2013), to support asymptomatic persistence and long-range dispersal (Calisher *et al*., 2006; Wang and Anderson, 2019).

Tabel 1. Key molecular features of representative endemic animal viruses and their ecological consequences

Virus family	Representative virus (host)	Key molecular features	Ecological consequences	Spillover potential
Orthomyxoviridae	Avian influenza (waterfowl)	Segmented genome, high mutation/reassortment	Stable in aquatic reservoirs	High (swine, humans)
Paramyxoviridae	Henipaviruses (bats)	Receptor-binding plasticity (Ephrin-B2/B3)	Persistence in bat colonies	High (livestock, humans)
Herpesviridae	MCF viruses (ruminants)	Latency, immune evasion	Asymptomatic in wild hosts	High (cattle, bison)

Herpesviridae: Latency as a Strategy

Herpesviridae in wild ruminants utilize latency for persistence. Molecular analysis shows that viral genomes establish episomal maintenance, with latency-associated proteins preventing immune clearance (Davison, 2010). However, the synthesis of pathological data reveals that these same immune evasion strategies trigger lethal immunopathology (Malignant Catarrhal Fever) in spillover hosts like cattle and bison (Alcami and Koszinowski, 2000; Huang *et al*., 2025).

Integration of molecular and ecological determinants

The review findings support a conceptual framework where molecular traits are not static but are modulated by ecological forces.

1. Receptor-Binding Flexibility: Enables host jumps when ecological barriers (e.g., habitat segregation) are breached (Bonaparte *et al*., 2005).
2. Viral Shedding and Stability: Molecular traits determining environmental stability interact with host density to drive transmission intensity (Plowright *et al*., 2017).
3. Spillover Interfaces: The data highlights that spillover is most frequent at anthropogenic interfaces (e.g., pig farms for Nipah virus), where molecular susceptibility meets high contact rates (Chua *et al*., 2000; Jones *et al*., 2013).

The synthesis of molecular and ecological data suggests that endemic viruses are not merely "waiting" to emerge but are actively shaped by the co-evolutionary pressures of their reservoir hosts (Woolhouse et al., 2012; Geoghegan et al., 2016).

The Balance of stasis and plasticity

A critical finding of this review is the dichotomy between stability and plasticity. Paramyxoviruses maintain high genomic conservation to survive in bats yet possess "generalist" receptor-binding domains to exploit new hosts (Marsh and Helenius, 2006; Can *et al*., 2024). Conversely, Influenza A relies on constant genomic flux (reassortment) to persist (Keawcharoen *et al*., 2008; de Wit and Munster, 2015; Taylor *et al*., 2023). This suggests that surveillance

strategies must be tailored: sequencing for reassortants in influenza, versus serological screening for conserved antigens in paramyxoviruses.

Immunological dissonance

The severity of disease in spillover hosts often stems from "immunological dissonance." The review highlights that immune evasion mechanisms evolved for the reservoir host (e.g., bat interferon antagonism) often cause hyper-inflammation in novel hosts (Russell *et al*., 2009). This supports the theory that pathogenicity is an accidental byproduct of host mismatch rather than an evolved viral trait (Afonso *et al*., 2006).

Ecological drivers of molecular evolution

The discussion must also address the anthropogenic drivers. Agricultural intensification acts as an evolutionary incubator, selecting for viral variants with higher replication rates or altered tropism (Jones *et al*., 2008; Daszak *et al*., 2000). The Nipah virus case study illustrates this: the virus did not change significantly, but the *context* (piggeries) amplified a strain capable of efficient porcine replication (Epstein *et al*., 2006; Luby *et al*., 2009).

Limitations and future directions

Current literature is biased towards viruses with known zoonotic potential (e.g., Influenza, Nipah), leaving the "virosphere" of non-pathogenic endemic viruses largely unexplored (Shi *et al*., 2016). Future research must focus on unbiased metagenomics to understand the baseline diversity of endemic viruses before they emerge (Letko *et al*., 2020; Tong *et al*., 2013).

CONCLUSION

Endemic animal viruses embody a delicate balance between molecular adaptability and ecological stability. This critical review demonstrates that persistence is maintained through finely tuned strategies,antigenic plasticity, receptor versatility, and latency, which can become liabilities during ecological disruption. We conclude that molecular data alone are insufficient to predict emergence; they must be interpreted within specific ecological and evolutionary contexts. Effective pandemic preparedness requires a "One Health" execution strategy that integrates molecular surveillance with ecological monitoring to anticipate spillover risks before they manifest.

REFFERENCES

Afonso C.L., E.R. Tulman, G. Delhon, Z. Lu, G.J. Viljoen, D.B. Wallace, G.F. Kutish, D.L Rock, 2006. Genome of Crocodilepox Virus. J Virol80:.https://doi.org/10.1128/jvi .80.10.4978-4991.2006

Alcami A and U.H. Koszinowski, 2000. Viral mechanisms of immune evasion. Trends Microbiol. 2000 Sep;8(9):410-8. doi: 10.1016/s0966-842x(00)01830-8. PMID: 10989308; PMCID: PMC7141568.

Baker M.L., T. Schountz, and L.F. Wang, 2013. Antiviral immune responses of bats: a review. Zoonoses and Public Health, 60 (1): 104-116.

Bonaparte M.I., A.S. Dimitrov, K.N. Bossart, G. Crameri, B.A. Mungall, K.A. Bishop, V, Choudhry, D.S. Dimitrov, L.F. Wang, B.T. Eaton, C.C. Broder, 2005. Ephrin-B2 ligand is a functional receptor for Hendra virus and Nipah virus. Proc Natl Acad Sci U S A. 2005 Jul 26;102(30):10652-7. doi: 10.1073/pnas.0504887102. Epub 2005 Jul 5. PMID: 15998730; PMCID: PMC1169237.

Calisher C.H., J.E. Childs, H.E. Field, K.V. Holmes, and T. Schountz, 2006. Bats: important reservoir hosts of

emerging viruses. Clinical Microbiology Reviews, 19(3): 531–545.

Can C., X. Wang, Y. Liu, 2024 Stability of ecological systems: A theoretical review, Physics Reports, Volume 1088, 2024, Pages 1-41, ISSN 0370-1573, https://doi.org/10.1016/j.physrep.2024.08.001.

Chua K.B., W.J. Bellini, P.A. Rota, B.H. Harcourt, A. Tamin, S.K Lam, T.G Ksiazek, P.E Rollin, S.R. Zaki, W. Shieh, C.S. Goldsmith, D.J. Gubler, J.T. Roehrig, B. Eaton, A.R.Gould, J. Olson, H. Field, P. Daniels, A.E. Ling, C.J. Peters, L.J. Anderson, B.W. Mahy, 2000. Nipah virus: a recently emergent deadly paramyxovirus. Science (New York, N.Y.), *288*(5470), 1432–1435. https://doi.org/10.1126/science.288.5470.1432

Cunningham, A.A., P. Daszak, and J.L.Wood, 2017. One Health, emerging infectious diseases and wildlife: two decades of progress? Philosophical Transactions of the Royal Society B: Biological Sciences, 372(1725), 20160167.

Daszak P., A.A. Cunningham, and A.D. Hyatt, 2000. Emerging infectious diseases of wildlife--threats to biodiversity and human health. Science, 287(5452), 443-449.

Davison A.J. 2010. Herpesvirus systematics. Veterinary Microbiology, 143(1): 52-69.

de Wit E., and V.J. Munster, 2015. Evolution of avian influenza viruses in wild birds. *EcoHealth*, 12(1), 122–133.

Domingo E., J. Sheldon, and C. Perales, 2012. Viral quasispecies evolution. Microbiology and Molecular Biology Reviews, 76(2): 159-216.

Drexler, J. F., V. M. Corman, M. A. Müller, G.D. Maganga, P. Vallo, T. Binger, F. Gloza-Rausch, V.M. Cottontail, A. Rasche, S. Yordanov, A. Seebens, M. Knörnschild, S. Oppong, Y. Adu Sarkodie, C. Pongombo, A.N. Lukashev, J. Schmidt-Chanasit, A. Stöcker, A.J. Carneiro, S. Erbar, A. Maisner, F. Fronhoffs, R. Buettner, E.K.V. Kalko, T.Kruppa, C.R. Franke, R. Kallies, E.R.N. Yandoko, G. Herrler, C. Reusken, A. Hassanin, D.H. Krüger, S. Matthee, R.G. Ulrich, E.M. Leroy, C. Drosten, 2012. Bats host major mammalian paramyxoviruses. Nature communications, 3, 796. https://doi.org/10.1038/ncomms1796

Epstein J.H., H.E. Field, S. Luby, J.R. Pulliam, and P. Daszak, 2006. Nipah virus: impact, origins, and causes of emergence. Current Infectious Disease Reports, 8(1), 59-65.

Escudero-Pérez B., A. Lalande, C. Mathieu, P. Lawrence, 2023 Host-Pathogen Interactions Influencing Zoonotic Spillover Potential and Transmission in Humans. Viruses. 2023 Feb 22;15(3):599. doi: 10.3390/v15030599. PMID: 36992308; PMCID: PMC10060007.

Geoghegan J. L., A.M. Senior, F. Di Giallonardo, and E.C. Holmes, 2016. Virological factors that increase the transmissibility of emerging human viruses. Proceedings of the National Academy of Sciences of the United States of America, 113(15), 4170–4175. https://doi.org/10.1073/pnas.1521582113

Halpin K., A.D. Hyatt, R.K. Plowright, J.H. Epstein, P. Daszak, H.E. Field, L.

Wang, P.W. Daniels, and Henipavirus Ecology Research Group, 2007. Emerging viruses: coming in on a wrinkled wing and a prayer. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America, 44(5): 711–717. https://doi.org/10.1086/511078

Han J.J., H.A Song, S.L. Pierson, J. Shen-Gunther, Q. Xia, 2023. Emerging Infectious Diseases Are Virulent Viruses-Are We Prepared? An Overview. Microorganisms. 2023 Oct 24;11(11):2618. doi: 10.3390/microorganisms11112618. PMID: 38004630; PMCID: PMC10673331.

Holmes E.C., 2009. The evolution and emergence of RNA viruses. Genetics, 183(2), 357-369.

Huang H.S., L. Chen, J. Chi, S. Lai, J. Pi, Y. Shao, and J.Xu, 2025. Stress granules and cell death: crosstalk and potential therapeutic strategies in infectious diseases. Cell Death Dis **16**, 495 (2025). https://doi.org/10.1038/s41419-025-07800-z

Jones K.E., N.G. Patel, M.A. Levy, A. Storeygard, D. Balk, J.L. Gittleman, and P. Daszak, 2008. Global trends in emerging infectious diseases. Nature, 451(7181): 990–993.

Jones B.A., Grace, D., Kock, R., et al. (2013). Zoonosis emergence linked to agricultural intensification and environmental change. Proceedings of the National Academy of Sciences, 110(21): 8399-8404.

Kaján G. L., A. Doszpoly, Z.L Tarján, M.Z. Vidovszky, and T. Papp, 2020. Virus-Host Coevolution with a Focus on Animal and Human DNA Viruses. Journal of molecular evolution, 88(1), 41–56. https://doi.org/10.1007/s00239-019-09913-4

Keawcharoen J., D. van Riel, G. van Amerongen, T. Bestebroer, W.E. Beyer, R. van Lavieren, A.D. Osterhaus, R.A. Fouchier, and T. Kuiken, 2008. Wild ducks as long-distance vectors of highly pathogenic avian influenza virus (H5N1). Emerging infectious diseases, *14*(4): 600–607. https://doi.org/10.3201/eid1404.071016

Letko M., S.N. Seifert, K.J. Olival, R.K. Plowright, and V.J. Munster, 2020. Bat-borne virus diversity, spillover and emergence. Nature Reviews Microbiology, 18(8), 461-471.

Lipkin W.I. and C. Firth, 2013. Viral surveillance and discovery. Current Opinion in Virology, 3(2), 199-204.

Luby S.P., M.J. Hossain, E. Gurley, B.N. Ahmed, S. Banu, S.U. Khan, N. Homaira, P.A. Rota, P.E. Rollin, J.A. Comer, E. Kenah, T.G. Ksiazek, M. Rahman, 2009. Recurrent zoonotic transmission of Nipah virus into humans, Bangladesh, 2001-2007. Emerging Infectious Diseases, 15(8), 1229.

Morse S.S., J.A. Mazet, M. Woolhouse, C.R. Parrish, D. Carroll, W.B. Karesh, C. Zambrana-Torrelio, W.I. Lipkin, and P. Daszak, 2012. Prediction and prevention of the next pandemic zoonosis. Lancet (London, England), 380(9857), 1956–1965. https://doi.org/10.1016/S0140-6736(12)61684-5

Olsen B., V.J. Munster, A. Wallensten, J. Waldenström, A.D. Osterhaus, and R.A. Fouchier, 2006. Global patterns of influenza a virus in wild birds. Science (New York, N.Y.), 312(5772), 384–388. https://doi.org/10.1126/science.1122438

Parrish C. R., E.C. Holmes, D.M. Morens, E.C. Park, D.S. Burke, C.H. Calisher, C.A. Laughlin, L.J. Saif, and P. Daszak, 2008. Cross-species

virus transmission and the emergence of new epidemic diseases. Microbiology and molecular biology reviews : MMBR, 72(3), 457–470. https://doi.org/10.1128/MMBR.00004-08

Plowright R.K., R.P. Colin, H. McCallum, P.J. Hudson, A.I. Ko, A.L. Graham, and J.O. Lloyd-Smith, 2017. Pathways to zoonotic spillover. Nat Rev Microbiol 15, 502–510 (2017). https://doi.org/10.1038/nrmicro.2017.45

Russell G.C., J.P. Stewart, and D.M. Haig, 2009. Malignant catarrhal fever: a review. The Veterinary Journal, 179(3): 324-335.

Shi M., X.D. Lin, J.H. Tian, L.J. Chen, X. Chen, C.X. Li, X.C. Qin, J. Li, J.P. Cao, J.S. Eden, J. Buchmann, W. Wang, J. Xu, E.C. Holmes, and Y.Z. Zhang, 2016. Redefining the invertebrate RNA virosphere. Nature, 540(7634), 539–543. https://doi.org/10.1038/nature20167

Gavin J. D. Smith, D. Vijaykrishna, J. Bahl, S.J. Lycett, M. Worobey, O.G. Pybus, S.K. Ma, C.L. Cheung, J. Raghwani, S. Bhatt, J.S.M. Peiris, Y. Guan, and A. Rambaut, 2009. Origins and evolutionary genomics of the 2009 swine-origin H1N1 influenza A epidemic. Nature 459, 1122–1125. https://doi.org/10.1038/nature08182

Taubenberger J.K., and J.C. Kash, 2010. Influenza virus evolution, host adaptation, and pandemic formation. Cell Host & Microbe, 7(6): 440-451.

Taylor K.Y., I. Agu, I. José I, S. Mäntynen, A.J. Campbell, C. Mattson, T.W. Chou, B. Zhou, D. Gresham, GE. hedin E, Díaz, S.L.Muñoz, 2023. Influenza A virus reassortment is strain dependent. PLoS Pathog. 2023 Mar 1;19(3):e1011155. doi: 10.1371/journal.ppat.1011155. PMID: 36857394; PMCID: PMC10010518.

Tong S., X. Zhu, Y. Li, M. Shi, J. Zhang, M. Bourgeois, H. Yang, X. Chen, S. Recuenco, J. Gomez, L.M. Chen, A. Johnson, Y. Tao, C. Dreyfus, W. Yu, R. McBride, P.J. Carney, A.T. Gilbert, J. Chang, Z. Guo, C.T. Davis, J.C. Paulson, J. Stevens, C.E. Rupprecht, E.C. Holmes, I.A. Wilson, R.O. Donis, 2013. New world bats harbor diverse influenza A viruses. PLoS pathogens, 9(10), e1003657. https://doi.org/10.1371/journal.ppat.1003657

Wang L.F. and D.E. Anderson, 2019. Viruses in bats and potential spillover to animals and humans. Current Opinion in Virology, 34, 79-89.

Woolhouse M.E.J., and S. Gowtage-Sequeria, 2005. Host range and emerging and reemerging pathogens. Emerging Infectious Diseases, 11(12), 1842–1847.

Woolhouse M., F. Scott, Z. Hudson, R. Howey, and M. Chase-Topping, 2012. Human viruses: discovery and emergence. Philosophical transactions of the Royal Society of London. Series B, Biological sciences, 367(1604), 2864–2871. https://doi.org/10.1098/rstb.2011.0354