MEDWIN PUBLISHERS
Committed to Create Value for Researchers

International Journal of Zoology and Animal Biology
ISSN: 2639-216X

Respiratory Viral Illnesses in Wild Primates Human-Associated Respiratory Viral Infections in Wild Great Apes: Epidemiology, Risk Factors, Clinical Manifestations, Mortality, and Conservation Implications-A Narrative Review

Suresh Antony J*

Center for Infectious diseases and Travel Medicine, Department of infectious diseases, USA

***Corresponding author:** Suresh J Antony, Center for Infectious diseases and Travel Medicine, Department of infectious diseases, ORCID 0000-0002-7677-5369, USA

Review Article
Volume 9 Issue 6
Received Date: November 17, 2026
Published Date: November 26, 2026
DOI: 10.23880/izab-16000707

Abstract

Respiratory viral disease is a major cause of illness and death in wild non-human primates, especially African great apes. Long-term field studies and molecular research have shown that human-associated respiratory viruses can cross species and cause disease in wild chimpanzees, gorillas, and bonobos. Viruses identified so far include human metapneumovirus (HMPV), human respiratory syncytial virus (HRSV), human rhinovirus C (HRV-C), human respirovirus 3, and human coronavirus OC43. Clinical signs can be very mild or as severe as pneumonia and death. Outcomes depend on the pathogen, the population, and individual factors such as age and health status. The connection between human activity and several documented outbreaks shows that reverse zoonosis is a significant conservation concern.

Diagnosing respiratory disease in free-ranging primates remains challenging because direct examination, respiratory sampling, and post-mortem analysis are usually not possible. However, the evidence base has grown with non-invasive molecular techniques, long-term behavioral observations, and better disease surveillance. This paper reviews the literature on epidemiology, transmission, risk factors, clinical signs, mortality, diagnostic challenges, and prevention of respiratory viral disease in wild primates. The evidence points to the need for better surveillance, reduced human-primate contact, and greater coordination among conservation, veterinary, and public health sectors. Major limitations include underdiagnosis, inconsistent case definitions, incomplete mortality data, and a focus on African great apes.

Keywords: Wild Primates; Respiratory Viruses; Chimpanzees; Gorillas; Reverse Zoonosis; Human Metapneumovirus; Rhinovirus; Conservation Medicine; Mortality; Disease Surveillance

Abbreviations

HMPV: Human Metapneumovirus; HRSV: Human Respiratory Syncytial Virus; HRV-C: Human rhinovirus C.

Introduction

Respiratory disease is a recognized threat to the health and conservation of wild nonhuman primates. Although

habitat loss, hunting, and other anthropogenic pressures remain major drivers of primate population decline, infectious disease may contribute to mortality in vulnerable populations. Köndgen and Kaur documented respiratory outbreaks associated with human-related viruses in wild chimpanzees [1,2].

Respiratory outbreaks have been investigated in wild chimpanzees in Côte d'Ivoire, Uganda, and Tanzania, and respiratory viruses of human origin have also been detected in other great apes. Molecular studies by Köndgen, Emery Thompson, and Negrey and colleagues provide evidence of human-associated pathogens in affected populations [1,4-7]. Such transmission is described as reverse zoonosis or anthroponosis; viral sequence similarity alone does not establish an individual source or precise transmission route.

Scully et al. investigated a 2013 outbreak of human rhinovirus C in wild chimpanzees in Uganda that was associated with several deaths [5]. Separate outbreaks involving human metapneumovirus and human respirovirus 3 occurred in neighboring chimpanzee communities in Uganda during 2016–2017 [7]. These findings emphasize that similar respiratory syndromes may have distinct etiologies.

Studying respiratory disease in wild primates is very difficult. Researchers cannot routinely capture and examine the animals, carcasses are often not recovered, and respiratory specimens are hard to obtain. As a result, most studies rely on behavioral observations, fecal samples, opportunistic post-mortems, and molecular analysis [4-14].

This paper reviews the published literature on respiratory viral disease in wild primates, focusing on epidemiology, risk factors, clinical signs, mortality, diagnosis, and conservation implications.

Materials and Methods

Review Design

We conducted a narrative review of published evidence on respiratory viral disease in free-ranging nonhuman primates. The literature considered included outbreak investigations, longitudinal observational studies, molecular diagnostic studies, reviews, and conservation guidelines. This was not designed as a systematic review or meta-analysis.

Literature Search Strategy

The manuscript was developed from searches of PubMed and relevant journal and institutional repositories using combinations of the following concepts. Exact search dates, full search strings, numbers screened, and duplicate-handling procedures were not documented; consequently, the search should not be regarded as exhaustive or reproducible. These details should be added if available before submission:

- Wild primates and respiratory disease
- Wild chimpanzees and respiratory virus outbreaks
- Human metapneumovirus and nonhuman primates
- Human respiratory syncytial virus and great apes
- Human rhinovirus C and chimpanzees
- Human coronavirus and wild apes
- Reverse zoonosis and primate conservation.
- Respiratory disease risk factors and primate mortality
- Noninvasive viral surveillance in wild primates

Inclusion and Exclusion Criteria

We focused on peer-reviewed original research on naturally occurring respiratory disease in free-ranging primates. We included studies that examined one or more of the following: outbreak epidemiology, pathogen identification, clinical signs, mortality, individual risk factors, diagnostic techniques, or disease prevention.

We included reviews and conservation guidelines that contributed to understanding transmission routes or recommended prevention.

We excluded studies on other viral diseases from the main synthesis unless they directly contributed to our understanding of respiratory disease surveillance or transmission.

Data Extraction and Synthesis

We organized the extracted findings into five sections: pathogen identification, epidemiology and transmission, risk factors, clinical presentation, and mortality. Diagnostic approaches and conservation implications were considered separately.

Published studies vary in observation period, population size, diagnostic certainty, and definitions of respiratory illness; therefore, the reported estimates should not be taken as directly comparable measures of risk across all primate species.

Results of the Literature Review

Respiratory Viruses Identified in Wild Primates

Several published investigations have examined human-associated respiratory viruses in wild great apes. Work by Köndgen, Scully, Emery, Negrey and Patrono indicated that human respiratory viruses can cross the species barrier

Copyright© Suresh Antony J.

and, in certain circumstances, cause significant disease in susceptible primate populations [1,4-8].

The principal human-associated respiratory viruses described in the selected literature are summarized below.

Human metapneumovirus (HMPV) has been identified in respiratory outbreaks among wild chimpanzees and associated with severe illness and death. Reported clinical signs include nasal discharge, cough, lethargy, and weakness. Molecular analyses support a close relationship between ape-associated viruses and human viral lineages [1,2,4,7].

Human respiratory syncytial virus (HRSV). HRSV has been identified during respiratory outbreaks in chimpanzees. Non-invasive testing of fecal samples has proven valuable in investigating outbreaks where direct respiratory sampling is not possible [4]. This specimen-collection approach has become more widely used in primate epidemiology. Respiratory disease due to HRSV has also been reported in other great ape populations [9].

Human rhinovirus C (HRV-C). HRV-C was the virus implicated in a fatal respiratory outbreak in a group of wild chimpanzees in Uganda in 2013. Scully described the molecular work and epidemiological analyses which together provided strong evidence that this was the causative agent of the outbreak [5].

Human respirovirus 3 (HRV3; formerly human parainfluenza virus 3) was identified during a respiratory outbreak in the Kanyawara chimpanzee community in Uganda in 2016–2017. Negrey et al. investigated this outbreak alongside a distinct HMPV outbreak in the neighboring Ngogo community [7]. The abbreviation HRV3 follows the cited study and should not be confused with human rhinovirus C (HRV-C).

Human coronavirus OC43. Patrono et al. found a human coronavirus during their investigation into an outbreak in wild chimpanzees in Côte d'Ivoire. This extends the list of human respiratory viruses that are seen in wild primates [8].

However, identifying these viruses does not prove that a human pathogen causes every respiratory outbreak in wild primates. Outbreaks will remain undiagnosed, and respiratory signs are not sufficient to differentiate between viral and bacterial infection, co-infection, or any number of other causes that can present similarly.

Epidemiology and Transmission

Respiratory outbreaks have been documented at long-term primate research sites including Taï National Park in Côte d'Ivoire, Kibale National Park in Uganda, and sites in Tanzania [1,2,4-7]. These observations demonstrate that outbreaks occur in multiple populations and geographic settings; however, the relative contributions of different exposure pathways remain uncertain.

Phylogenetic similarity between viruses detected in apes and circulating human lineages supports a human-associated origin in several investigations but does not identify the specific transmitting individual or establish the exact route [1,7,11]. Respiratory particles and close contact are plausible transmission pathways; the relative importance of contaminated surfaces remains uncertain. Conservation guidelines recommend reducing opportunities for human-to-ape pathogen transmission [12].

Habituated great apes may tolerate the proximity of researchers, trackers, tourists, and other personnel, creating opportunities for pathogen exposure. Transmission risk depends on distance, duration, environmental conditions, and the pathogen involved. People with symptomatic infection are a particular concern, although mild or asymptomatic infections may also contribute to transmission [11,12]. Specific visitor distances should be reported only when supported by site-level observations or guidelines.

Outbreak dynamics are pathogen specific. Negrey and colleagues documented simultaneous 2016–2017 outbreaks of HMPV at Ngogo and human respirovirus 3 at Kanyawara in Uganda. The estimated basic reproduction numbers were 1.27 and 1.48, respectively; these estimates are specific to the observed outbreaks and should not be generalized to other populations [7].

Non-invasive molecular surveillance as described earlier in the paper has improved our ability to identify pathogens in wild primates. Köndgen and colleagues showed that fecal samples collected during an outbreak can detect HMPV and HRSV, and that viral sequences can be compared with those obtained from tissue samples [4]. This is particularly valuable when obtaining respiratory specimens would require capture or restraint.

Risk Factors

Risk factors for respiratory illness can be considered at the population, individual, and environmental levels.

Human exposure: Introduction of human-associated pathogens is an important potentially preventable risk. Proximity to habituated apes may facilitate respiratory exposure involving researchers, tourists, park staff, or local communities. Nevertheless, the source of an individual outbreak is often difficult to establish [11,12].

Copyright© Suresh Antony J.

Detection of a viral sequence closely related to human lineages supports a human-associated origin, but does not identify the individual source, transmission direction in every case, or precise exposure pathway.

Age: Age appears to affect both susceptibility and clinical outcome, as seen in the Ugandan outbreaks examined by Negrey and colleagues, where HMPV-associated mortality occurred predominantly in infants and older chimpanzees [7].

These age-related patterns are study- and pathogen-dependent and may reflect differences in population structure, exposure, previous immunity, and sample size.

Social status and demographic characteristics: Long-term observations of wild chimpanzees have identified associations between respiratory signs and individual characteristics, including age and aspects of social status. In the Kanyawara community, respiratory signs were more common among older individuals and lower-ranking adolescent males [6].

These observational associations do not establish causality, because social rank may correlate with stress, behavior, contact patterns, or nutritional status.

Population contact and social behavior: Primates are social animals, and their proximity to one another will facilitate pathogen transmission. Grooming, proximity, and repeated social contact likely contribute to the spread of respiratory infections after introduction into a population. Primate population density and patterns of intergroup contact may also influence transmission, though field studies have not consistently quantified each factor's contribution.

Environmental and health-related factors: Habitat disturbance, nutritional stress and changes in human activity could all contribute to disease susceptibility. Scientific evidence linking any of these specific factors to the incidence or severity of respiratory viral disease in wild primates remains limited. This is less well established than the evidence for the introduction of human-associated pathogens.

Further work is needed to establish how environmental change, coinfection, immune status, and other underlying factors influence clinical outcome.

Clinical Presentation

Clinical manifestations range from mild respiratory signs to severe illness and death. Because direct examination is generally impractical in free-ranging animals, clinical descriptions usually rely on observed behavior and visible respiratory signs; necropsy findings provide additional information when carcasses are recovered.

Reported signs include:

- Coughing and sneezing
- Nasal discharge
- Increased respiratory effort or abnormal breathing
- Lethargy and reduced activity
- Decreased feeding or changes in normal behavior
- Signs consistent with lower respiratory tract disease
- Sudden deterioration and death in severe cases

These manifestations are nonspecific and may occur in viral infections, bacterial infections, or coinfections. Clinical signs alone cannot establish the etiologic agent [5,7].

Severe outbreaks may involve pneumonia and pulmonary pathology. Molecular analysis of respiratory tissue, combined with pathology and epidemiologic evidence, can strengthen etiologic attribution, as demonstrated in the 2013 HRV-C investigation [5]. Definitive pathological confirmation generally requires appropriate tissue sampling, most often at necropsy.

Coinfection is another issue. Bacterial pathogens can contribute to respiratory disease, and the presence of a virus doesn't rule out other infectious etiologies. The investigation should therefore consider the clinical picture, molecular findings, pathology, and epidemiological pattern (Tables 1-3).

Age	Infants and older individuals may be more vulnerable to severe respiratory disease and mortality.
Social status	Social rank may influence exposure, stress, and susceptibility to infection.
Social interactions	Grooming, close proximity, and group living may facilitate transmission within communities.
Population density	High population density and frequent contact may increase opportunities for pathogen spread.
Environmental stress	Habitat disturbance, nutritional stress, and other environmental pressures may affect immune function and disease susceptibility.
Coinfections	Concurrent viral or bacterial infections may contribute to more severe clinical outcomes.

Source: Synthesized from the studies discussed in this review [1,4-7,11,12].
Table 1: Potential risk factors for respiratory viral illness in wild primates.

Copyright© Suresh Antony J.

Respiratory virus	Reported great-ape host / setting	Associated clinical significance
Human metapneumovirus (HMPV)	Wild chimpanzees, Uganda	Respiratory outbreaks, pneumonia, and mortality.
Human respiratory syncytial virus (HRSV)	Wild chimpanzees and other great apes	Respiratory illness; identified through molecular testing, including noninvasive sampling.
Human rhinovirus C (HRV-C)	Wild chimpanzees, Uganda (2013)	Lethal respiratory outbreak associated with substantial mortality.
Human respirovirus 3 (HRV3)	Wild chimpanzees, Uganda (2016–2017)	Respiratory outbreak in a chimpanzee community.
Human coronavirus OC43	Wild chimpanzees, Taï National Park, Côte d'Ivoire	Identified during an outbreak investigation, demonstrating another potential human-to-ape transmission pathway.

Source: Synthesized from published outbreak investigations [1,4-8].
Table 2: Major human-associated respiratory viruses identified in wild great apes.

Mortality Rates and Population Consequences

Mortality differs substantially among outbreaks. The observed outcome depends on the pathogen, host susceptibility, population characteristics, and the degree of case and death ascertainment. No single mortality range is applicable across all respiratory viral infections in wild primates.

Three findings illustrate this variation:

Human rhinovirus C outbreak, Uganda, 2013: an outbreak investigation estimated that around 8.9% of the Kanyawara chimpanzee community died in association with the outbreak [5,14].

Human metapneumovirus outbreak, Uganda 2016–2017: The Ngogo outbreak had a mortality of 12.2%, with deaths occurring predominantly in infants and older chimpanzees [7].

Human respirovirus 3 outbreak, Uganda, 2016–2017: The Kanyawara outbreak was not associated with deaths directly attributed to this virus [7].

Mahale Mountains, Tanzania (2003, 2005, and 2006): Observed mortality was 6.9%, 3.2%, and 4.6%, respectively, with all observed deaths occurring among infants. For 2006, the estimate could rise to 18.5% if nine missing chimpanzees were presumed dead. A human-related metapneumovirus was detected in an affected chimpanzee in 2006, but attribution of every outbreak to that virus was not established [2].

These examples illustrate that viral detection, or the presence of respiratory signs cannot be used to infer a uniform mortality risk. Differences in pathogen biology, host factors, and ascertainment complicate comparisons between studies.

Long-term observations add context. At Kanyawara, respiratory disease was a major cause of death over the many years of the study by Emery Thompson, et al. [6] (27% of the recorded deaths). That applies only to the study period and population defined in the study, not to a general mortality rate for wild primates.

Mortality estimates are constrained by unrecovered carcasses, disappearance of animals, uncertain denominators, and variation in diagnostic confirmation. These limitations may be particularly consequential in small populations, where even a limited number of deaths can affect conservation outcomes.

Diagnostic Challenges and Surveillance

Diagnosing respiratory viral disease in wild primates is difficult and requires field-appropriate methods. Direct examination and sampling of the respiratory tract are often not feasible, and the availability of fresh carcasses is unpredictable.

Several approaches are useful: Behavioral and clinical surveillance. Standardized recording of coughing, nasal discharge, breathing abnormalities, lethargy, and changes in activity can help detect an emerging outbreak. Consistent case definitions will aid comparison over time.

Non-invasive molecular testing. Fecal samples have been used to detect respiratory viruses in wild chimpanzees as an alternative when respiratory specimens aren't available [4]. The sensitivity of a negative result will vary according to the specimen, the time of sampling, and the assay used;

Copyright© Suresh Antony J.

a negative fecal test does not preclude respiratory infection.

Post-mortem (necropsy) investigation of respiratory tissues can help establish the etiology of severe or fatal disease. Carcass degradation and delayed recovery will, of course, limit diagnostic yield in many cases.

Longitudinal monitoring. Long-term studies provide baseline data on respiratory signs and mortality, enabling investigators to distinguish an outbreak from normal fluctuations in health. Analysis. Sequencing can compare viral strains from primates and humans and help investigate likely pathogen origins. Sequence similarity supports epidemiological inference but may not, by itself, prove the precise direction or route of transmission.

An integrated approach combining standardized observations, noninvasive sampling, necropsy, molecular diagnostics, and epidemiologic investigation is preferable to reliance on a single method.

Discussion

Conservation Significance

Respiratory viral disease may cause acute mortality in already threatened great-ape populations. Detection of human-associated viruses reinforces the need to address infectious disease alongside habitat loss and hunting [1,11]. Potential longer-term effects on social structure and reproduction require further investigation.

The most compelling evidence comes from African great apes, particularly chimpanzees. It would be inappropriate to assume that the same viruses, risk factors, or mortality rates apply to all wild primates. Evidence from monkeys and other primate groups remains very weak, and more species-specific surveillance is needed to interpret mortality.

The evidence also highlights the distinction between detecting a virus and demonstrating clinically significant disease. Pathogen attribution is strongest when molecular findings are supported by consistent clinical, epidemiologic, and pathological observations.

Mortality estimates must be interpreted in light of the population at risk, observation period, diagnostic certainty, and whether deaths were observed or inferred from disappearance. These differences reduce comparability across studies and should be explicitly reported.

Future work would be improved by using standardized outbreak definitions, consistently reporting demographic characteristics, and clearly distinguishing all-cause mortality from mortality specifically attributable to the pathogen in question.

Prevention and Control

Prevention is central to conservation because treatment of free-ranging primates is often impractical and may itself introduce risk. Measures should aim to reduce opportunities for human-to-primate pathogen transmission.

Recommended measures include:

- Exclude people with respiratory symptoms from close-contact field activities.
- Require appropriate face masks when personnel work near great apes, in accordance with site-specific protocols.
- Maintain appropriate distances between visitors, researchers, and the animals.
- Apply hand hygiene and equipment cleaning procedures.
- Limit the number of people permitted near habituated primates.
- Train staff and visitors to recognize respiratory signs and report suspected outbreaks promptly.

Develop coordinated protocols for specimen collection, laboratory testing, and outbreak response.

Gilardi, et al. have defined conservation guidelines which stress the need for structured health monitoring and disease control in great ape populations [12]. Emerging evidence also suggests that more robust disease-prevention protocols can reduce signs of respiratory disease and viral burden at long-term field sites, supporting the utility of such preventive measures [15].

Protocols should be reviewed regularly and adapted to species, local conditions, human-contact patterns, and known pathogens. The objective is to permit essential conservation activities while minimizing preventable disease transmission.

Research Priorities

Respiratory disease surveillance is geographically uneven. More data are needed from populations beyond established African great-ape research sites, including monkeys and other threatened primates in additional regions.

The contributions of age, sex, social status, nutrition, stress, coinfection, and prior exposure require closer examination through longitudinal studies combining individual histories with pathogen testing.

Copyright© Suresh Antony J.

Noninvasive diagnostic methods require further validation, including assessment of specimen-specific sensitivity and optimal sampling times for individual pathogens.

Improved population-level estimates are needed for recurrent and subclinical infections, which may have consequences not captured by investigations of major outbreaks alone.

Integrated surveillance linking wildlife health, human respiratory pathogen monitoring, veterinarians, and conservation organizations could improve early detection and transmission investigations. This is consistent with a One Health approach.

Conclusion

Human-associated respiratory viruses pose a potentially serious threat to wild great apes. Outbreak investigations have implicated HMPV, HRSV, HRV-C, human respirovirus 3, and coronavirus OC43 in respiratory illness in free-ranging populations. Evidence is strongest for African chimpanzees, and findings should not be generalized uncritically to all wild primates.

Clinical outcomes range from mild illness to severe respiratory disease and death. Mortality differs markedly among outbreaks, and interpretation is limited by incomplete surveillance, variable diagnostic confirmation, and inconsistent ascertainment of deaths.

Priorities include standardized clinical surveillance, noninvasive molecular diagnostics, infection-prevention measures at human–ape interfaces, and expanded study of less-investigated primate populations. Recent field evidence of reduced respiratory signs after prevention protocols supports the practical importance of these measures [15].

Collaboration among primatologists, veterinarians, epidemiologists, conservation managers, and public health professionals is essential to strengthen outbreak preparedness and protect vulnerable primate populations.

Virus or topic	Location or population	Principal findings	Conservation significance
Human metapneumovirus	Taï chimpanzees, Côte d'Ivoire	Detected during respiratory outbreaks using noninvasive fecal sampling [4]	Supports molecular surveillance when direct sampling is impractical
Human rhinovirus C	Kanyawara chimpanzees, Uganda, 2013	Associated with a lethal outbreak; estimated community mortality approximately 8.9% [5,14]	Demonstrates that a common human respiratory virus can cause severe disease in wild apes
Human metapneumovirus	Ngogo chimpanzees, Uganda, 2016–2017	Reported mortality of 12.2%, particularly among infants and older animals [7]	Highlights potential age-related vulnerability and substantial outbreak impact
Human respirovirus 3	Kanyawara chimpanzees, Uganda, 2016–2017	Outbreak without direct mortality attributed to the virus in the reported study [7]	Shows that outbreaks caused by different viruses can have different outcomes
Human coronavirus OC43	Wild chimpanzees, Côte d'Ivoire	Identified in an outbreak investigation [8]	Broadens the range of human-associated viruses relevant to surveillance
Longitudinal respiratory surveillance	Kanyawara chimpanzees, Uganda	Respiratory illness contributed to 27% of recorded deaths over the study period [6]	Shows the value of long-term health and mortality records
Disease prevention	Long-term chimpanzee field site, Uganda	Stronger prevention protocols were associated with reductions in respiratory signs and viral quantity [15]	Supports preventive measures at human–primate interfaces

Note: Findings represent different studies, populations, observation periods, and methods of mortality ascertainment. They should not be interpreted as directly comparable estimates of risk across primate species.
Table 3: Summary Table of Selected Evidence.

Copyright© Suresh Antony J.

References

1. Köndgen S, Kühl H, N'Goran PK (2008) Pandemic human viruses cause decline of endangered great apes. Curr Biol 18(4): 260-264.

2. Kaur T, Singh J, Tong S (2008) Descriptive epidemiology of fatal respiratory outbreaks and detection of a human-related metapneumovirus in wild chimpanzees (*Pan troglodytes*) at Mahale Mountains National Park, western Tanzania. Am J Primatol 70(8): 755-765.

3. Williams JM, Lonsdorf EV, Wilson ML (2008) Causes of death in the Kasekela chimpanzees of Gombe National Park, Tanzania. Am J Primatol 70(8): 766-777.

4. Köndgen S, Schenk S, Pauli G, Boesch C, Leendertz FH (2010) Noninvasive monitoring of respiratory viruses in wild chimpanzees. EcoHealth 7(3): 332-341.

5. Scully EJ, Basnet S, Wrangham RW (2018) Lethal respiratory disease associated with human rhinovirus C in wild chimpanzees, Uganda, 2013. Emerg Infect Dis 24(2): 267-274.

6. Emery Thompson M, Machanda ZP, Scully EJ (2018) Risk factors for respiratory illness in a community of wild chimpanzees (*Pan troglodytes schweinfurthii*). Royal Society Open Science 5(9): 180840.

7. Negrey JD, Reddy RB, Scully EJ (2019) Simultaneous outbreaks of respiratory disease in wild chimpanzees caused by distinct viruses of human origin. Emerg Microbes Infect 8(1): 139-149.

8. Patrono LV, Samuni L, Corman VM (2018) Human coronavirus OC43 outbreak in wild chimpanzees, Côte d'Ivoire, 2016. Emerging Microbes & Infections 7: 118.

9. Grützmacher KS, Köndgen S, Keil V (2018) Human respiratory syncytial virus and *Streptococcus pneumoniae* infection in wild bonobos. EcoHealth 15(2): 462-466.

10. Lonsdorf EV, Gillespie TR, Wolf TM (2018) Socioecological correlates of clinical signs in two communities of wild chimpanzees (*Pan troglodytes*) at Gombe National Park, Tanzania. Am J Primatol 80.

11. Dunay E, Apakupakul K, Leard S, Palmer JL, Deem SL (2018) Pathogen transmission from humans to great apes is a growing threat to primate conservation. EcoHealth 15(1): 148-162.

12. Gilardi KV, Gillespie TR, Leendertz FH (2015) Best Practice Guidelines for Health Monitoring and Disease Control in Great Ape Populations. Gland, Switzerland: IUCN SSC Primate Specialist Group.

13. Unwin S, Chatterton J, Chantrey J (2013) Management of severe respiratory tract disease caused by human respiratory syncytial virus and *Streptococcus pneumoniae* in captive chimpanzees (*Pan troglodytes*). J Zoo Wildlife Med 44(1): 105-112.

14. Negrey JD, Mitani JC, Wrangham RW (2022) Viruses associated with ill health in wild chimpanzees. Am J Primatol 84(2).

15. Negrey JD, Angedakin S, Reddy RB, Sandel AA, Jombela S, et al. (2025) Decreases in chimpanzee respiratory disease signs and enteric viral quantity following implementation of anthroponotic disease prevention protocols at a long-term research site. Biological Conservation 308: 111225.

Copyright© Suresh Antony J.

YOUR RESEARCH DESERVES GLOBAL VISIBILITY

• Publish • Discover • Share • Connect

MEDICAL	CLINICAL	LIFESCIENCES	PHARMA
90+ OPEN ACCESS JOURNALS		400+ RESEARCH TOPICS	

SUBMIT YOUR RESEARCH
Submit your manuscript through our online submission system.

REPRINTS
If you require a reprint of any article, please contact us.
info@medwinpublishers.com

BECOME A REVIEWER
Share your expertise and contribute to scholarly publishing.

SCAN TO EXPLORE MEDWIN JOURNALS

• Journals • Submit Manuscript • Reviewer Program • Article Tracking

Our Journals indexed in

Embase® ELSEVIER | PubMed National Library of Medicine NLM | Crossref | Google Scholar | INDEX COPERNICUS INTERNATIONAL | ISSN INTERNATIONAL STANDARD SERIAL NUMBER INTERNATIONAL CENTRE | ROAD DIRECTORY OF OPEN ACCESS SCHOLARLY RESOURCES

EuroPub | Scilit | WorldCat® | Academic Resource Index ResearchBib | ADVANCED SCIENCE INDEX | INTERNATIONAL Scientific Indexing | ICMJE INTERNATIONAL COMMITTEE of MEDICAL JOURNAL EDITORS

This article published by **Medwin Publishers,** 28475 Witherspoon Dr, Novi, MI 48377, United States, +1-248-247-6042, WhatsApp +1 (248) 275-1133, info@medwinpublishers.com

© 2026, by the Authors. The articles published from this journal are distributed to the public under "**Creative Commons Attribution License**" (https://creativecommons.org/licenses/by/4.0/). Therefore, upon proper citation of the original work, all the articles can be used without any restriction or can be distributed in any medium in any form.