IJBPAS, March, 2024, 13(3): 1091-1114

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)
'A Bridge Between Laboratory and Reader'

www.ijbpas.com

A REVIEW ON VIRUS OUTBREAK AFFECTING HUMAN ERA DURING 2000-2022

VASANTHA G[1*], RAVI KIRAN D[2], SIRISHA K[3]

Vignan Institute of Pharmaceutical Technology, Vishakapatnam, India

***Corresponding Author: Dr. Galanki Vasantha: E Mail: drvasanthaniper@gmail.com**

Received 15th Feb. 2023; Revised 25th April 2023; Accepted 14th July 2023; Available online 1st March 2024

https://doi.org/10.31032/IJBPAS/2024/13.3.7835

INTRODUCTION

Viruses are infectious microbes that have genome of DNA or RNA that is enveloped by protein coat. They have small obligate intracellular parasites. These are categorized upon their shape, size, chemical composition, structure of its genome and its replication mode. They have been categorized mainly upon its structure, composition of its chemicals and its replication process. Viruses which cause infections in humans were now clustered into 21 families **[1]**. Virus cannot replicate on its own hence it infects the host cell to make copies. The 21 century has been with a lot of viral pandemics that affected millions of individuals **[2]**. The viruses which are emerged from certain species of animals then to the mammals and finally spreads to the humans which leads to disease. Initially the virus, mainly depends on its ability to spread the virus within the human beings can cause an individual to get infected and can lead to few sporadic cases, which results in local endemic and can lead to a larger epidemic or world pandemic. These pandemic events from the past two centuries are huge. These include the viral infections that were not encountered earlier, such as SARS and MERS **[3]**. The 21st century has major epidemics, which includes pandemics, that are by previously emerged viruses such as cholera, plague and yellow fever, as well as emerging diseases such as SARS, Ebola, Zika, MERS, HIV, influenza A and COVID **[4]**.

Table: Viruses details							
VIRUS	**FAMILY**	**VIRUS SIZE**	**VIRUS SHAPE**	**ORIGIN OF REPLICATION**	**GENOME**	**TRANSMISSION**	**SYMPTOMS**
Dengue fever	**Flaviviridae**	**40- 60 nm**	**Spherical**	**B cells**	**Single stranded - RNA**	**-Animals -Insect bites -Stings**	**-Sudden high fever,Headache,Swollen lymph glands, Skin rash.**
Junin Virus	**Arenaviridae**	**40-200nm**	**T-shape**	**Cytosol**	**Bi-Segmented negative stranded-RNA**	**-Infected rodents Blood, saliva, urine, feces -Aerosolized**	**-Photophobia -Retro orbital pain,Malaise,Petechiae,Epigastric pain**
Lassa Virus	**Arenaviridae**	**70- 150 nm**	**T-shape**	**Cytoplasm**	**Bi-segmented Single stranded - RNA**	**-Ingestion -Inhalation -Infected rodents urine, feces**	**-Fever, General weakness,Malaise ,Sore throat Chest pain**
Bird Flu Virus	**Orthomyxoviridae**	**80- 120 nm**	**Roughly spherical**	**Nucleus**	**Eight segmented Single- stranded RNA genome**	**Infected birds saliva, mucous, feces**	**-Muscle pains -Fever,Runny nose,Shortness of breath,Cough,Headache**
Ebola virus	**Filoviridae**	**14000- 80 nm.**	**Thread like shape changed to circular or filamentous**	**Cytoplasm**	**Negative stranded RNA**	**-Infected blood, body fluids of wild animals and humans**	**-Abdominal pain,Chest pain,Muscle pains,Chills -Dehydration,Vomiting blood,Red eyes,Sore throat,Red spots on skin -Coughing up blood**
SARS Cov	**Coronaviridae**	**20- 500 nm**	**Roughly spherical or Ellipsoidal shape**	**Upper respiratory epithelia**	**Linear RNA**	**-Aerosolic -Direct contact with infected person**	**-Muscle pains -Shortness of breathe,Chills,Fever,Headache,Cough,Malaise,Respiratory distress**
Mers Cov	**Coronaviridae**	**30.1 kb**	**Roughly spherical or Ellipsoidal shape**	**Airway epithelial cell (lungs)**	**Single stranded positive-sense RNA**	**-Infected dromedary camels -Infected humans direct contact**	**-Fever,Cough -Shortness of breathe -Pneumonia -Chills**
Monkey Pox	**Poxviridae**	**190 kb**	**Oval**	**Cytoplasm**	**Double- stranded DNA**	**-Direct contact with contaminated objects -From person to person -From animal to animal**	**-Swollen lymph nodes -Headache,Chills, Fever,Muscle pains ,Nasal congestion,Cough ,Sore throat**

Nipha	**Paramyxoviridae**	**120- 500 nm**	**Helical**	**Epithelial surfaces of respiratory route**	**Single stranded negative sense RNA**	**-Direct contact with infected animals like bats, pigs -Contaminated food products with infected animals -Infected people secretions and excretions**	**-Severe headache -Unconsciousness -Fever -Twitching of facial muscles -Nausea -Encephalitis -Vomiting**
Zika	**Flaviviridae**	**50 nm**	**Icosahedral Tiles**	**Fetal brains and placenta**	**Positive sense single stranded RNA**	**- Bite of an infected mosquito of Aedes species -From infected mother to baby -Sexual transmission**	**-Fever -Vomiting -Rashes -Conjunctivitis -Headache -Joint pain -Fatigue**

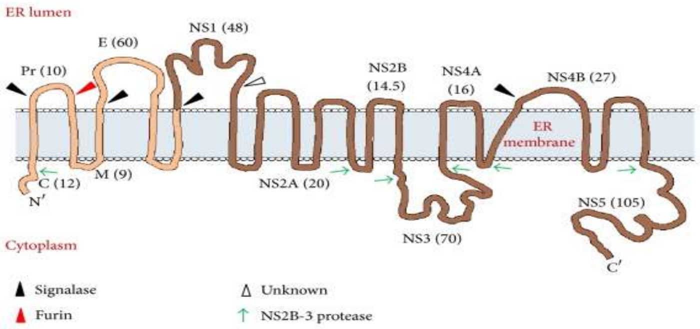
DENGUE

INTRODUCTION:

According to the data, around three billion of people, who are in urban areas of tropical and subtropical are considered for getting Dengue **[5]**. For more than 200 years sporadic dengue fever was known to the world, but the cause for these epidemics of dengue is not known clearly **[6]**. This is a mosquito born infection mainly transmits by a female mosquito known as Aedes mosquito. This infection is mostly seen in regions of both tropical and subtropical **[7]**. Other species of mosquitoes such as Albopictus, Polynesiensis, Scutellaris complex, and Niveus of Aedes family were also the vectors for transmitting this viral infection **[8]**. This virus comes under the family of Flaviviridae.

CHARACTERISTICS:

The genome of this virus is a positive sense RNA with 11kb length. These viruses are about 40 to 60 nm in length. The genome of this virus is then converted into a polyprotein and it codes 3 protein structuresand they are named Capsid, Pre-membrane and Envelope. It also encodes for 7 nonstructural proteins **[9]**.

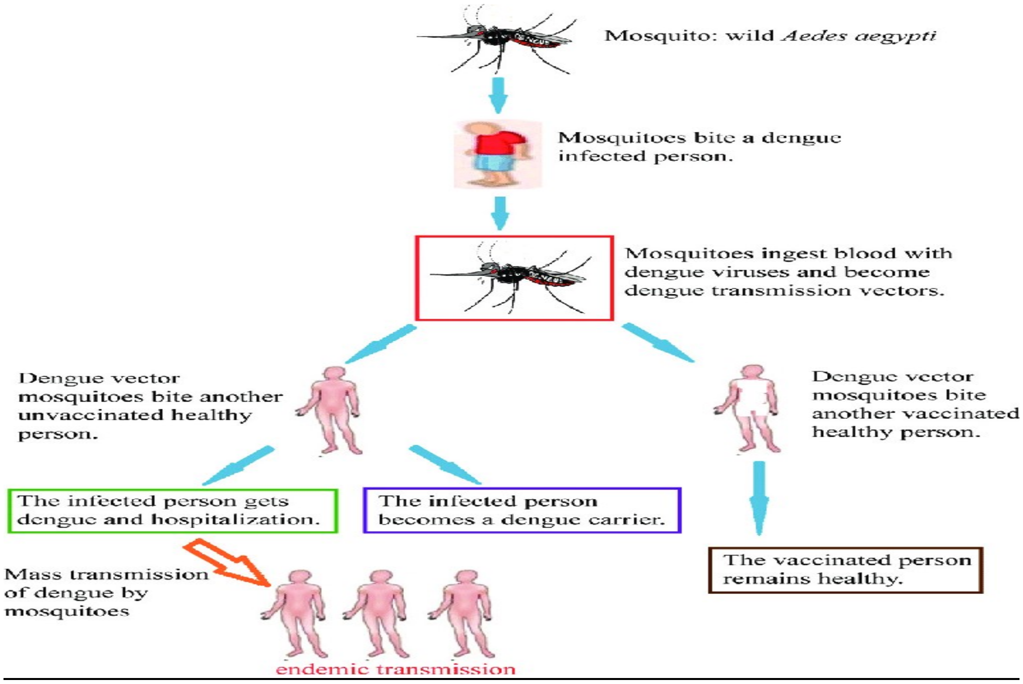


https://encrypted-tbn0.gstatic.com/images?q=tbn:ANd9GcR0kEE0MZn8o-WSF9tqEnVSmes9Y5zI24EXMA&usqp=CAU

The above image is about the organization and membrane of dengue virus. These Non structural proteins are mainly processed in the cytoplasm with NS2B and NS3. NS2A, 2B, 4A, 4B belongs to the proteins of transmembrane. These are present in the endoplasmic reticulum.

TRANSMISSION:

Mainly the transmission of this virus is by mosquito bites. The infected mosquito with the virus bites a healthy person and transmits the virus. This virus can also transmits among the two individuals in which one of the persons will act as a carrier to transmit the virus to a healthy individual.



https://www.researchgate.net/publication/318776357/figure/fig1/AS:521795427987456@1501417132286/Dengue-transmission-Aedes-mosquitoes-A-aegypti-or-A-albopictus-bite-a.png

SYMPTOMS:

Mostly the persons who are infected with this virus are asymptomatic in nature. Sometimes undifferentiated fever, Dengue fever are also seen **[10]**.

The common symptoms that are seen in the infected persons are abdominal pain **[11]**, myalgia, joint pains **[12]**, gastrointestinal tract hemorrhage **[13]**, Dengue shock syndrome **[14]** and Swollen lymph glands.

JUNIN VIRUS

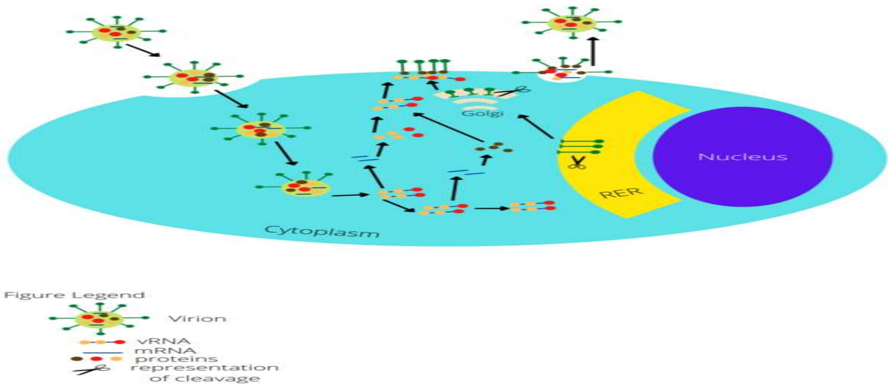
INTRODUCTION:

Argentina hemorrhagic fever was first identified in 1953, and several years later the isolation of this junin virus took place.

This virus is the causative agent for Argentine hemorrhagic fever. The disease that causes pandemic in the regions of Argentina. This fever leads to the risk of five million people's lives **[15]**. This disease primarily affected the agricultural workers in Argentina. Junín virus comes under the family of Arenaviridae. The studies have confirmed that the main hosts for this infection are musculinus and other rodents of family Calomys. TheC. rodents include C. laucha, A. azarae and O. flavescence **[16]**.

CHARACTERISTICS:

Genome of this virus is bi-segmented and contains a negative stranded RNA **[17]**. The RNA contains segments from 7.3kb to 3.5kb respectively. This virus woks with an ambisense coding strategy which encodes the two reading frames which are opened in opposite direction, and are divided by non-coding intergenic region which acts as a termination signal for the viral polymerase for its transcription process **[18]**.



https://microbewiki.kenyon.edu/images/thumb/8/80/JuninVirus.png/400px-JuninVirus.png

Visual representation of Junin Virus genome structure and life cycle. The virion of this virus first enters the host cell through the endocytosis process and then it releases the RNA. There are two stages of mRNA: early and late. Early mRNA goes through NP and LP translation while late mRNA goes through Z translation **[19]**.

TRANSMISSION:

This virus directly transmits by infected mucus and also with the contaminated objects with the infected virus. This virus can also be transmitted with an individual to individual but it is rare and also possible through infected persons excreta, blood, mucus secretions and also nosocomial infections are reported [20].

SYMPTOMS:

The main symptoms are fever, headache, anorexia, rashes on the soft palate, trunk and edema [21]. Other symptoms that are seen are dry cough, muscle aches, joint pains, severe malaise, sore throat and runny nose.

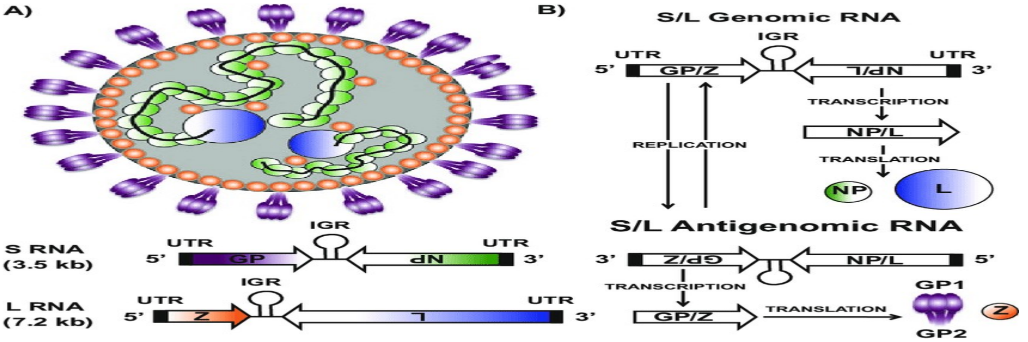
LASSA VIRUS

INTRODUCTION:

Lassa virus causes hemorrhagic fever. This virus mainly caused the pandemic to West Africa. This virus belongs to the Old World of arenavirus [22] and comes under the family Arenaviridae. Rodents plays as main hosts of this virus, which belongs to the genus of Mastomys and these are mainly seen in the sub-Saharan region of Africa [23]. The lassa virus caused severe global burden among all the viral hemorrhagic fevers (except dengue fever) [24].

CHARACTERISTICS:

Like other arenaviruses, the genome of this virus has two single-stranded RNA. The genome consists of two segments: ranging from 3.4kb to 7 kb in length [25].

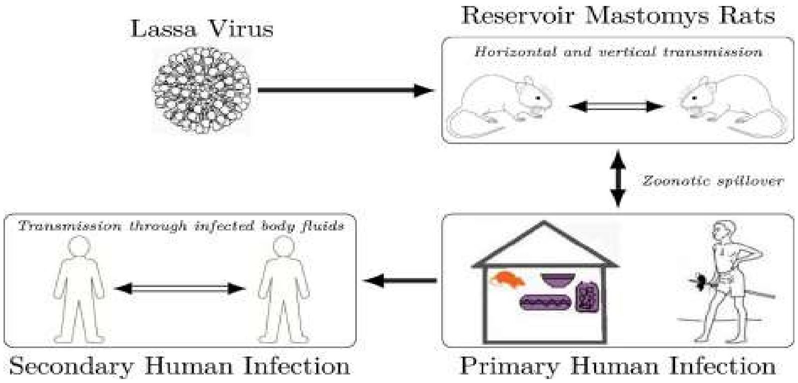


https://www.researchgate.net/publication/317177291/figure/fig18/AS:1017895203139600@1619696536666/Lassa-virion-structure-genome-replication-and-gene-transcription-a-Virion-structure.png

The above image is about the structure of virionits replication and transcription process [26].

TRANSMISSION:

The transmission to humans is by direct or indirect contact with the contaminated food items with the excreta of infected multimammate rats. The transmission is through respiratory and gastrointestinal tracts of humans. The person to person transmission is also seen [27].



https://ars.els-cdn.com/content/image/1-s2.0-S1468121821000225-gr1.jpg

SYMPTOMS:

The symptoms are seen after 2 to 21 days of exposure with the virus. Most of the people who are infected with this virus are asymptomatic in nature. The commonly seen symptoms are muscle aches, fever, nausea, facial swelling, sore throat, vomiting, nose bleeds, diarrhea, headache, chest pain, mouth bleeds and gastrointestinal hemorrhages.

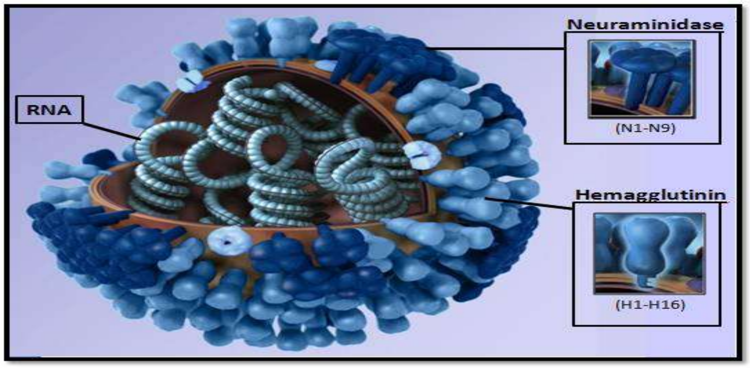
BIRD FLU

INTRODUCTION:

Bird flu is also called as Avian Flu. The pandemic has occurred in 29 countries of Europe in 2016 and in the year 2017. The largest poultry outbreaks and wild birds are dead in the European region and this has been the largest ever recorded **[28]**. Avian influenza viruses are the causative agents for the bird flu infection. In the year 1997 the 1st emergence of this infection in humans was observed in the region of Hong Kong **[29]**. The main reservoirs for this virus are the wild birds that are migratory waterfowl in nature.

CHARACTERISTICS:

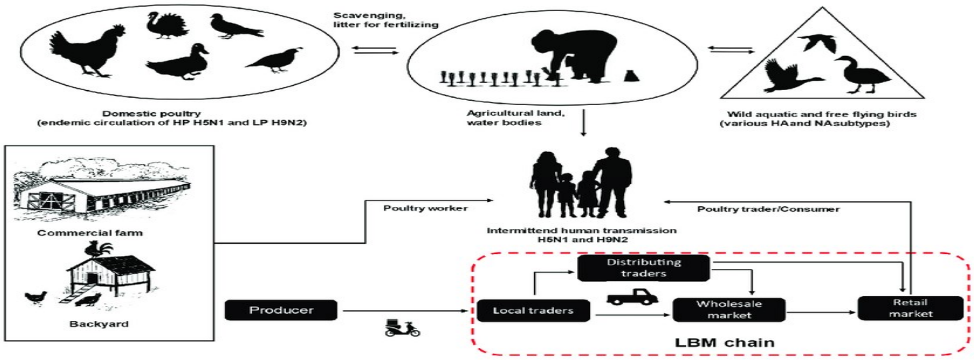
These are the influenza A viruses and the genome of this virus isa single stranded negative sense RNA with eight segments in it. These viruses have surface proteins of H1–16 and N1–9 subtypes **[30]**.



https://campus.extension.org/file.php/423/moodle_pics/AIstructureCDC.jpg

TRANSMISSION:

These viruses are mainly discovered in live bird markets and the poultries thrughout the year. It means that this virus circulates in the poultry birds all year and it leads to the infections in humans **[31]**. The transmission can be both in direct and indirect form. The transmission is by consuming the poultry meat that was infected with virus and person to person transmission can also be seen.



https://www.researchgate.net/publication/342877973/figure/fig1/AS:912432105013249@1594552174884/An-overview-of-avian-influenza-virus-transmission-and-live-bird-market-trading-chains.png

SYMPTOMS:

The symptoms that are seen in the people infected with bird flu are as follows: high temperature, dry cough, chesty cough, sore throat, headache, tiredness, joint pains, diarrhea, blocked nose, limb pain, stomach upset, runny nose, sneezes, loss of appetite, shortness of breath, insomnia, malaise and muscle aches.

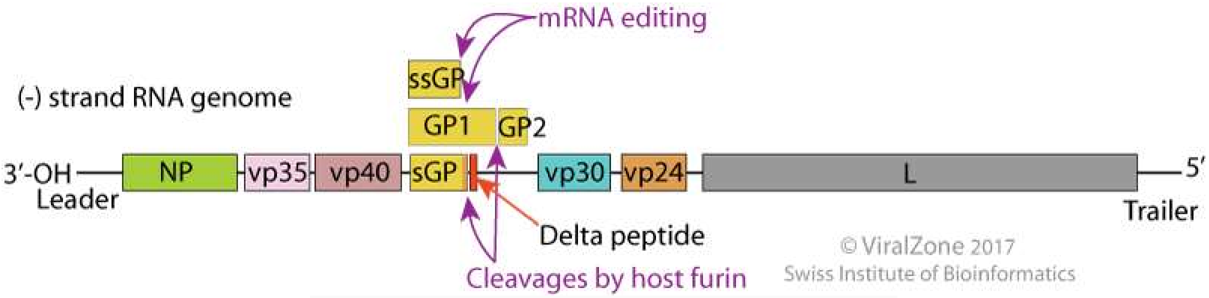
EBOLA

INTRODUCTION:

Ebola virus caused several pandemics from the past 38 years. In the year 2014, the Ebola virus led to the pandemic that was spreaded from Africa to other continents **[32]**. Among all viral haemorrhagic fevers, Ebola is highly pathogenic in nature. Mortality is up to 90%. It is mainly due to multi-organ failure and severe bleeding. Ebola comes under Filoviridae family with Bundibugyo and Sudan. Ebola viruses are also associated with disease outbreaks in humans **[33]**.

CHARACTERISTICS:

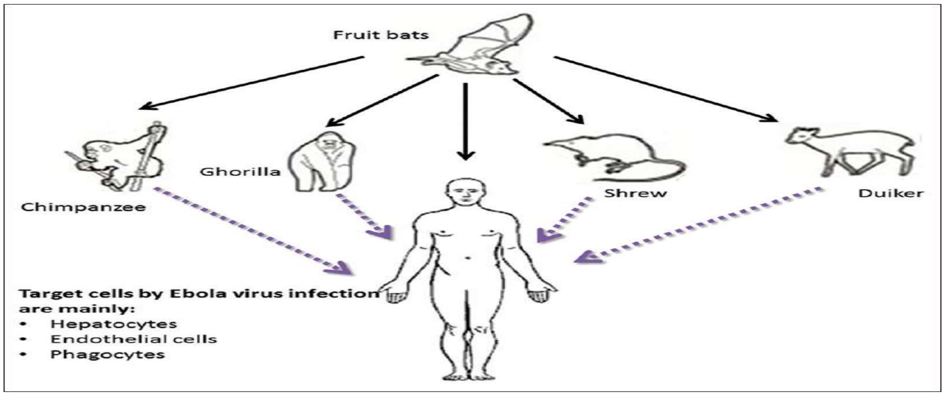
The genome is ofsingle stranded negative-sense RNA. It encodes seven viral proteins that include nucleoprotein, glycoprotein, polymerase, VP24, VP30, VP35, VP40. The genome of this virus is 19-kb **[34]**.



https://viralzone.expasy.org/resources/Filovirus_genome.png

TRANSMISSION:

Transmission to humans are by close contact with wild bats or by manipulating contaminated wildlife. Direct transmission in between two persons is also possible by infected persons blood, excreta, mucous secretions or from contaminated surfaces with the virus **[35]**.



https://www.jmsjournal.net/articles/2016/21/1/images/JResMedSci_2016_21_1_84_192500_f3.jpg

SYMPTOMS:

Symptoms that include fever, lack of appetite, hiccups, sore throat, red eyes, breathing difficulty, headache, swallowing difficulty, chest ache, muscle pains, internal bleeds, joint pains, muscle weakness, stomach pain, vomiting, nausea, rashes on skin and diarrhea.

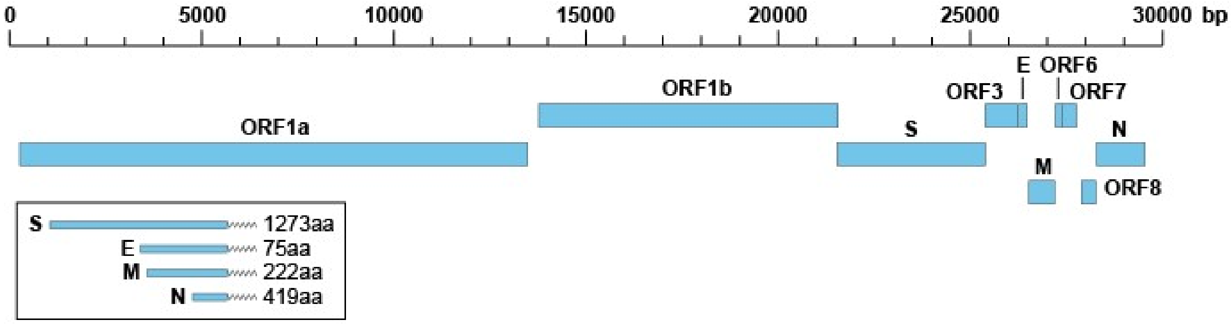
SARS COV

INTRODUCTION:

The 21st century people have experienced this huge pandemic in their lives which totally changed the way of their living. The pandemic was caused by the coronaviruses, that are SAR Cov of year 2003, later it is called as SARS CoV 2 in Dec 2019 **[36]**. This SARS-CoV-2 is a highly contagious and severe pathogenic coronavirus. Later the Chinese scientists have identified that the causative agent of pandemic disease is a betacoronavirus **[37]**. Till 2nd Dec 2020 1.4 million deaths and 64 million cases are reported across the world **[38]**.

CHARACTERISTICS:

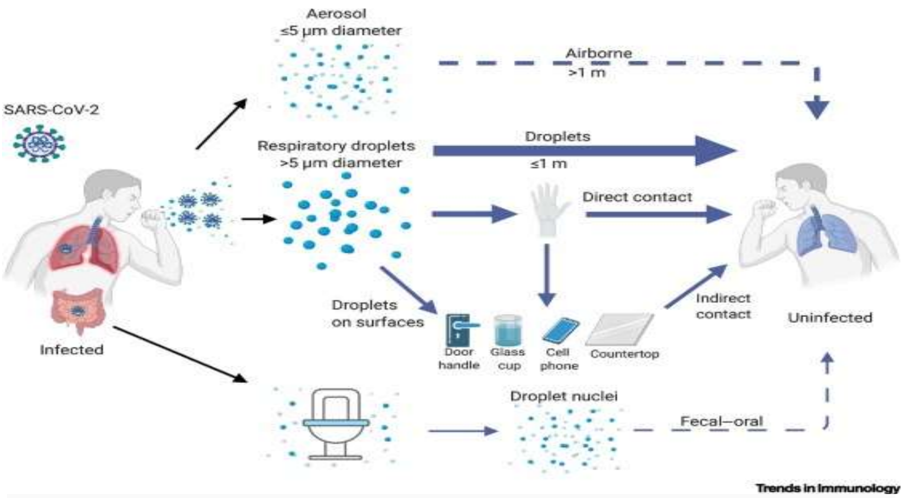
The genomic of this virus is 29.8 kb to 29.9 kb. The genomic structure is with specific gene characteristics of known corona viruses. The 5' genome contains orf1ab which encodes orf1a polyproteins. When it comes to the 3′ it encodes proteins that are structural in nature, that include Surface, Envelope, Membrane, and Nucleocapsid proteins. This virus contains approximately 6 accessory proteins that encodes by ORF-3a, ORF-6, ORF-7a, ORF-7b, and ORF-8 genes **[39]**.



https://images.novusbio.com/design/dw-nb-sars-cov-2-genome.png

TRANSMISSION:

These virus particles are mainly transmitted by droplet mode of transmission. And it is the most widely pronounced and heavily implicated mode of transmission that was reported during the pandemic situations. The transmission is by direct contact, the virus spreads from one infected person to the other person very rapidly. Human to human transmission can be seen more mainly in households with close interactions among the family members **[40]**.



https://www.cell.com/cms/attachment/212ae577-5665-40a3-97dc-ac8e99cde858/gr2_lrg.jpg

SYMPTOMS:

The symptoms of this virus are like the flu symptoms. The most commonly seen symptoms in the patients who are infected with this virus are headache, anosmia, cough, ageusia, sore throat, cold, fever, feeling unwell and dyspnea. The symptoms are seen worse in the patients who have some secondary diseases like asthma, eczema, OCD, anxiety, depression or previous ebola virus infection.

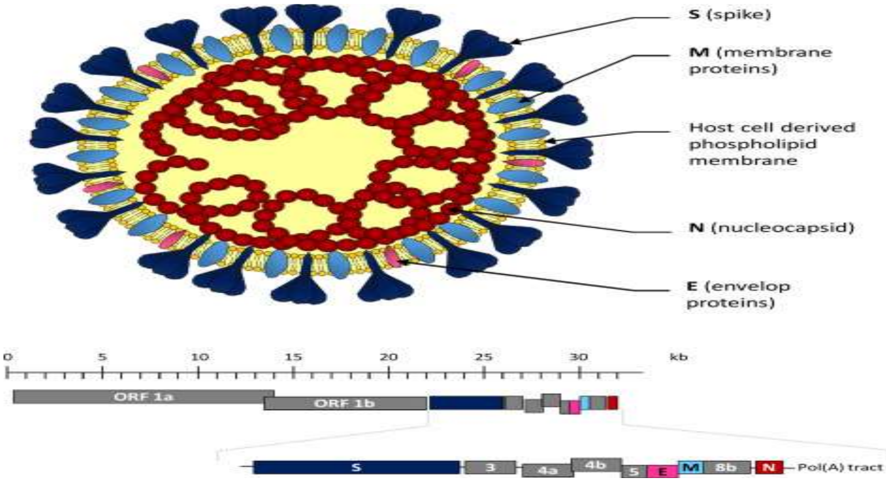
MERS COV

INTRODUCTION:

This is also a type of coronavirus and was first reported in 2012 by Saudi Arabia. This was called as the MERS CoV. This virus is responsible for acute human respiratory syndrome. This virus is mainly transmitted by animals and person to person transmission is also possible. The research explains that this virus was mainly associates with the bats as they are the reservoirs, although this is not confirmed **[41]**. According to the WHO reports 2,040 laboratory-confirmed cases, 712 fatalities are seen from 27 countries across the globe **[42]**.

CHARACTERISTICS:

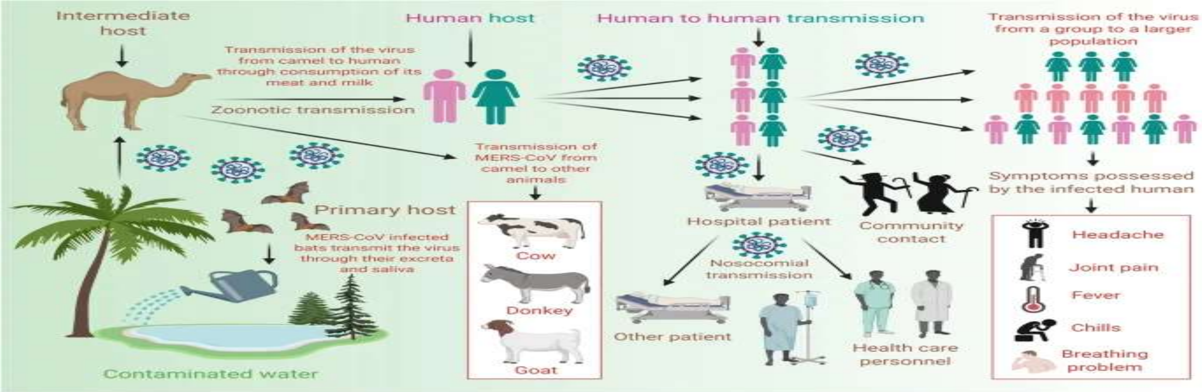
The genome of MERS-CoV is a large RNA. The length of this genome is approximately 26-33 kb **[43]**. This virion is a single stranded RNA of Coronaviridae family. Totally 4 coronavirus genus described as alpha, beta, gamma and delta **[44]**.



https://ars.els-cdn.com/content/image/1-s2.0-S0399077X19310546-gr3.jpg

TRANSMISSION:

Transmission of this virus to humans from animals is very rare and the evidence is limited **[45]**. And direct Human-to-human transmission is possible mainly by two major factors that are family and healthcare.



https://media.springernature.com/lw685/springer-static/image/art%3A10.1208%2Fs12249-021-02062-2/MediaObjects/12249_2021_2062_Fig2_HTML.png?as=webp

SYMPTOMS:

The most commonly seen symptoms of MERS CoV include high fever, cough, vomiting, dyspnea, nausea, diarrhea, pneumonia and renal failure are also seen in severely infected patients.

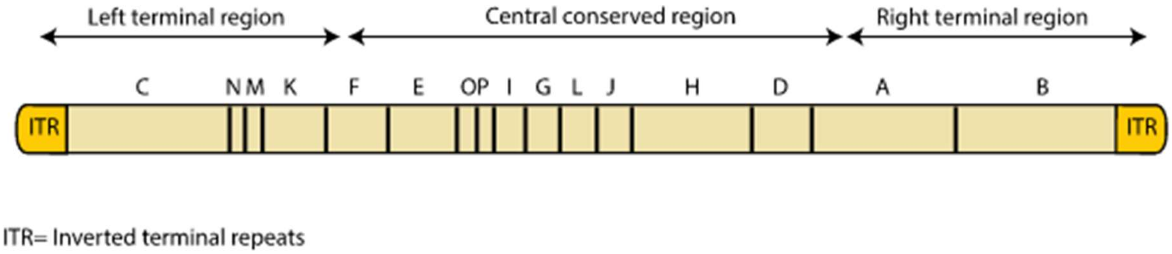
MONKEYPOX

INTRODUCTION:

This is an Orthopox virus which is sameas Smallpox. Wide number of monkeypox infections to humans occurred in Central Africa. This virus is transmitted accidentally to humans when they are in contact with the infected animals **[46]**. This virus was first isolated in 1958 in Copenhagen during the pandemics of a smallpox like disease which occurred in a group of cynomolgus monkeys. In between the years 1960 and 1968 several outbreaks of monkeypox virus were reported in the United States and the Netherlands monkeys. The first monkeypox infection in humans was reported in the year 1970 **[47]**.

CHARACTERISTICS:

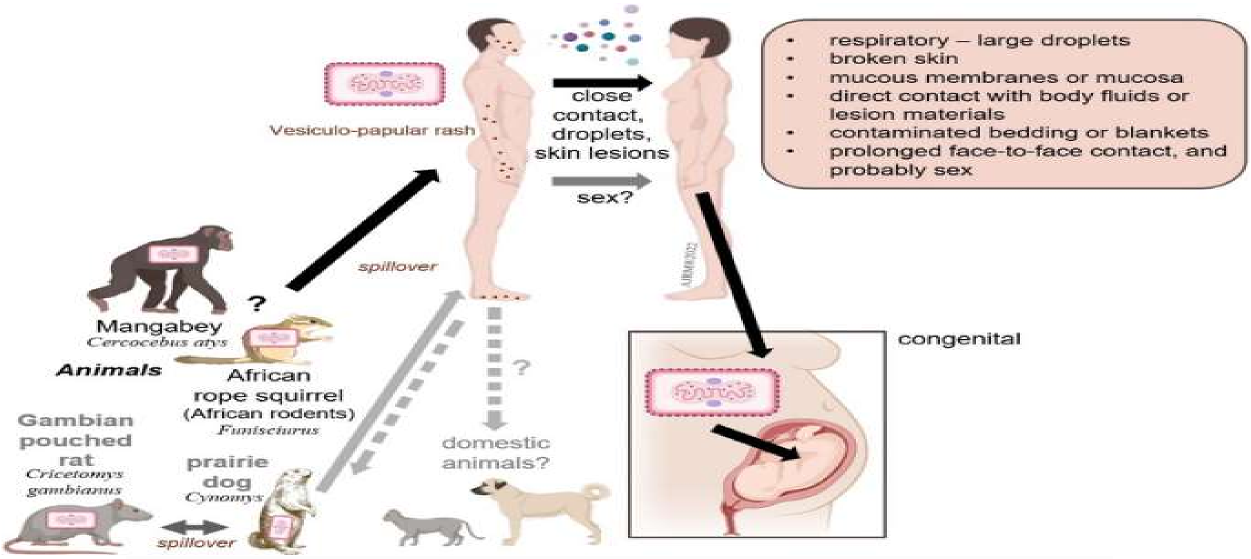
This virus comes under Poxviridae family. Genome of monkey pox has double-stranded DNA and it resembles as brick-like particles which ranges from 200–250 nm in diameter. This can be seen by an electron microscope in magnification of 10000X.



https://viralzone.expasy.org/resources/Poxvirus_genome.svg

TRANSMISSION:

This virus is primarily transmitted to humans by infected animals mainly by monkeys. Transmission is also possible through the direct contact with the infected persons. It can also be transmitted by mucous secretions of nose and by the clothes which are contaminated with virus particles. Present epidemiological data says that high frequency of human-to-human transmission can cause further outbreaks mainly in men who have sexual contact with men **[48]**.



https://media.springernature.com/lw685/springer-static/image/art%3A10.1186%2Fs12941-022-00518-2/MediaObjects/12941_2022_518_Fig1_HTML.png

SYMPTOMS:

The symptoms of monkeypox virus include muscle pain, back ache, fatigue, rashes on face, swollen lymph nodes , palms, fever, hands, feet, groin, headache, genital parts and in anal regions. The rashes are flat in nature and they are filled with liquid and these can also be found in the mouth, throat, and on the eyes too. The rashes on the entire body can range up to thousands.

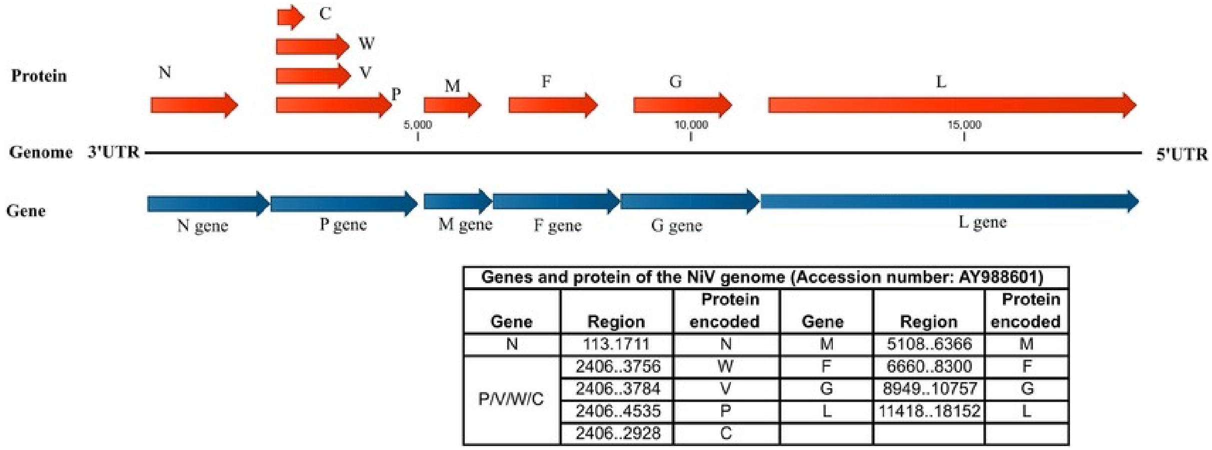
NIPHA

INTRODUCTION:

This virus leads to severe febrile encephalitis which results in mortality of 40- 75 percent of infected cases. The primary natural reservoir for this Nipah virus is fruit bats. At first this virus emerged in Malaysia in the year 1998 and caused human nervous and respiratory problems who are in contact with live pigs which are infected with nipah virus[49]. Nipah is a paramyxovirus related to Hendra virus. Since 1999 Malaysia has not had any cases, but outbreaks of nipah virus had continued to Bangladesh and India[50].

CHARACTERISTICS:

The virus is helical in shape. It is about 40-1900 nm. The genome is single stranded negative sense RNA **[51]**. Nipah virus was morphologically similar with other viruses of Paramyxoviridae family. Nipah virus has 6 genomes and that encodes for Fusion protein, Glycoprotein, Matrix, Nucleocapsid, Phosphoprotein and Polymerase. Nipha virus is very much related to Hendra virus. The genome of this virus has twelve nucleotides larger than Hendra virus **[52]**.



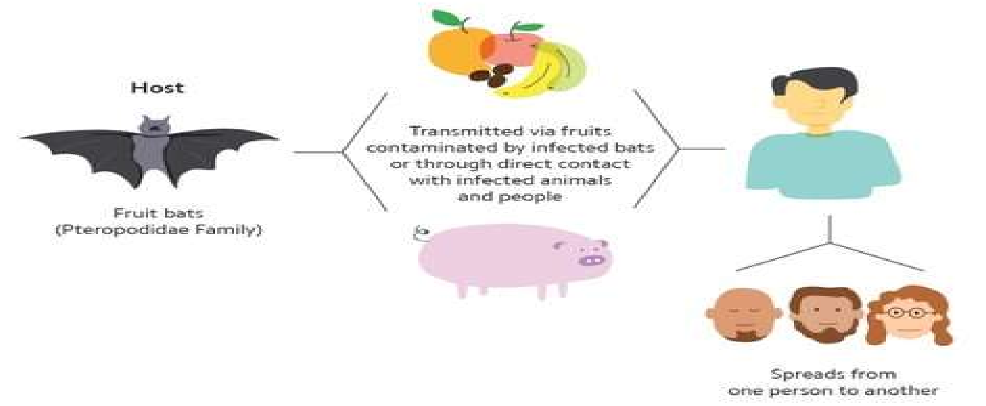
Genes and protein of the NiV genome (Accession number: AY988601)

Gene	Region	Protein encoded	Gene	Region	Protein encoded
N	113.1711	N	M	5108..6366	M
P/V/W/C	2406..3756	W	F	6660..8300	F
	2406..3784	V	G	8949..10757	G
	2406..4535	P	L	11418..18152	L
	2406..2928	C			

https://www.researchgate.net/publication/349165648/figure/fig1/AS:989835246444544@1613006521226/Nipah-virus-genome-Linear-diagrammatic-view-of-the-different-genes-present-on-the-NiV.png

TRANSMISSION:

In Bangladesh person to person direct virus transmission is commonly seen. Mainly the transmission takes place by direct contact with the infected people and their secretions. During patient care the transmission of this virus is very common **[53]**. The main transmission pathways in Bangladesh are via consumption of contaminated raw dates and palms by infected bats and also by the infected animals like cattle, pigs, and goats. Transmission is also by the food that was contaminated with infected bats saliva or by its excreta. Maximum one-half of patients in Bangladesh were infected by human to human transmission of the virus **[54]**.



https://www.practostatic.com/health-wiki/images/d1dc221a5b428f412212a5b7e1cb6680.jpg

SYMPTOMS:

Patients infected with this virus show the following symptoms: fever, headache, dizziness, vomiting, and severe encephalitis. More patients have shown reduced levels of alertness and brainstem dysfunction . Other symptoms that are shown are aseptic meningitis, diffuse encephalitis, and focal brainstem dysfunction. Cerebellar signs were common in the patients infected with this virus **[55]**.

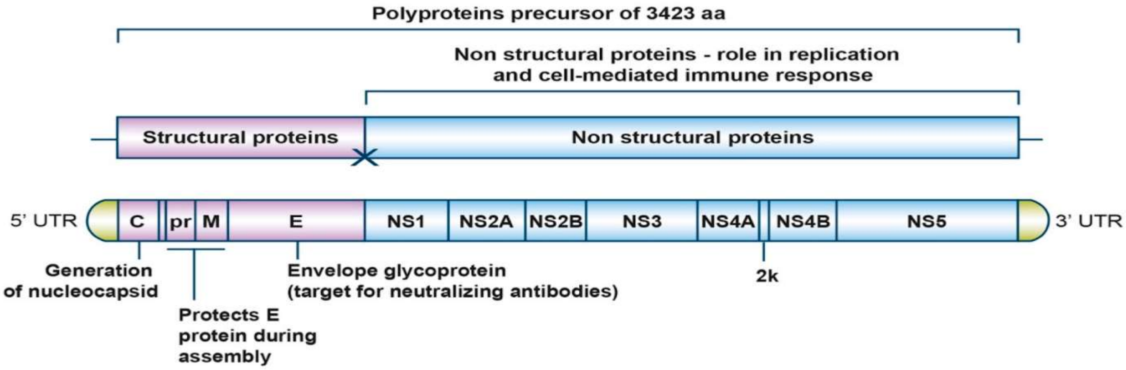
ZIKA

INTRODUCTION:

This is a flavivirus which belongs to Flaviviridae family. This virus was first isolated from A. Africanus mosquitoes in the year 1947 **[56]**. At the beginning there was no evidence that this virus can cause disease in humans. In 1947 Zika virus was initially isolated from nonhuman primates and in 1948 it was isolated from mosquitoes of Africa. This viral infection in humans was pandemic for half decade in Africa before its emergence in Pacific and Americas **[57]**. In 1954 the first three cases of human infection by Zika virus were identfied in Nigeria. The main pandemic of this virus was in 2007, with its endemic in western Pacific island called Yap island. During 2013 and 2014 a large pandemic was seen in French Polynesia and the south Pacific . In those areas the severe complications were noticed. The non vector borne transmission of this virus was also reported **[58]**. Sero Surveillance studies in humans say that this virus is widespread in Africa, Asia, and Oceania regions **[59]**.

CHARACTERISTICS:

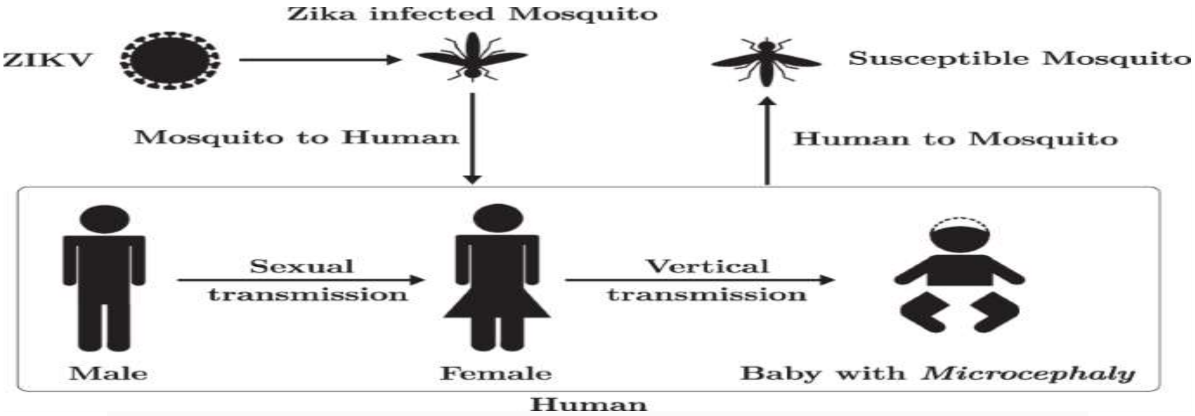
The genome is a single stranded positive sense RNA. Zika virus comes under the Flaviviridae family. Viruses of this flaviviridae causes severe morbidity across the world **[60]**. The genome is 10.8-kb and it contains ~100 nt 5′ untranslated region, an open reading frame of ~10 kb, and ~420 nt 3′ **[61]**. The unique character of this virus is high homologous recombination activity. This virus has undergone multiple recombination events during the years 1947 and 2007. Such an active recombination is not so common among these viruses and this serves as the main mechanism for Zika virus adaptation to the A. dalziel vectors **[62]**.



https://www.mdpi.com/pharmaceuticals/pharmaceuticals-12-00101/article_deploy/html/images/pharmaceuticals-12-00101-g001.png

TRANSMISSION:

Transmission is by two distinct transmission cycles such as ecological and phylogenetic. Most of these flaviviruses such as dengue fever, yellow fever are arboviruses and these has 2 distinct transmission cycles: evolutionarily and ecologically, a sylvatic transmission cycle, in which the virus circulates between the zoonotic vertebrate reservoir and the amplification hosts and arboreal mosquitoes, and an urban transmission cycle, where this virus circulates between the humans and the peridomestic Aedes Mosquitoes **[63]**. This virus is mainly transmitted to humans by the bite of infected mosquitoes. The transmission can also be possible from the mother to the child during pregnancy or by sexual contact, breastfeeding, or even by transfusion of blood with infected virus particles **[64]**. In African countries this zika virus twirls in a sylvatic transmission cycle involving nonhuman primates and forest-dwelling Aedes species mosquitoes, the elongation of transmission of virus to humans is not defined well due of the lack of surveillance as well as serologic cross-reactivity with other circulating flaviviruses **[65]**.



https://media.springernature.com/m685/springer-static/image/art%3A10.1038%2Fs41598-019-53062-z/MediaObjects/41598_2019_53062_Fig1_HTML.png

SYMPTOMS:

The most common symptoms were rashes, pruritus, arthralgia, headache, myalgia, fever, asthenia, and conjunctivitis **[66]**. The typical symptoms were seen after 2 to 12 days after mosquito bite. Other symptoms include joint pains, and clinical symptoms lasts for a week. Various symptoms like muscle ache , headache, stomach pain, nausea, diarrhea, mucosal ulcers, and pruritus are rarely observed. Guillain-Barré syndrome was also reported in some adults that were severely infected with this virus **[67]**.

REFERENCES

[1] Baron S, Medical microbiology, 4thEd, University of Texas Medical Branch, Galveston, 1996.

[2] Payne K, Kenny P, Scovell J. M, Khodamoradi K, Ramasamy R,Twenty-first century viral pandemics: a literature review of sexual transmission and fertility implications in men. Sexual medicine reviews, 8(4),2020,518-530.

[3] Grubaugh ND, Ladner JT, Lemey P, Pybus OG, Rambaut A, Holmes EC, Andersen KG,Tracking virus outbreaks in the twenty-first century,Nature microbiology, 4(1),2019, 10-19.

[4] Catherine Wei Min Ong, Giovanni Battista Migliori, European Respiratory Journal, 56(4), 2020, 2001727, DOI: 10.1183/13993003.01727-2020.

[5] Hamel R, Dejarnac O, Wichit S, Ekchariyawat P, Neyret A, Luplertlop N, Missé D,Biology of Zika virus infection in human skin cells, Journal of virology, 89(17), 2015, 8880-8896.

[6] Nimmannitya S. Dengue haemorrhagic fever: current issues and future research, Asian-Oceanian Journal of Paediatrics and Child Health, 1, 2002, 1-21.

[7] Chen R, Vasilakis N, Dengue—quo tu et quo vadis? , Viruses, 3(9), 2011, 1562-1608.

[8] Malavige GN, Fernando S, Fernando DJ, Seneviratne SL.Dengue viral infections, Postgraduate medical journal, 80(948), 2004, 588-601.

[9] Murphy BR, Whitehead SS,Immune response to dengue virus and prospects for a vaccine, Annual review of immunology, 29(1), 2011, 587-619.

[10] Ahmed FU, Mahmood CB, Sharma JD, Hoque SM, Zaman R, Hasan MS, Dengue and Dengue Haemorrhagic Fever in children during the 2000 outbreak in Chittagong, Bangladesh 25,2001, 33-39.

[11] Pancharoen C, Mekmullica J, Thisyakorn U, Primary dengue infection: what are the clinical distinctions from secondary infection?. The Southeast Asian journal of tropical medicine and public health, 32(3), 2001,476-480.

[12] Whitehorn J, Farrar J, Dengue,British medical bulletin, 95(1), 2010, 161-173.

[13] Kabra SK, Juneja R, Jain Y, Singhal T, Dar L, Kothari SS, Broor S, Myocardial dysfunction in children with dengue haemorrhagic fever. National Medical Journal of India, 11,1998, 59-61.

[14] Huy NT, Van Giang T, Thuy DHD, Kikuchi M, Hien TT, Zamora J, Hirayama K, Factors associated with dengue shock syndrome: a systematic review and meta-analysis, PLoS neglected tropical diseases, 7(9),2013, e2412.

[15] Parodi AS, Coto CE, Boxaca M, Lajmanovich S, González S, Characteristics of Junin virus. Etiological agent of Argentine hemorrhagic fever. Archiv fur die gesamte Virusforschung, 19(4),1966, 393-402.

[16] Briknarová K, Thomas CJ, York J, Nunberg JH, Structure of a zinc-binding domain in the Junin virus envelope glycoprotein. Journal of Biological Chemistry, 286(2),2011, 1528-1536.

[17] Buchmeier MJ, Arenaviridae: the viruses and their replication, Fields virology, 5th Ed, lipincott williams, philadelphia, 2007, 1792-1827.

[18] Tortorici MA, Albarino CG, Posik DM, Ghiringhelli PD, Lozano ME, Pomar RR, Romanowski V, Arenavirus nucleocapsid protein displays a transcriptional antitermination activity in vivo, Virus research, 73(1), 2001. 41-55.

[19] Grant A, Seregin A, Huang C, Kolokoltsova O, Brasier A, Peters C, Paessler S, Junin virus pathogenesis and virus replication, Viruses, 4(10),2012, 2317-2339.

[20] Fields BN, Fields' virology, 5thEd,vol 1, Lippincott Williams & Wilkins, philadelphia, 2007.

[21] Safronetz D, Feldmann H, Falzarano D, Arenaviruses and filoviruses: viral haemorrhagic fevers, In Medical microbiology, 18th Ed, Churchill Livingstone, London, 2012, 546-558.

[22] Schaeffer J, Carnec X, Reynard S, Mateo M, Picard C, Pietrosemoli N, Baize S, Lassa virus activates myeloid dendritic cells but suppresses their ability to stimulate T cells, PLoS pathogens, 14(11),2018, 1-25.

[23] Salazar-Bravo J, Ruedas LA, Yates TL, Mammalian reservoirs of arenaviruses. Arenaviruses I: Salazar-Bravo J, Ruedas LA, Yates TL, Mammalian reservoirs of arenaviruses. Arenaviruses I: the epidemiology, molecular and cell biology of arenaviruses, Current Topics in Microbiology and Immunology, 262,2002, 25-63.

[24] Falzarano D, Feldmann H, Vaccines for viral hemorrhagic fevers—progress and shortcomings, Current opinion in virology, 3(3), 2013, 343-351.

[25] Lukashevich IS, Stelmakh TA, Golubev VP, Stchesljenok EP, Lemeshko NN, Ribonucleic acids of Machupo and Lassa viruses, Archives of virology, 79, 1984, 189-203.

[26] Perez DR, Angel M, Gonzalez-Reiche AS, Santos J, Obadan A, Martinez-Sobrido L, Plasmid-based reverse genetics of influenza A virus. Reverse Genetics of RNA Viruses: Methods and Protocols, 1602, 2017,251-273.

[27] Ibrahim MA, Dénes A, A mathematical model for Lassa fever transmission dynamics in a seasonal environment with a view to the 2017–20 epidemic in Nigeria, Nonlinear Analysis: Real World Applications, 60, 2021, 1-21.

[28] ECDC E, European Food Safety Authority, European Centre for Disease Prevention and Control: the European Union Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Food-borne Outbreaks in 2014, EFSA J, 13(19),2015, 120-125.

[29] Mittal N, Medhi B, The bird flu: a new emerging pandemic threat and its pharmacological intervention, International journal of health sciences, 1(2),2007, 277-283.

[30] Criado MF, Leyson CM, Youk S, DeBlois S, Olivier T, Killian ML, Pantin-Jackwood MJ, The Pathobiology of H7N3 Low and

High Pathogenicity Avian Influenza Viruses from the United States Outbreak in 2020 Differs between Turkeys and Chickens, Viruses, 13(9), 2021, 2-17.

[31] Parvin R, Nooruzzaman M, Kabiraj CK, Begum JA, Chowdhury EH, Islam MR, Harder T, Controlling avian influenza virus in Bangladesh: Challenges and recommendations, Viruses, 12(7), 2020, 2-20.

[32] Murray MJ, Ebola virus disease: a review of its past and present,Anesthesia & Analgesia, 121(3),2015, 798-809.

[33] Centers for Disease Control and Prevention. Ebola Virus Disease Distribution Map: Cases of Ebola Virus Disease in Africa Since 1976.

[34] Sanchez A, Khan AS, Zaki SR, Nabel GJ, Ksiazek TG, Peters CJ, Filoviridae: Marburg and Ebola viruses, Fields virology, 4th ed, vol 2, Lippincott, Philadelphia, 2001, 1279-1304.

[35] Alfson KJ, Avena LE, Beadles MW, Staples H, Nunneley JW, Ticer A, Griffith A, Particle-to-PFU ratio of Ebola virus influences disease course and survival in cynomolgus macaques, Journal of virology, 89(13), 2015, 6773-6781.

[36] Tu YF, Chien CS, Yarmishyn AA, Lin YY, Luo YH, Lin YT Chiou SH, A review of SARS-CoV-2 and the ongoing clinical trials, International journal of molecular sciences, 21(7), 2020, 1-19.

[37] Zhou P, Yang XL, Wang XG, Hu B, Zhang L, Zhang W, Shi ZL, A pneumonia outbreak associated with a new coronavirus of probable bat origin, nature, 579(7798),2020, 270-273.

[38] Dong E, Du H, Gardner L, An interactive web-based dashboard to track COVID-19 in real time, Lancet Infect Dis, 20,2020,533–534.

[39] Li FLi W, Farzan M, Harrison SC, Structure of SARS coronavirus spike receptor-binding domain complexed with receptor, Science, 309(5742),2005, 1864-1868.

[40] Yu IT, Li Y, Wong TW, Tam W, Chan A T, Lee J H, Ho T, Evidence of airborne transmission of the severe acute respiratory syndrome virus, New England Journal of Medicine, 350(17), 2004,1731-1739.

[41] Ramadan N, Shaib H, Middle East respiratory syndrome coronavirus (MERS-CoV): A review, Germs, 9(1),2019, 35-42.

[42] Ramadan N, Shaib H, Middle East respiratory syndrome coronavirus (MERS-CoV): A review, Germs, 9(1),2019, 35-42

[43] Woo PC, Huang Y, Lau SK, Yuen KY, Coronavirus genomics and bioinformatics analysis, viruses, 2(8),2010, 1804-1820.

[44] Hilgenfeld R, Peiris M, From SARS to MERS: 10 years of research on highly pathogenic human coronaviruses, Antiviral research, 100(1),2013, 286-295.

[45] Memish ZA, Cotten M, Meyer B, Watson SJ, Alsahafi AJ, Al Rabeeah AA, Drosten C, Human infection with MERS coronavirus after exposure to infected camels, Saudi Arabia, 2013, Emerging infectious diseases, 20(6),2014, 1012-1015.

[46] Rizk JG, Lippi G, Henry BM, Forthal DN, Rizk Y, Prevention and treatment of monkeypox, Drugs, 82(9),2022, 957-963.

[47] Ladnyj ID, Ziegler P, Kima E, A human infection caused by monkeypox virus in Basankusu Territory, Democratic Republic of the Congo, Bulletin of the World Health Organization, 46(5),1972, 593-597.

[48] Huang Y, Mu L, Wang W, Monkeypox: epidemiology, pathogenesis, treatment and prevention, Signal Transduction and Targeted Therapy, 7(1),2022, 1-22.

[49] Epstein JH, Field HE, Luby S, Pulliam JR, Daszak P, Nipah virus: impact, origins, and causes of emergence, Current infectious disease reports, 8(1),2006, 59-65.

[50] Looi LM, Chua KB, Lessons from the Nipah virus outbreak in Malaysia, Malaysian Journal of Pathology, 29(2), 2007,63-67.

[51] Wang LF, Mackenzie JS, Broder CC, Henipaviruses, In: Knipe DM, Howley PM, eds. Fields Virology,6th Ed,Vol 1, Lippincott Williams & Wilkins, Philadelphia, 2013,1070-1085.

[52] Goldsmith CS, Whistler T, Rollin PE, Ksiazek TG, RotaPA, Bellini WJ, Zaki SR, Elucidation of Nipah virus morphogenesis and replication using ultrastructural and molecular approaches, Virus research, 92(1),2003, 89-98.

[53] Harcourt BH, Tamin A, Halpin K, Ksiazek TG, Rollin PE, Bellini WJ, Rota PA, Molecular characterization of the polymerase gene and genomic termini of Nipah virus, Virology, 287(1),2001, 192-201.

[54] Clayton BA, Nipah virus: transmission of a zoonotic paramyxovirus, Current opinion in virology, 22, 2017,97-104.

[55] Hughes JM, Wilson ME, Luby SP, Gurley ES, Hossain MJ, Transmission of human infection with Nipah virus, Clinical Infectious Diseases, 49(11),2009, 1743-1748.

[56] Dick GW, Kitchen SF, Haddow AJ, Zika virus (II). Pathogenicity and physical properties, Transactions of the royal society of tropical medicine and hygiene, 46(5),1952,521-534.

[57] Musso D, Gubler DJ, Zika virus: following the path of dengue and chikungunya?, The Lancet, 386(9990),2015, 243-244.

[58] Baud D, Gubler DJ, Schaub B, Lanteri MC, Musso D, An update on Zika virus infection, The Lancet, 390(10107),2017, 2099-2109.

[59] Macnamara FN, Zika virus: a report on three cases of human infection during an epidemic of jaundice in Nigeria, Transactions of the royal society of tropical medicine and hygiene, 48(2),1954, 139-145.

[60] Huang YJS, Higgs S, Horne KM, Vanlandingham DL, Flavivirus-mosquito interactions, Viruses, 6(11),2014, 4703-4730.

[61] Kuno G, Chang GJ, Full-length sequencing and genomic characterization of Bagaza, Kedougou, and Zika viruses, Archives of virology, 152,2007, 687-696.

[62] Faye O, Freire CC, Iamarino A, FayeO, de Oliveira JVC, Diallo M, Sall AA, Molecular evolution of Zika virus during its emergence in the 20th century, PLoS neglected tropical diseases, 8(1), 2014,1-10.

[63] Vasilakis N, Weaver SC, Flavivirus transmission focusing on Zika, Current opinion in virology, 22,2017, 30-35.

[64] Song BH, Yun SI, Woolley M, Lee YM, Zika virus: History, epidemiology, transmission, and clinical presentation, Journal of neuroimmunology, 308,2017, 50-64.

[65] Gregory CJ, Oduyebo T, Brault AC, Brooks JT, Chung KW, Hills S, Petersen LR, Modes of transmission of Zika virus, The Journal of infectious diseases, 216(10),2017, 875-883.

[66] Garcell HG, García FG, Nodal MR, Lozano AR, Díaz CRP, Valdés AG, Alvarez LG, Clinical relevance of Zika symptoms in the context of a Zika Dengue epidemic, Journal of infection and public health, 13(2),2020, 173-176.

[67] Garcell HG, García FG, Nodal MR, Lozano AR, Díaz CRP, Valdés AG, Alvarez LG, Clinical relevance of Zika symptoms in the context of a Zika Dengue epidemic. Journal of infection and public health, 13(2),2020, 173-176.