

Analytical Arguments on Viruses and Viral Diseases

Shyam Patidar¹ Abhishek Vyas²

patidarshyam5492@gmail.com¹ abhishekvyasav49@gmail.com²

Dept. of Chemical Sciences, Christian Eminent College, Indore (MP) India¹ Dept. of Biotechnology, Govt. Holkar Science College, Indore (MP) India²

Abstract

The evolutionary account of viruses represents a captivating, topic for virologists, immunologist and cell biologists. Analytical sequence about the origin, structure and life cycles of viruses with their interaction with immune system and response to different kind of medication lend a hand to budding scientists in their research as a single point reference. Due to pandemic lots of analytical work could not be done on analyzers. However researchers will find this paper very useful in their studies.

Introduction

Arguments on Origin

There is much debate among virologists about this question. Three main hypotheses have been articulated. The progressive, or escape, hypothesis states that viruses arose from genetic elements that gained the ability to move between cells; the regressive, or reduction, hypothesis asserts that viruses are remnants of cellular organisms; and the virus-first hypothesis states that viruses predate or coevolved with their current cellular hosts. Because of the great diversity among viruses, biologists have struggled with how to classify these entities and how to relate them to the conventional tree of life. They may represent genetic elements that gained the ability to move between cells. They may represent previously free-living organisms that became parasites. They may be the precursors of life

Concept of Viruses

We know that viruses are quite diverse. Unlike all other biological entities, some viruses, like poliovirus, have RNA genomes and some, like herpes virus, have DNA genomes. Further, some viruses (like influenza virus) have single-stranded genomes, while others (like smallpox) have double-stranded genomes. Their structures and **replication** strategies are equally diverse. Viruses, do, however, share a few features: First, they generally are quite small, with a diameter of less than 200 nanometers (nm). Second, they can replicate only within a host cell. Third, no known virus contains **ribosomes**, a necessary component of a cell's protein-making translational machinery.

Living or Non Living

To consider this question, we need to have a good understanding of what we mean by "life." Although specific definitions may vary, biologists generally agree that all living organisms exhibit several key properties. They can grow, reproduce, maintain an internal homeostasis, respond to stimuli, and carry out various metabolic processes. In addition, populations of living organisms evolve over time.

Hypothesis

According to this hypothesis, viruses originated through a progressive process. Mobile genetic elements, pieces of genetic material capable of moving within a genome, gained the ability to exit one cell and enter another.

The progressive and regressive hypotheses both assume that cells existed before viruses. What if viruses existed first? Recently, several investigators proposed that viruses may have been the first replicating entities. Koonin and Martin (2005) postulated that viruses existed in a precellular world as self-replicating units. Over time these units, they argue, became more organized and more complex. Eventually, enzymes for the synthesis of membranes and cell walls evolved, resulting in the formation of cells. Viruses, then, may have existed before bacteria, archaea, or eukaryotes (Figure 4; Prangishvili *et al.* 2006).

Most biologists now agree that the very first replicating molecules consisted of RNA, not DNA. We also know that some RNA molecules, ribozymes, exhibit enzymatic properties; they can catalyze chemical reactions. Perhaps, simple replicating RNA molecules, existing before the first cell formed, developed the ability to infect the first cells. Could today's single-stranded RNA viruses be descendants of these precellular RNA molecules?

Identification of Viruses (Vishaanu) according to Ayurveda

We know that all diseases occurs due to virus, bacteria, and fungus. Virus is mostly at top for causes of most of diseases. Description of virus is found in all modern books. If someone is ask that, is there is description of virus in Ayurveda? than might everyone will be confused and probable answer is negative. Introduction Life on earth was began nearly 3.5 billion years ago. Various changes occurs on earth from that that time to till present. Evidences show that condition on earth is different than todays. The atmosphere doesn't has oxygen and ozone layers. Harmful radiations and all unfavourable for origin of life, still life starts to developed in such extreme condition [1]. Microorganisms cover a large part of the earth. Microorganisms are microscopic organisms or cell clusters. Microorganisms has major role in maintain of eco systems. at some cases Microorganisms causes serious harm. Bacteria, archaea, fungi, protozoa, algae and virus are types of Microorganisms. Virus is one of the types among that six. We all known that Ayurveda is not only one pathy but its science of life.as virus is one of the part of life than it must be described in Ayurvedic samhita. We need to take effort to find out its descriptions. In Ayurvedic view, we can compare virus with an aam. This aam produced virkut rasa dhatu. Ultimately it produced abnormal cell. This cell has abnormal DNA and gens. Protein synthesis occurs in RNA will be abnormal.at last gene which is itself protein molecules will be become abnormal. This genetic material made from abnormal protein is virus

Classification of Viruses

Morphology

Viruses are grouped on the basis of size and shape, chemical composition and structure of the genome, and mode of replication. Helical morphology is seen in nucleocapsids of many filamentous and pleomorphic viruses. Helical nucleocapsids consist of a helical array of capsid proteins (protomers) wrapped around a

helical filament of nucleic acid. Icosahedral morphology is characteristic of the nucleocapsids of many “spherical” viruses. The number and arrangement of the capsomeres (morphologic subunits of the icosahedron) are useful in identification and classification. Many viruses also have an outer envelope.

Chemical Composition and Mode of Replication

The genome of a virus may consist of DNA or RNA, which may be single stranded (ss) or double stranded (ds), linear or circular. The entire genome may occupy either one nucleic acid molecule (monopartite genome) or several nucleic acid segments (multipartite genome). The different types of genome necessitate different replication strategies.

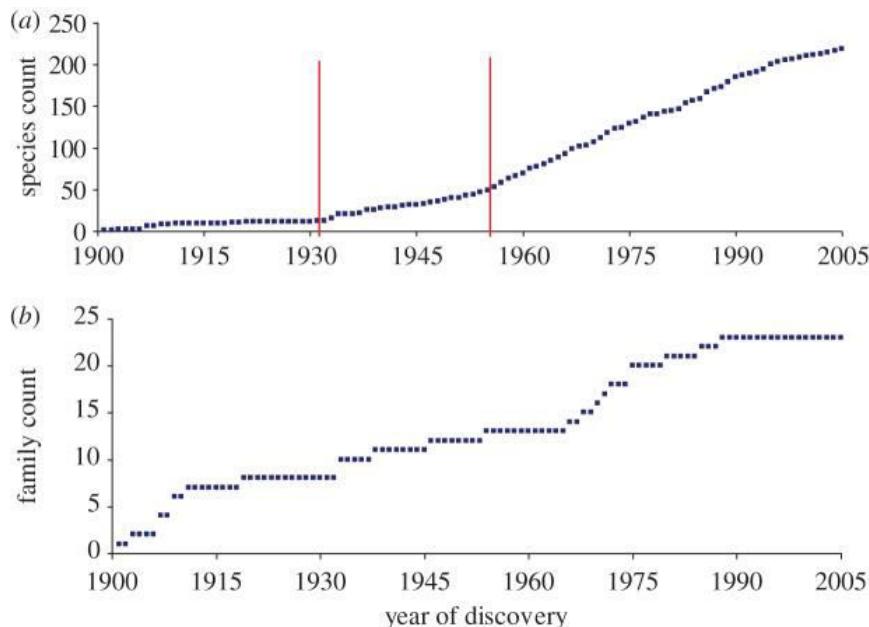
RNA Virus Genomes

RNA viruses, comprising 70% of all viruses, vary remarkably in genome structure. Because of the error rate of the enzymes involved in RNA replication, these viruses usually show much higher mutation rates than do the DNA viruses. Mutation rates of 10^{-4} lead to the continuous generation of virus variants which show great adaptability to new hosts. The viral RNA may be single-stranded (ss) or double-stranded (ds), and the genome may occupy a single RNA segment or be distributed on two or more separate segments (segmented genomes). In addition, the RNA strand of a single-stranded genome may be either a sense strand (plus strand), which can function as messenger RNA (mRNA), or an antisense strand (minus strand), which is complementary to the sense strand and cannot function as mRNA protein translation. Sense viral RNA alone can replicate if injected into cells, since it can function as mRNA and initiate translation of virus-encoded proteins. Antisense RNA, on the other hand, has no translational function and cannot per se produce viral components

Human Viruses and associated Pathologies

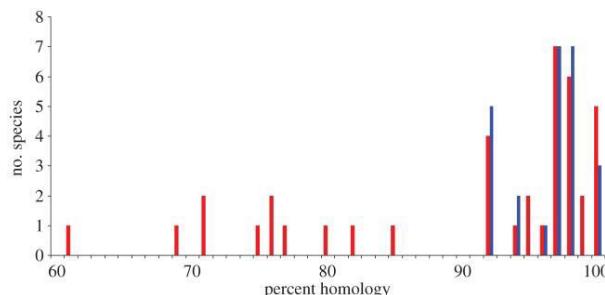
The table below displays the list of human viral pathogens, with transmission and general facts about associated pathologies.

Useful Charts And Graphs For The Analytical Approach



Discovery curves for human viruses. (a) Virus discovery curve by species. Cumulative number of species reported to infect humans. Statistically significant upward breakpoints are shown (vertical lines). (b)

Virus discovery curve by family. Cumulative number of families containing species reported to infect humans.



Number of virus species with broad (blue bars) or narrow (red bars) host range as a function of the percent homology of the cell receptor used

Latest Virus - Analytical Review

Asymptomatic -2019–20 Corona Virus Pandemic

The **2019–20 corona virus pandemic** is an ongoing pandemic of corona virus disease 2019 (COVID-19), caused by severe acute respiratory syndrome corona virus 2 (SARS-CoV-2).^[4] The outbreak was first identified in Wuhan, Hubei, China, in December 2019, and was recognised as a pandemic by the World Health Organization (WHO) on 11 March 2020.^[5] As of 24 March, more than 392,000 cases of COVID-19 have been reported in more than 190 countries and territories, resulting in more than 17,100 deaths and more than 102,000 recoveries.

The virus is typically spread from one person to another via respiratory droplets produced during coughing. It primarily spreads when people are in close contact but may also spread when one touches a contaminated surface and then one's face. It is most contagious when people are symptomatic, although spread may be possible before symptoms appear.^[1] The time between exposure and symptom onset is typically around five days, but may range from two to fourteen days. Common symptoms include fever, cough, and shortness of breath. Complications may include pneumonia and acute respiratory distress syndrome. There is currently no vaccine or specific antiviral treatment. Primary treatment is symptomatic and supportive therapy. Recommended preventive measures include hand washing, covering the mouth when coughing, maintaining distance from other people, and monitoring and self-isolation for people who suspect they are infected.

Prevention Efforts

To prevent the virus spreading include travel restrictions, quarantines, curfews, event postponements and cancellations, and facility closures. These include a quarantine of Hubei, nationwide quarantines in Italy and elsewhere in Europe, curfew measures elsewhere in China and South Korea various border closures or incoming passenger restrictions, screening at airports and train stations, and travel advisories regarding regions with community transmission. Schools and universities have closed either on a nationwide or local basis in more than 124 countries, affecting more than 1.2 billion students.

The pandemic has led to global socioeconomic disruption, the postponement or cancellation of sporting, religious, and cultural events,^[25] and widespread fears of supply shortages which have spurred panic buying. Misinformation and conspiracy theories about the virus have spread online and there have been incidents of xenophobia and racism against Chinese and other East or Southeast Asian people.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), previously known by the provisional name **2019 novel coronavirus (2019-nCoV)**, is a positive-sense single-stranded RNA virus. It is contagious in humans and is the cause of the ongoing pandemic of coronavirus disease 2019 (COVID-19) that has been

designated a Public Health Emergency of International Concern by the WHO.



Conclusion

This paper is an effort to contribute in better understanding of the materialization of new human viruses as a biological and ecological process. This approach will allow researchers to refine their work looking currently available information which is very crude notions to study kinds of pathogens, or the kinds of circumstances, Researchers should be most concerned about the present situation before putting their efforts for detection and prevention the cause more efficiently.

Bibliography

- Caspar DLD: Design principles in virus particle construction. In Horsfall FL, Tamm I (eds): Viral and Rickettsial Infections in Man. 4th Ed. JB Lippincott, Philadelphia, 1975 .
- Fields BN (ed): Virology. 3rd Ed. Lippincott-Raven Press, 1995 .
- Gajdusek DC. Unconventional viruses and the origin and disappearance of kuru. *Science*. 1977;197:943. [PubMed]
- Gelderblom HR. Assembly and morphology of HIV: potential effect of structure on viral function. *AIDS*. 1991;5:617–637. [PubMed]
- Mattern CFT: Symmetry in virus architecture. In Nayak DP (ed): Molecular Biology of Animal Viruses. Marcel Dekker, New York, 1977 .
- 1. Levine A. J., Enquist L. W. 2007. History of virology. In Fields virology (eds Fields B. N., Knipe D. M., Howley P. M.), pp. 565–604, 5th edn Philadelphia, PA: Lippincott Williams & Wilkins [[Google Scholar](#)]
- 2. Woolhouse M. E. J., Gaunt E. 2007. Ecological origins of novel human pathogens. *Crit. Rev. Microbiol.* 33, 1–1210.1080/10408410601172164 ([doi:10.1080/10408410601172164](https://doi.org/10.1080/10408410601172164)) [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 3. Woolhouse M. E. J., Dye C. 2001. Population biology of emerging and re-emerging pathogens: preface. *Phil. Trans. R. Soc. Lond. B* 356, 981–98210.1098/rstb.2001.0899 ([doi:10.1098/rstb.2001.0899](https://doi.org/10.1098/rstb.2001.0899)) [[CrossRef](#)] [[Google Scholar](#)]
- 4. Parrish C. R. 1995. Molecular epidemiology of parvoviruses. *Semin. Virol.* 6, 415–41810.1016/S1044-5773(05)80018-6 ([doi:10.1016/S1044-5773\(05\)80018-6](https://doi.org/10.1016/S1044-5773(05)80018-6)) [[CrossRef](#)] [[Google Scholar](#)]
- 5. Woolhouse M. E. J., Howey R., Gaunt E., Reilly L., Chase-Topping M., Savill N. 2008. Temporal trends in the discovery of human viruses. *Proc. R. Soc. B* 275, 2111–

- 211510.1098/rspb.2008.0294 ([doi:10.1098/rspb.2008.0294](https://doi.org/10.1098/rspb.2008.0294)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 6. International Committee on Taxonomy of Viruses. www.ictvdb.org/Ictv/ICTVdBsearch.htm. (accessed 16 August 2010)
 - 7. Bebber D. P., Marriot F. H. C., Gaston K. J., Harris S. A., Scotland R. W. 2007. Predicting unknown species numbers using discovery curves. *Proc. R. Soc. B* 274, 1651–165810.1098/rspb.2007.0464 ([doi:10.1098/rspb.2007.0464](https://doi.org/10.1098/rspb.2007.0464)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 8. Storch G. A. 2007. Diagnostic virology. In *Fields virology* (eds Fields B. N., Knipe D. M., Howley P. M.), pp. 565–604, 5th edn Philadelphia, PA: Lippincott Williams & Wilkins [[Google Scholar](#)]
 - 9. Jones K. E., Patel N. G., Levy M. A., Storeygard A., Balk D., Gittleman J. L., Daszak P. 2008. Global trends in emerging infectious diseases. *Nature* 451, 990–99410.1038/nature06536 ([doi:10.1038/nature06536](https://doi.org/10.1038/nature06536)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 10. Keusch G. T., Pappaioanou M., Gonzalez M. C., Scott K. A., Tsai P. (eds) 2009. *Sustaining global surveillance and response to emerging zoonotic diseases*. Washington, DC: The National Academies Press [[Google Scholar](#)]
 - 11. Allander T., Tammi M. T., Eriksson M., Bjerkner A., Lindell A. T., Bjorn A. 2005. Cloning of a human parvovirus by molecular screening of respiratory tract samples. *Proc. Natl Acad. Sci. USA* 102, 12 891–12 89610.1073/pnas.0504666102 ([doi:10.1073/pnas.0504666102](https://doi.org/10.1073/pnas.0504666102)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 12. Greninger A. I., Runckel C., Chiu C. Y., Haggerty T., Parsonnet J., Ganem D., DeRisi J. L. 2009. The complete genome of klassevirus—a novel picornavirus in pediatric stool. *Virol. J.* 6, 82.10.1186/1743-422X-6-82 ([doi:10.1186/1743-422X-6-82](https://doi.org/10.1186/1743-422X-6-82)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 13. Allander T., Andreasson K., Gupta S., Bjerkner A., Bogdanovic G., Persson M. A. A., Dalianis T., Ramqvist T., Andersson B. 2007. Identification of a third human polyomavirus. *J. Virol.* 81, 4130–413610.1128/JVI.00028-07 ([doi:10.1128/JVI.00028-07](https://doi.org/10.1128/JVI.00028-07)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 14. Chua K. B., et al. 2007. A previously unknown reovirus of bat origin is associated with an acute respiratory disease in humans. *Proc. Natl Acad. Sci. USA* 104, 11 424–11 42910.1073/pnas.0701372104 ([doi:10.1073/pnas.0701372104](https://doi.org/10.1073/pnas.0701372104)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 15. Gaynor A. M., et al. 2007. Identification of a novel polyomavirus from patients with acute respiratory tract infections. *PLoS Pathog.* 3, e64.10.1371/journal.ppat.0030064 ([doi:10.1371/journal.ppat.0030064](https://doi.org/10.1371/journal.ppat.0030064)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
 - 16. Finkbeiner S. R., Kirkwood C. D., Wang D. 2008. Complete genome sequence of a highly divergent astrovirus isolated from a child with acute diarrhea. *Virol. J.* 5, 117.10.1186/1743-422X-5-117 ([doi:10.1186/1743-422X-5-117](https://doi.org/10.1186/1743-422X-5-117)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]

- 17. Towner J. S., et al. 2008. Newly discovered Ebola virus associated with hemorrhagic fever outbreak in Uganda. *PLoS Pathog.* 4, e1000212.10.1371/journal.ppat.1000212 ([doi:10.1371/journal.ppat.1000212](https://doi.org/10.1371/journal.ppat.1000212)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 18. Kapoor A., et al. 2008. A newly identified bocavirus species in human stool. *J. Infect. Dis.* 199, 196–20010.1086/595831 ([doi:10.1086/595831](https://doi.org/10.1086/595831)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 19. Kapoor A., et al. 2008. A highly prevalent and genetically diversified Picornaviridae genus in South Asian children. *Proc. Natl Acad. Sci. USA* 105, 20 482–20 48710.1073/pnas.0807979105 ([doi:10.1073/pnas.0807979105](https://doi.org/10.1073/pnas.0807979105)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 20. Holtz L. R., Finkbeiner S. R., Kirkwood C. D., Wang D. 2008. Identification of a novel picornavirus related to cosaviruses in a child with acute diarrhea. *Virol. J.* 5, 159–16410.1186/1743-422X-5-159 ([doi:10.1186/1743-422X-5-159](https://doi.org/10.1186/1743-422X-5-159)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 21. Finkbeiner S. R., et al. 2009. Identification of a novel astrovirus (astrovirus VA1) associated with an outbreak of acute gastroenteritis. *J. Virol.* 83, 10 836–10 83910.1128/JVI.00998-09 ([doi:10.1128/JVI.00998-09](https://doi.org/10.1128/JVI.00998-09)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 22. Li L., Barry P., Yeh E., Glaser C., Schnurr D., Delwart E. 2009. Identification of a novel human gammapapillomavirus species. *J. Gen. Virol.* 90, 2413–241710.1099/vir.0.012344-0 ([doi:10.1099/vir.0.012344-0](https://doi.org/10.1099/vir.0.012344-0)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 23. Li L., et al. 2009. A novel picornavirus associated with gastroenteritis. *J. Virol.* 83, 12 002–12 00610.1128/JVI.01241-09 ([doi:10.1128/JVI.01241-09](https://doi.org/10.1128/JVI.01241-09)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 24. Briese T., et al. 2009. Genetic detection and characterization of Lujo virus, a new hemorrhagic fever-associated arenavirus from Southern Africa. *PLoS Pathog.* 5, e1000455.10.1371/journal.ppat.1000455 ([doi:10.1371/journal.ppat.1000455](https://doi.org/10.1371/journal.ppat.1000455)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 25. Taylor L. H., Latham S. M., Woolhouse M. E. J. 2001. Risk factors for human disease emergence. *Phil. Trans. R. Soc. Lond. B* 356, 983–98910.1098/rstb.2001.0888 ([doi:10.1098/rstb.2001.0888](https://doi.org/10.1098/rstb.2001.0888)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 26. Woolhouse M. E. J., Gowtage-Sequeria S. 2005. Host range and emerging and re-emerging pathogens. *Emerg. Inf. Dis.* 11, 1842–184710.3201/eid1112.050997 ([doi:10.3201/eid1112.050997](https://doi.org/10.3201/eid1112.050997)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 27. Woolhouse M. E. J., Taylor L. H., Haydon D. T. 2001. Population biology of multi-host pathogens. *Science* 292, 1109–111210.1126/science.1059026 ([doi:10.1126/science.1059026](https://doi.org/10.1126/science.1059026)) [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 28. Simmonds P. 2001. Reconstructing the origins of human hepatitis viruses. *Phil. Trans. R. Soc. Lond. B* 356, 1013–102610.1098/rstb.2001.0890 ([doi:10.1098/rstb.2001.0890](https://doi.org/10.1098/rstb.2001.0890)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 29. Keele B. F., et al. 2006. Chimpanzee reservoirs of pandemic and non-pandemic HIV-1. *Science* 313, 523–52610.1126/science.1126531 ([doi:10.1126/science.1126531](https://doi.org/10.1126/science.1126531)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]

- 30. Wolfe N. D., Dunavan C. P., Diamond J. 2007. Origins of major human infectious diseases. *Nature* 447, 279–28310.1038/nature05775 ([doi:10.1038/nature05775](https://doi.org/10.1038/nature05775)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 31. Woolhouse M., Antia R. 2008. Emergence of new infectious diseases. In *Evolution in health and disease* (eds Stearns S. C., Koella J. K.), pp. 215–228, 2nd edn Oxford, UK: Oxford University Press [[Google Scholar](#)]
- 32. Wolfe N. D., et al. 2004. Naturally acquired simian retrovirus infections in central African hunters. *Lancet* 363, 932–93710.1016/S0140-6736(04)15787-5 ([doi:10.1016/S0140-6736\(04\)15787-5](https://doi.org/10.1016/S0140-6736(04)15787-5)) [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 33. Keeling M. J., Rohani P. 2008. Modelling infectious diseases in humans and animals. Princeton, NJ: Princeton University Press [[Google Scholar](#)]
- 34. Cleaveland S., Laurenson M. K., Taylor L. H. 2001. Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. *Phil. Trans. R. Soc. Lond. B* 356, 991–99910.1098/rstb.2001.0889 ([doi:10.1098/rstb.2001.0889](https://doi.org/10.1098/rstb.2001.0889)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 35. Antia R., Regoes R. R., Koella J. C., Bergstrom C. T. 2003. The role of evolution in the emergence of infectious diseases. *Nature* 426, 658–66110.1038/nature02104 ([doi:10.1038/nature02104](https://doi.org/10.1038/nature02104)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 36. Institute of Medicine 2003. Microbial threats to health: emergence, detection, and response. Washington, DC: National Academy Press [[Google Scholar](#)]
- 37. Woolhouse M. E. J., Haydon D. T., Antia R. 2005. Emerging pathogens: the epidemiology and evolution of species jumps. *Trends Ecol. Evol.* 20, 238–24410.1016/j.tree.2005.02.009 ([doi:10.1016/j.tree.2005.02.009](https://doi.org/10.1016/j.tree.2005.02.009)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 38. Streicker D. G., Turmelle A. S., Vonhof M. J., Kuzmin I. V., McCracken G. F., Rupprecht C. E. 2010. Host phylogeny constrains cross-species emergence and establishment of rabies virus in bats. *Science* 329, 676–67910.1126/science.1188836 ([doi:10.1126/science.1188836](https://doi.org/10.1126/science.1188836)) [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 39. Davies T. J., Pedersen A. B. 2008. Phylogeny and geography predict pathogen community similarity in wild primates and humans. *Proc. R. Soc. B* 275, 1695–170110.1098/rspb.2008.0284 ([doi:10.1098/rspb.2008.0284](https://doi.org/10.1098/rspb.2008.0284)) [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- 40. Universal Protein Resource. <http://beta.uniprot.org/> (accessed 05 April 2008)
- 41. King D. A., Peckham C., Waage J. K., Brownlie J., Woolhouse M. E. J. 2006. Infectious diseases: preparing for the future. *Science* 313, 1392–139310.1126/science.1129134 ([doi:10.1126/science.1129134](https://doi.org/10.1126/science.1129134)) [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]