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ZOONOTIC DISEASES AT THE WILDLIFE-HUMAN INTERFACE OF SHARED PATHOGENS AND EMERGING HEALTH THREATS

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Abstract
This study is to investigate zoonotic diseases related to the interaction between humans and wildlife, with a focus especially on shared pathogens and emerging health hazards. This study investigates not only diseases that are transmitted directly, such as Rabies, Ebola Virus Disease, Leptospirosis, and Hantavirus Pulmonary Syndrome, but also diseases that are transmitted by mosquitoes, such as Eastern Equine Encephalitis, Western Equine Encephalitis, Japanese Encephalitis, Tick-borne Encephalitis, Omsk Hemorrhagic Fever, and Kyasanur Forest Disease, as well as diseases that are transmitted by birds, such as Parrot Fever and Avian influenza. A study is carried out on each disease, taking into consideration its historical background, transmission dynamics, vectors, vertebrate hosts, and outbreaks that are related to it. The research highlights the linked interaction that exists between humans, animals, and the environment.

Keywords: Diseases, Pathogens, Transmitted, Threats, Zoonotic

Introduction

Wildlife diseases can result in severe health complications and mortality in animals, hence exerting a substantial influence on the total population size of wildlife (1). Zoonoses are illnesses that may be transmitted from animals in the wild to people. In the previous century, the majority of newly identified infectious diseases originated from animals, particularly those capable of infecting both animals and people (2). The transfer of diseases from wildlife or domestic animals involves several methods of spillover, including direct transmission or transmission through a vector (3). Although certain diseases, such as the rabies virus, and Hantavirus Pulmonary Syndrome, have long been recognized as causing repeated spillover instances, different pathogens are occasionally identified after outbreaks occur (4). The infectious agents or parasites that are responsible for certain zoonotic illnesses may be acquired from wild animals either by direct bites, or through the bite of vectors such as mosquitoes, fleas, ticks, and mites that have previously eaten on an animal that is sick with the disease (5). Hunters, field assistants, Biologists and other persons who have direct contact with animals have a heightened susceptibility to getting these illnesses directly from animal hosts or their ectoparasites. Plague and leptospirosis have been contracted by the manipulation and removal of,

rabbits, rodents, and carnivores' skin (6). Humans often get illnesses such as Lyme disease, Rocky Mountain spotted fever, and tick fever when they have been in environments that are ideal for disease-carrying organisms and their hosts (7). “The One Health Initiative is based on the fundamental principle that the health of different entities, such as people, captive animals, wildlife, and their respective surroundings, is interrelated. The purpose and vision statements of the project reflect this connection. Within the framework of One Health, wildlife has importance for two primary rationales. The physical well-being of people, domesticated animals, and nature is closely intertwined due to the transfer of common illnesses (8). Pathogens may be transmitted among various communities, emphasizing the need for a comprehensive health strategy that takes into account the interconnectedness of different groups (9). Furthermore, the mental and emotional state of people may be impacted by their interpretation of animal well-being, namely via the connection between humans and animals. The emotional connection between humans and animals may be influential on human emotions and general well-being, therefore making the health of wildlife a significant factor.

"Wildlife diseases threats to public health: Transmission concerns"
Directly Transmitted Diseasez

1.1 Rabies

Rabies is a viral illness that leads to inflammation of the brain in humans and other animals. The rabies virus is classified as the type species of the *Lyssavirus* genus, which belongs to the family Rhabdoviridae and the order Mononegavirales (10, 11). The transmission occurs by the act of an infected animal biting or scratching a person or another animal. Dogs are the most widespread mammal worldwide. In countries where dogs often get the disease, over 99% of rabies cases may be directly attributed to dog bites. The most common kind of rabies infection that occurs in people in the Americas is transmitted via bat bites, whereas canines are responsible for less than 5% of cases. Rabies is a very uncommon infection in rodents (12).

Transmission

The transmission of the rabies virus occurs by direct contact, specifically via damaged skin, nose, or mouth, with saliva or brain/nervous system tissue from an infected animal (13). Rabies is often transmitted to humans by the bite of an animal infected with the rabies virus. Non-bite exposures, such as scratches, abrasions, or open wounds that come into contact with saliva or other infectious material from a rabid animal, may also lead to rabies transmission, but these occurrences are few. Engaging in activities such as stroking a rabid animal or coming into contact with the blood, urine, or feces of a rabid animal does not provide a risk of infection and is not recognized to be a cause for worry for rabies transmission (14). Alternative means of transmission, apart from bites and scratches, are infrequent. Exposure to rabies virus by inhalation of aerosolized particles is a possible non-bite route, however, it is unlikely for the general population to come into contact with such aerosols, save for those working in laboratories. Instances of rabies transmission by corneal and solid organ transplants have been documented, however they are very uncommon (15). Since 2008, there have been just two documented cases of solid organ donors with rabies in the United States. Several organ procurement organizations have included a screening inquiry on potential exposure to rabies into their protocols for assessing the eligibility of each donor.

Outbreaks

The mortality rate due to rabies declined from 54,000 in 1990 to around 26,000 in 2010 (16). The majority of deaths were mostly centered in the regions of Asia and Africa. India had the largest number of cases in 2015, totaling around 20,847 China with approximately 6,000 and the Congo with 5,600 (17). Rabies causes around 59,000 deaths worldwide each year, with children under the age of 15 making up more than 40% of these fatalities (18). Over 95% of deaths caused by rabies are centered in Asia and Africa.

Symptoms

Symptoms of rabies might extend for many days. Subsequently, the symptoms advance to brain dysfunction, anxiety, confusion, and agitation. As the condition advances, individuals may encounter delirium, atypical conduct, hydrophobia, and sleeplessness. Providing treatment within 10 days of exposure may effectively avoid the onset of the condition.

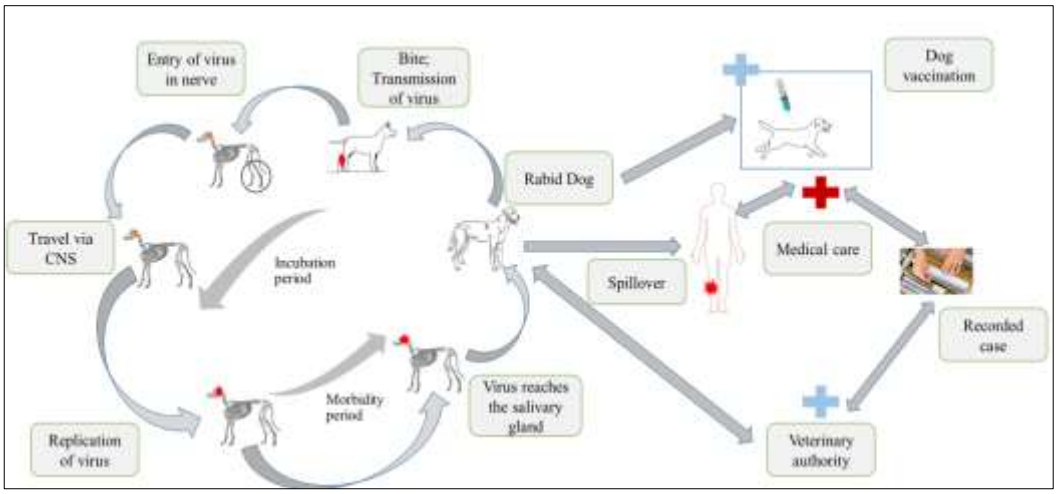

Figure 1: Image shows the pattern of transfer rabies virus from Dog to Human

1.2 Ebola virus disease (EVD)

Ebola virus disease (EVD) and Ebola hemorrhagic fever (EHF) are viral hemorrhagic fevers that affect humans and other primates (19). These fevers are caused by *ebolaviruses*. The first discovery of Ebola occurred in 1976 (20), during two concurrent instances of the illness. One epidemic took place in Nzara, a town located in South Sudan, while the other occurred in Yambuku, a community near the Ebola River in the Democratic Republic of the Congo, from whence the disease derived its name.

Transmission

An *ebolavirus* is first transmitted to humans via contact with an infected animal, such as a fruit bat or monkey. This occurrence is sometimes referred to as a spillover incident. Subsequently, the virus disseminates by interpersonal transmission, possibly impacting a significant number of individuals (21). Ebolaviruses are transmitted by direct contact with blood or bodily fluids (such as urine, saliva, perspiration, feces, vomit, breast milk, amniotic fluid, and semen) of an individual who is afflicted with or has succumbed to Ebola illness. This contact may occur via damaged skin or mucous membranes in the eyes, nose, or mouth. Items, such as garments, linens, syringes, and medical apparatus, have been tainted with bodily fluids from an individual afflicted with or deceased from Ebola illness. Fruit bats or nonhuman primates (such as apes and monkeys) that have been infected (22).

Outbreaks

From 1976 to 2012, the World Health Organization recorded 24 instances of Ebola outbreaks, which led to a cumulative total of 2,387 reported cases and 1,590 fatalities. The most extensive Ebola outbreak on record occurred in West Africa between December 2013 and January 2016, resulting in 28,646 infections and 11,323 fatalities (23). On March 29, 2016, it was officially announced that the situation was no longer considered an emergency. Additional outbreaks in Africa started in the Democratic Republic of the Congo in May of both 2017 and 2018. The World Health Organization has designated the Congo Ebola epidemic as a global health emergency in July 2019.

Symptoms
The main indications and manifestations of Ebola illness include fever, aches, and pains, such as intense headache and muscle and joint pain, as well as weakness and weariness. Pharyngitis, Anorexia, Manifestations of gastrointestinal distress including stomach discomfort, loose bowel movements, and emesis.

Treatment
There are two medications used for Ebola Virus Disease (EVD) which are appropriate for individuals of all ages, including both adults and children. In October 2020, Inmaze, a medication composed of a combination of three monoclonal antibodies, received its first approval. Ebanga, the second drug, is a monotherapy monoclonal antibody that obtained regulatory clearance in December 2020.

1.3 Leptospirosis
Leptospirosis is a bacterial infection caused by the pathogen *Leptospira* (24). Leptospirosis may be acquired by the entry of animal urine-contaminated water or soil into the nasal passages, oral cavity, ocular region, or through an open wound (25). Leptospirosis may induce influenza-like symptoms that can escalate into Weil's syndrome, a potentially fatal condition, in a minority of individuals.

Transmission
Leptospira primarily inhabits animals. Nevertheless, reptiles and ectothermic creatures such as frogs, snakes, turtles, and toads are susceptible to the illness. The existence of reservoirs of human infection is uncertain (26). Rats, mice, and moles serve as significant main hosts, although other species such as rabbits, deer, opossums, dogs, sheep, hedgehogs, pigs, cows, skunks, and raccoons may also act as carriers of the illness. Several animal species in Africa have been identified as carriers, including the shrews, banded mongoose, Rusa deer, and Egyptian foxes (27). Animals may infect each other via a variety of techniques. Dogs may acquire the urine of an infected animal by licking it off the grass or dirt, or by drinking from a contaminated puddle (28). Leptospirosis has been transmitted to domestic dogs who are confined to the home, seemingly as a result of licking the urine of mice that are afflicted with the disease. Leptospirosis may also be spread via the seminal fluid of affected animals.

Outbreaks
Approximately one million cases of leptospirosis, which is marked by its severity, are reported each year, resulting in 58,900 fatalities (29). Severe instances are 5-15% of the total number of leptospirosis patients. Leptospirosis is present in urban and rural locations in tropical, subtropical, and temperate zones. The quantification of the worldwide impact of leptospirosis on public health may be accomplished by assessing the disability-adjusted life year. The incidence rate is 42 per 100,000 individuals annually, surpassing that of other illnesses like filariasis, and rabies (30).

Symptoms
Symptoms may vary from asymptomatic to moderate (such as headaches, muscular pains, and fevers) to severe (such as pulmonary hemorrhage or meningitis). Weil's illness, the acute and severe manifestation of leptospirosis, induces jaundice (yellowing of the skin and eyes), renal failure, and hemorrhaging in the sick person.

Treatment
Doxycycline, an antibiotic, effectively prevents leptospirosis infection. vaccinations for humans have limited efficacy, but vaccinations for other species are more readily accessible.

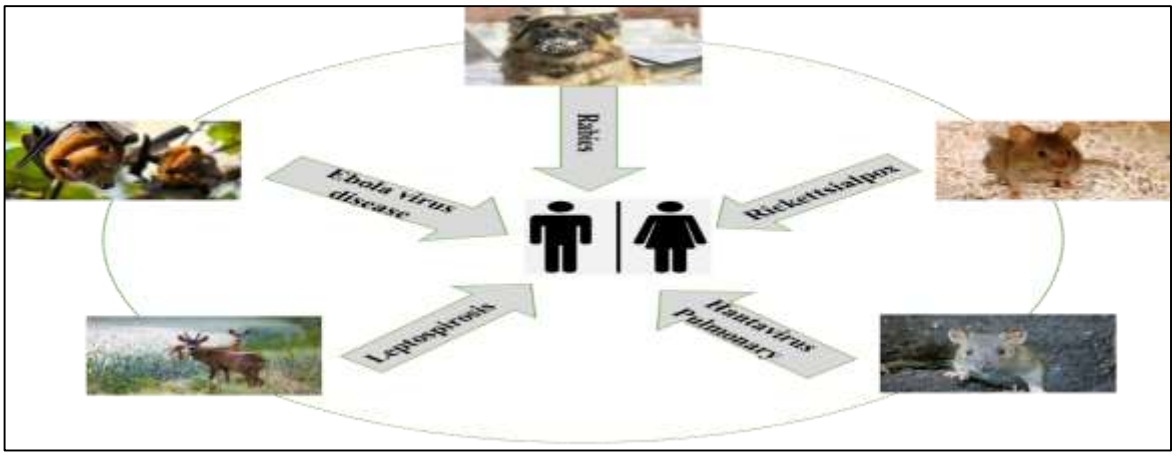

Figure 2: Image shows the pattern of direct transmitted diseases into Human

1.4 Hantavirus Pulmonary Syndrome

Two potentially lethal syndromes of zoonotic origin are produced by species of hantavirus. One of these diseases is known as hantavirus pulmonary syndrome (HPS) (31). Among them are the New York ortho-hantavirus (NYV), the Monongahela virus (MGLV), the Black Creek Canal virus (BCCV), the Sin Nombre ortho-hantavirus (SNV), and a few additional members of the hantavirus genera that are indigenous to the United States of America and Canada (32). Some rodents serve as the primary hosts for the hantaviruses. One of these rodents is the hispid cotton rat in southern Florida. This particular rodent is the primary host for the Black Creek Canal virus. Sin Nombre virus is mostly transmitted by the deer mouse, which is found in Canada and the western United States. The New York virus is most often transmitted by the white-footed mouse, which is found in the eastern United States. The long-tailed mouse, along with other species belonging to the genus *Oligoryzomys*, has been identified as the reservoir for the Andes virus in South America.

Transmission

Humans may get the virus by direct exposure to the virus via a bite or inhalation of viral particles that have become airborne (33). These particles are released from the feces, urine, or saliva of a mouse that serves as the natural host for the virus (34). There is no evidence to suggest that hantaviruses, whether in the pulmonary or hemorrhagic forms, may be transmitted by droplets or contaminated objects (fomites).

Outbreaks

Since the initiation of monitoring in 1993, a total of 850 cases of hantavirus illness have been documented in the United States in 2021 (35).

Symptoms

The symptoms include fever, cough, myalgia, cephalalgia, lethargy, dyspnea, nausea, emesis, and diarrhea. During the subsequent 5-7 days of the cardiopulmonary phase, the patient experiences a fast decline in their state, leading to acute respiratory failure. In 2017, the death rate among patients in the US due to HPS was 36%. Hantavirus Pulmonary Syndrome (HPS) currently lacks either curative treatment or preventive vaccination.

1.5 Rickettsialpox

Rickettsialpox is an innocuous and nonlethal illness that has a resemblance to chicken pox (36). The condition is attributed to a microorganism called *Rickettsia akari*, which is spread from house mice to humans by the bite of an infected house mouse mite (*Liponyssoides sanguineus*) (37). Rickettsialpox has been documented in many cities in this nation, including Boston, West Hartford, New York, Cleveland, and Philadelphia.

1.6 Rat-bite fever (RBF)

Rat-bite fever (RBF) is a sudden and feverish sickness in humans caused by germs that are spread by rats, usually by their urine or mucous secretions, and transferred to humans (38). Rat-bite fever is also known as streptobacillary fever, *streptobacillosis*, spirillary fever, bogger, and epidemic arthritic erythema (39). Rat-bite fever (RBF) is an uncommon illness transmitted by infected rats and caused by two distinct species of bacteria: *Streptobacillus moniliformis*, the only documented bacterium responsible for RBF in North America (known as streptobacillary RBF), and *Spirillum minus*, prevalent in Asia. The majority of instances are concentrated in Japan, however distinct variations of the illness may also be seen in the United States, Europe, Australia, and Africa. Certain instances are identified after patients being exposed to the urine or body fluids of an infected animal. The mouse may secrete these substances from its mouth, nose, or eyes. Most instances are caused by the animal's bite. Additionally, it may be spread by consumption of food or water that has been contaminated with rat excrement or urine. Additional species susceptible to this illness include mustelids, gerbils, and rodents. Household pets, such as dogs or cats, may transmit the illness to people if they come into contact with these animals.

Table 1: Direct transmitted wildlife diseases that affects the Human

Disease	Pathogen	Hosts	Geographical distribution	Impact on wildlife	Impact on Human health
Rabies	*Lyssavirus* or rabies virus	Dogs, foxes, bats, Human	Asia, Africa, United States	Multiple populations in extinction danger	Inflammation of the brain in humans
Ebola virus disease	*Ebolavirus*	Fruit bats or nonhuman primates (apes and monkeys)	Africa, Democratic Republic of the Congo, Uganda	25% of chimpanzees died in 1990s. Cause multiple infections in primates	11,323 deaths and 28,646 illnesses in West Africa due to this disease.
Leptospirosis	*Leptospira*	Rabbits, deer, opossums, dogs, sheep, hedgehogs	Southeast Asia, Africa, United States	Cause infection in horses, sheep, dogs, and hedgehogs.	Every year, there are 500,000 cases of leptospirosis in humans globally.
Hantavirus Pulmonary Syndrome	Hantavirus	Rodents	America, Canada, southern Florida, Canada, eastern United States	Cause the mild infections in wild animals.	850 cases of hantavirus in US in 2021. In 2017, the mortality rate in the US due to HPS was 36%.
Rickettsialpox	*Rickettsia akari*	Rodents like house mice	Boston, West Hartford, New York	Cause the infection in rodents.	Cause the serious infections and mild febrile illness in humans
Rat-bite fever	*Streptobacillus moniliformis, Spirillum minus*	Rats, gerbils	US, Europe, Australia, and Africa	Cause the infection in wild animals.	Zoonotic sickness and acute relapsing fever

Birds- Borne Diseases

1.7 Psittacosis (Parrot Fever)

Psittacosis, often known as parrot fever or ornithosis, is a zoonotic infectious disease in humans caused by the bacterium *Chlamydia psittaci*. The infection may be transmitted by birds that are affected, including parrots such as cockatiels, budgerigars, and macaws, among others (40). The illness may also be transmitted by other species such as sparrows, gulls, ducks, pigeons, hens, and numerous avian species. The incidence of infection is believed to be lower in canaries and finches as compared to psittacine birds.

Transmission
Psittacosis, caused by the *Chlamydia psittaci* bacteria, may spread by direct contact between the mouth and beak, or by inhaling airborne particles of dried feces or feather dust from sick birds (41). Transmission between individuals is feasible, however infrequent.

Outbreaks
Between 2002 and 2009, the Centers for Disease Control and Prevention received reports of 66 instances of psittacosis in humans (42).

Symptoms
Firstly, in psittacosis, individuals have symptoms that closely resemble those of typhoid fever, including elevated body temperatures, arthralgia, gastrointestinal distress, conjunctivitis, epistaxis, and leukopenia. Horder's spots, also known as rose spots, may manifest at this phase. Antibiotics are used to treat parrot fever. Tetracycline and doxycycline are both efficacious medications for treating this condition.Avian influenza
Avian influenza is a viral infection caused by the virus which is known as influenza A that has the ability to infect humans (43). Influenza A virus can cause zoonotic diseases, mostly in birds, there are three kinds of influenza A, B, and C. Avian influenza viruses contain several subtypes, but only specific strains from five subtypes, namely, H7N7, H7N3, H7N9, H9N2, and H5N1 have been seen to infect humans (44). An old lady in Jiangxi Province, China, succumbed to pneumonia caused by the H10N8 strain in December 2013. She was the first human casualty officially attributed to that variant. Influenza A, although mostly suited to birds, is also capable of efficiently adapting and maintaining transmission between humans. The latest influenza study on the genetic makeup of the flu virus (Spanish) reveals that it has genes that have been modified from both human and avian strains (45). Pigs may get infected with influenza viruses that affect humans, birds, and other pigs, which allows for the mixing of genetic material and the creation of a new and unique virus. This can lead to an antigenic shift, resulting in the emergence of a new subtype of influenza A virus that is not well recognized by the immune systems of most individuals. Avian influenza strains are categorized according to their pathogenicity, specifically into two primary types: high pathogenicity (HP) and low pathogenicity (LP). H5N1 is the most generally known strain among highly pathogenic avian influenza (HPAI) variants. The first identification of H5N1 took place in a farmed goose in Guangdong Province, China, in 1996 (46). Furthermore, milder variations of the H5N1 strain have been recorded in North America. Pigeons may be infected with avian strains, but they seldom become sick and are not very effective in spreading the virus to people or other animals.

Transmission
Birds that are sick and those that are not are the main carriers of avian influenza, while contaminated equipment may also serve as an indirect vector. The virus is present in the excretions emanating from the nasal passages, oral cavity, and ocular region of avian hosts, including their fecal matter. The transmission of HPAI infection to humans often occurs via direct contact with infected chickens, particularly during activities like slaughter or plucking (47). While the virus may be transmitted by airborne secretions, it is important to note that the illness is not categorized as a disease spread through the air. Pathogens rapidly disseminate in groups of birds and can decimate an entire flock over 28 hours (48); Conversely, the less aggressive strain of the disease may affect the laying of eggs, but it is far less deadly. While it is feasible for people to acquire the virus that cause avian influenza from birds, transmitting the virus from one person to another is much more challenging without extended interaction.

Outbreaks
The first documentation of human infections occurred in 1997 in Hong Kong. Since 2003, the World Health Organization (WHO) has received reports of over 700 cases of Asian HPAI H5N1 in humans (49). These cases have predominantly originated from 15 countries throughout Asia, Africa, the

Pacific, Europe, and the Middle East. However, the impact of this virus has been seen in over 60 nations. From early 2013 to early 2017, the World Health Organization (WHO) received reports of 916 laboratory-confirmed cases of H7N9 in humans (50). On January 9, 2017, China's National Health and Family Planning Commission informed the World Health Organization about 106 instances of H7N9 reported from November to December. These cases resulted in 35 deaths, including 2 possible cases of transmission from person to person.

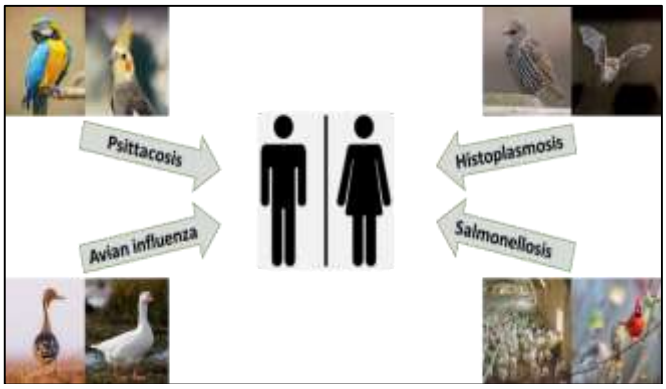

Figure 3: Image shows the pattern of transfer diseases into human

1.8 Histoplasmosis

AIDS patients often have histoplasmosis due to their compromised immune system. Immunocompetent persons who have been previously infected have a degree of protection against negative consequences if they are infected again. *H. capsulatum* thrives on soil and substances that have been tainted with bats or bird excrement (51). The fungus has been detected in bat habitats, caves, and bird roosts like starlings. Soil disturbance caused by excavation or building may release pathogens which are breathed and deposited in the lungs (52). The United States had 105 recorded cases from 1938 to 2013. From 1978 to 1979, almost 100,000 people in Indianapolis were recorded to have fungal exposure (53, 54). The affected individuals experienced a range of medical conditions including pericarditis, rheumatological syndromes, ulcers in the esophagus and vocal cords, interstitial nephritis, fibrosing mediastinitis, epididymitis, and intestinal lymphangiectasia. This disease closely resembles common respiratory illnesses such as colds, pneumonia, and influenza, and may be transmitted by bat droppings.

1.9 Salmonellosis

Salmonellosis is a bacterial illness caused by *Salmonella*, which resides in the gastrointestinal tracts of several animals, including birds (55). *Salmonella* is often spread to humans by the consumption of food that has been contaminated with animal excrement. Salmonella outbreaks associated with animals include a variety of species, including pet turtles, lizards, chickens, hedgehogs, and guinea pigs (56).

Outbreaks

Salmonellosis is a prevalent worldwide cause of diarrhea. In 2015, there were 90,300 fatalities attributed to nontyphoidal salmonellosis and 178,000 fatalities caused by typhoidal salmonellosis (57). Annually, around 1.35 million instances of non-typhoidal salmonellosis and 450 fatalities are reported in the United States.

Symptoms

The predominant symptoms seen in humans are diarrhea, fever, stomach cramps, and vomiting. The symptoms usually manifest within a timeframe of 12 to 36 hours after being exposed, and last for a duration of two to seven days. Antibiotics like ceftriaxone may destroy bacteria, although they are seldom needed.

Table 2: Birds-Borne diseases that affects the Human

Disease	Pathogen	Hosts	Geographical distribution	Impact on wildlife	Impact on Human health
Psittacosis (Parrot Fever)	*Chlamydia psittaci*	Parrots, cockatiels, and budgerigars	Worldwide	Affect the dynamics of local ecosystems and wildlife communities.	Cause of serious lung infection and Spleen enlargement.
Avian influenza	Influenza viruses (A, B, and C).	Birds,	Asia, Europe, Africa, and North America.	The virus primarily affects birds, and wild birds, especially waterfowl.	Cause severe respiratory illness in humans, with a high mortality rate.
Histoplasmosis	*H. capsulatum*	Poultry house, harboring bats, and birds.	North America, Central and South America, Africa, Asia.	In susceptible species, histoplasmosis can cause respiratory problems and other health issues.	Multiple infections are reported of Histoplasmosis.
Salmonellosis	*Salmonella*	Birds, turtles, lizards, hedgehogs and pigs	United States, Europe	Wildlife species may carry Salmonella which cause illness, such as diarrhea, lethargy, and weight loss.	Causes illnesses such as diarrhea, abdominal cramps, nausea, vomiting, and fever.

Mosquito-borne diseases

2 Indirectly Transmitted Diseases

2.1 Eastern equine encephalitis (EEE) disease

Eastern equine encephalitis (EEE), often known as Triple E or sleeping illness, is a zoonotic disease caused by a Togavirus that is transmitted by mosquitoes (58). The first documented case of Eastern Equine Encephalitis (EEE) occurred in Massachusetts, United States, in 1831 when 75 horses succumbed to an unexplained viral infection affecting the brain (59). Outbreaks of disease in horses have consistently happened regularly in the United States. The virus was obtained from the brain of a horse in Delaware (USA) during an equestrian pandemic in 1933. Subsequent evidence demonstrated that the Eastern equine encephalitis virus (EEEV) was responsible for widespread outbreaks in horses throughout the eastern coast of the United States in both 1831 and 1845 (60). The first documented instances of human infection were recorded in 1938 when a total of 30 children in the Northeastern United States succumbed to encephalitis (61). These incidences occurred simultaneously with epidemics in horses in the same locations. The human death rate is at 33%.

Transmission

The Eastern equine encephalitis (EEE) virus is sustained by a continuous process involving *Culiseta melanura* mosquitoes and bird hosts in freshwater hardwood swamps (62). Transmission to humans necessitates the involvement of an intermediary mosquito species that acts as a conduit between infected birds and uninfected animals, such as humans or horses. The majority of bridge species belong to the *Aedes*, *Coquillettidia*, and *Culex* genera (63). The transmission of EEE virus during organ donation has been reported, with one organ donor infecting 3 organ transplant recipients. Horses are very prone to the EEE virus infection, and a significant number of cases result in fatality.

Outbreaks

Since 2004, the virus has been more active in a number of the United States. At least 10 human cases of EEE were documented in Massachusetts between 2004 and 2006. New Hampshire also reported

several human cases. In September 2019, there was a noticeable spike in cases in Michigan and New England, leading several health officials to declare an epidemic (64). As of October 31, 2019, there have been five deaths in Michigan, three in Connecticut, one in Rhode Island, one in Alabama, one in Indiana, and three in Massachusetts. In addition, the virus was discovered in horses, deer, goats, and turkeys. There were five confirmed human cases between Massachusetts and Wisconsin as of September 9, 2020. There has been one death in Wisconsin and one in Michigan as of October 9, 2020 (65).

Vectors: The main mosquito vector is *Culiseta melanura*, an ornithophile; other significant vectors include *Culex erraticus, Uranotaenia sapphirina* (which feeds on reptiles and amphibians), *Aedes sollicitans, Coquillettidia perturbans* (which feeds on both mammals and birds), *Culex pedroi* in Peru, and *Aedes taeniorhynchus* and *Cx. Taeniopus* in Brazil.

Vertebrate hosts: Birds, amphibians, rodents, and reptiles in South America.

Symptoms

The illness starts with symptoms such as fever, loss of appetite, and abdominal pain. Subsequently, the virus targets the brain (encephalitis) and spinal cord (myelitis), which is accompanied by atypical behavior (such as stumbling, lack of coordination, and a propensity to wander in circles), drowsiness, paralysis, and seizures, ultimately culminating in fatality. There is currently no treatment for EEE.

2.2 Western equine encephalitis (WEE)

The virus was first obtained from the brain of a horse with encephalomyelitis in California in 1930 (66). The WEE virus is an arbovirus belonging to the family Togaviridae, which is transmitted by mosquitoes of the *Culex* and *Culiseta* genera (67). WEE is a hybrid virus formed by combining two different alphaviruses: a parent virus similar to Sindbis virus and a parent virus similar to the Eastern equine encephalitis virus (68). Since 1964, the number of confirmed cases in the U.S. has remained below 700. The envelope of this virus's genome consists of glycoproteins and nucleic acids. According to the CDC, this virus is found globally and is more common in regions near swamps with sparse human populations. WEE is often an asymptomatic illness, with symptomatic cases being rare. Nevertheless, the illness may result in severe complications in newborns and young individuals. Compared to Eastern equine encephalitis, Western equine encephalitis has a relatively low fatality rate (about 4%) and is mostly linked to infections in older individuals (69, 70). Roughly 15–20% of horses who get the virus will die or be euthanized. Currently, there is no available human vaccination for WEE and no approved treatment medications in the United States to treat this illness. The virus affects the central nervous system of the infected host, specifically targeting the brain and spinal cord.

Vectors: The mosquitoes *Culex tarsalis* (main carrier), *Culiseta inornata*, *Aedes melanimon*, and *Aedes vexans* are the primary vectors, sometimes seen in birds during the rainy summer months.

Vertebrate hosts: Jackrabbit *Lepus californicus,* Ground squirrel *Citellus richardsoni*, snakes probably also frog, and wild birds.

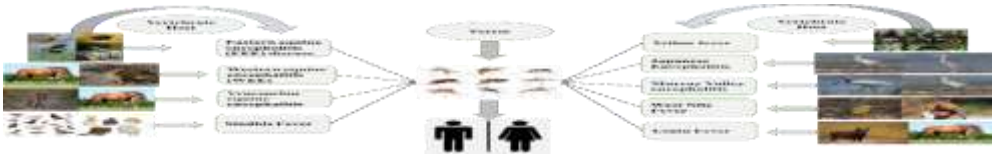

Figure 4: Image shows the pattern of transfer wildlife diseases into human through the Vectors

2.3 Venezuelan equine encephalitis

Venezuelan horse encephalitis virus is a viral disease transmitted by mosquitoes that leads to the development of Venezuelan equine encephalitis or encephalomyelitis (VEE) (71). The virus was first

obtained from the equine brain during an outbreak in Venezuelan Guajira in 1938 (72). Venezuelan Equine Encephalitis (VEE) may impact all members of the equine family, including, zebras, donkeys, and horses. Following infection, equines may have abrupt mortality or exhibit gradual central nervous system problems. This illness may also be acquired by humans.

Transmission

The transmission of VEE is mostly facilitated by mosquitoes, which acquire the virus by biting an infected animal and then transferring it to another animal or person via their bites and feeding (73). The rate of sickness transmission depends on the specific variant of the VEE virus and the number of mosquito populations. Enzootic subtypes of VEE pertain to diseases that are endemic to certain geographical areas. Usually, these serotypes do not spread to other regions. Enzootic subtypes are associated with the transmission cycle between rats and mosquitoes (74). While some variants of the virus may lead to human sickness, they typically do not impact the health of horses.

Outbreaks

There have been 21 documented occurrences of Venezuelan equine encephalitis virus epidemics throughout the Americas. From December 1992 until January 1993, In all, there were 28 documented instances of the sickness, resulting in 12 fatalities (75). In June 1993, there was a significant increase in the number of cases in the Venezuelan state of Zulia, resulting in the deaths of 55 people and 66 horses. In 1995, there was a much greater epidemic in Venezuela and Colombia. Over time, the epidemic extended both northward and southward. The epidemic resulted in about 11,390 instances of fever in humans, along with 16 fatalities (71). Approximately 500 instances of equine cases were documented, resulting in 475 fatalities. A disease epidemic transpired in Colombia in September 1995. The epidemic led to 14,156 confirmed instances of Venezuelan equine encephalitis virus infection in humans, resulting in 26 fatalities.

Vectors: The mosquitoes belonging to the genera *Culex* (specifically *Culex taenopius*), *Mansonia*, *Anopheles*, and *Aedes* (specifically *Aedes taeniorhynchus* and *Aedes albopictus*).

Vertebrate hosts: Rodents: *Oryzomys* spp *Sigmodon hispidus*, *Peromyscus gossypinus*, and *Proechimys*.), pigs, horses, canids, bats, and also birds.

Symptoms and Treatment

Healthy individuals who get the virus may have flu-like symptoms, including elevated body temperatures and headaches. Individuals with compromised immune systems as well as the geriatric and paediatric populations are at high risk of experiencing severe illness or mortality as a result of this ailment. An inactivated vaccine, which includes the C-84 strain of VEEV, is administered to horses for immunisation purposes. The TC-83 strain vaccination is only administered to those in military and laboratory roles who are at high risk of catching the virus. The administration of the human vaccination might lead to adverse reactions and does not provide complete immunity to the recipient. The TC-83 strain was created by subjecting the virus to 83 cycles of replication in a culture of guinea pig heart cells. C-84 is a variant of TC-83.

2.4 Sindbis Fever

Sindbis virus (SINV) is a kind of RNA virus that has a protective outer layer and belongs to the *Alphavirus* genus within the Togaviridae virus family. Sindbis virus is genetically linked to Chikungunya *Alphavirus* (76). The *Cx. univittatus* mosquitoes were captured in Sindbis village, Nile Delta, Egypt in 1952. Human instances were first reported in Uganda in 1961, followed by South Africa in 1963 and Australia in 1967 (77). The virus was later recognized as the primary cause of a condition characterized by both a rash and arthritis. The pathogenic condition resulting from SINV infection is referred to as Pogosta illness in Finland, Ockelbo sickness in Sweden, and Karelian fever in Russia.

Transmission
The principal vectors responsible for transmitting SINV to people are mosquitoes belonging to the genera *Culex* and *Culiseta*. However, the virus has also been found in *Aedes* and *Anopheles* mosquitoes (78). Human infections in Northern Europe mostly occur during August and September, coinciding with the high prevalence of the principal vector species. It is now believed that SINV infection confers lifetime protection against subsequent infections. There is no substantiated proof of transmission between individuals. There is currently no relevant data about the danger of blood donation, however, it cannot be ruled out. However, the period during which the SINV infection can be detected in the blood is shorter and the amount of virus present is smaller compared to the Chikungunya virus infection. This indicates that the likelihood of transmitting the SINV infection via blood donation is probably minimal.

Outbreaks
Sindbis virus infection in humans is mostly recorded in Northern Europe, where it is endemic and has sporadic significant outbreaks. Incidents are sporadically documented in Australia, China, and South Africa (77). Finland has annual instances, although more significant outbreaks happen every seven years. For instance, in 2002, there was an epidemic in the heavily affected area of North Karelia, with an incidence rate of 81 cases per 100,000 inhabitants. The prevalence of subclinical and moderate instances is likely to be substantial, however, the existing data is insufficient to accurately define it.

Vectors: The primary mosquitoes that are attracted to birds are generally ornithophilic species such as *Culex spp*., but also include, *Aedes spp*., *Coquillettidia richiardii*, *Culiseta morsitans* and *Mansonia Africana*.

Vertebrate hosts: wild birds, amphibians and bats, and rodents.

Symptoms
The precise duration of the incubation period for SINV infection is often less than seven days. The characteristic features of acute SINV infection include a rash that is maculopapular and frequently itchy, which appears on the trunk and limbs. There is currently no targeted antiviral therapy available for SINV infection.

2.5 Yellow fever

The yellow fever virus was obtained from a guy with a fever in Ghana in 1927 (79). The existence of YF has been documented since the early 1900s, with previous outbreaks in Cuba being studied by the Walter Reed Yellow Fever Commission about 1900 (80). Yellow fever is a viral illness that is transmitted by the bite of a mosquito carrying the yellow fever virus. This pathogen can infect not just humans, but also other primates and other species of mosquitoes. Within urban areas, the primary mode of transmission is by *Aedes aegypti*, a species of mosquito that is prevalent in tropical and subtropical regions (81, 82). The virus belongs to the Flavivirus genus and is classified as an RNA virus. Distinguishing the condition from other ailments, particularly in the first phases, may be challenging.

Transmission
The primary mode of transmission for the yellow fever virus is by the bite of *Aedes aegypti*. However, other mostly *Aedes*, such as *Aedes albopictus*, may also act as carriers for this virus (83). Similar to other arboviruses, which are spread by mosquitoes, the yellow fever virus is acquired by a female mosquito when it consumes the blood of an infected human or another primate. Viruses enter the mosquito's stomach and, if the quantity of the virus is sufficient enough, the viral particles may invade and reproduce inside the epithelial cells (84). Subsequently, they infiltrate the haemocoel, which is the circulatory system of mosquitoes, and subsequently the salivary glands. Upon the mosquito's next blood meal, it introduces its saliva into the wound, allowing the virus to enter the bloodstream of the

bitten individual. The presence of yellow fever virus transmission from a female *A. aegypti* mosquito to her eggs and subsequently to the larvae is evident via transovarial and transstadial transmission. There are three distinct infectious cycles in which the virus is transferred from mosquitoes to humans or other primates, each with its epidemiological characteristics. Only the yellow fever mosquito *Aedes aegypti* is implicated in the "urban cycle".

Outbreaks

According to the World Health Organisation (WHO), there are around 200,000 reported cases of yellow fever globally on an annual basis (85). Approximately 15% of those who have yellow fever develop a severe manifestation of the disease, and up to 50% of those cases result in fatality since there is now no remedy available for yellow fever (86). In May 2017, the epidemic of yellow fever in Brazil seemed to be decreasing with a total of over 3,000 suspected cases, 758 confirmed cases, and 264 confirmed fatalities (87). According to estimates from 2013, yellow fever resulted in around 130,000 severe illnesses and 78,000 fatalities across Africa. Africa accounts for almost 90 percent of the estimated 200,000 cases of yellow fever that occur annually. Since the 1980s, there has been a steady rise in the incidence of yellow fever.

Vectors: *Aedes aegypti*, *Ae. Fucifer taylori*, *Ae. simpsonii*, and *Ae. africanus* in Africa, while *Haemagogus spegazzinii* in the South-American jungle YF.

Vertebrate host: Primates

Symptoms and Treatment

The first symptoms may manifest as an abrupt onset of fever, chills, intense headache, back pain, overall body discomfort, nausea, vomiting, exhaustion, and weakness. Among those who have severe illness, the mortality rate ranges from 30% to 60%. A yellow fever vaccination that is both safe and effective has been accessible. Most individuals get permanent immunity with only one dosage.

2.6 Japanese Encephalitis

Japanese encephalitis (JE) is a viral disease that specifically targets the brain (88). The Japanese encephalitis virus (JEV) is the primary cause of this infection (89). JEV belongs to the flavivirus family and is typically spread by mosquito bites. Firstly, T. Mitamura and M. Kitaoka separated this virus from the brain of human in Tokyo in 1935 (90). JEV is belonging to the Japanese encephalitis serocomplex, which consists of 9 genetically and antigenically similar viruses. Certain viruses within this complex are known to cause serious illness in horses (91). Japanese encephalitis has been seen to infect the inside of the endoplasmic reticulum and quickly amass significant quantities of viral proteins.

Transmission

The transmission of the JE virus to humans occurs via the bite of infected mosquitoes belonging to the Culex species, namely *Culex tritaeniorhynchus* (92). The virus is sustained by a continuous exchange between mosquitoes and vertebrate hosts, mostly pigs and wading birds. Humans are considered accidental or dead-end hosts because of their inability to attain sufficiently high concentrations of the JE virus in their bloodstreams, which is necessary to infect mosquitoes that feed on them (93). The transmission of the JE virus predominantly occurs in rural agricultural regions, often linked to rice cultivation and flood irrigation.

Outbreaks

Japanese encephalitis (JE) is the primary reason for viral encephalitis in Asia, affecting about 3 billion individuals residing in endemic regions (94, 95). Approximately 68,000 instances of symptomatic individuals are reported annually, resulting in approximately 17,000 fatalities (96). The case-fatality

rates vary between 0.3% and 60% and are influenced by factors such as the population and age demographics. Frequently, instances arise during epidemics.

Vectors: The predominant vector for this disease is *Culex tritaeniorhynchus*, with *Culex vishnui* in India and *Culex gelidus* in Indonesia.

Vertebrate hosts: Water birds such as herons and egrets (*Nycticorax nycticorax, Egretta garzetta*), as well as pigs, bats, and fruit bats, are known to be hosts of the Japanese encephalitis virus (JEV).

Symptoms
Common early symptoms often include pyrexia, cephalalgia, and emesis. Within the following several days, the individual may experience disorientation, and weakness, and maybe go into a coma. Individuals who get neurological disorders. Neurologic disease occurs in less than 1% of those infected with the JE virus. Approximately 20-30% of people who have encephalitis, which is characterized by inflammation of the brain, do not survive.

Treatment
The only Japanese encephalitis (JE) vaccine authorized and accessible in the United States is IXIARO, which is produced as an inactivated Vero cell culture-derived vaccine (97). For those who are 3 years of age or older, the recommended dosage of IXIARO is 0.5 ml each dose. The recommended dosage for children between the ages of 2 months and 2 years is 0.25 ml each dose.

2.7 Murray Valley encephalitis

The Murray Valley encephalitis virus (MVEV) was obtained from the brain of the human during the pandemic in 1951. In humans, it may result in enduring neurological disorders or fatality (98). MVEV is associated with Kunjin virus, exhibiting a comparable ecological relationship, but with a lower morbidity rate. The arbovirus is native to Northern Australia but may sometimes extend its reach to the southern states during the summer monsoon season when excessive rainfall causes floods in the Murray-Darling River system. These outbreaks may occur sporadically, separated by long periods, during which no or just a few cases are detected.

Outbreaks
Since its first documentation in Australia in 1974 during a substantial flooding incident, there have been a cumulative total of 45 cases of Murray Valley encephalitis (MVE), resulting in nine documented fatalities (4) (99). The most recent documented human case in Victoria was reported in 1974.

Vectors: MVEV is an arbovirus that is perpetuated via a cycle involving birds and mosquitoes. Water birds belonging to the order Ciconiiformes, such as cormorants and herons, serve as the hosts for MVEV. *Culex annulirostris* is the primary carrier of mosquitoes.

Vertebrate hosts: water birds, mainly egrets.

Symptoms
Symptoms include fever, somnolence, cognitive impairment, cephalalgia, cervical rigidity, emesis, tremors, seizures, particularly in babies, irritability, and hypotonia. There are currently no vaccinations or medications available that may effectively prevent MVE.

2.8 West Nile Fever

West Nile fever is an infectious disease caused by the West Nile virus (WNV), which is mostly transmitted by mosquito bites (100). The West Nile virus (WNV) was first obtained from the lady suffering fever in the valley (West Nile) of Uganda in 1937. Subsequently, it was also isolated from

a baby in Egypt in 1950 (101). The primary mode of transmission to humans is via the bite of a mosquito carrying the infection. Instances of West Nile Virus (WNV) arise throughout the period of mosquito activity, begin in the summer and extending into the autumn.

Transmission
The primary mode of transmission of West Nile virus to humans is via the bite of a mosquito that is carrying the virus. Mosquitoes acquire infection via feeding on birds that are already affected (102). Infected mosquitoes transmit the West Nile virus to humans and other animals via their bites. West Nile virus has been transmitted in rare instances through: Laboratory exposure, Blood transfusion and organ transplantation, Maternal transmission to the infant during pregnancy, birth, or breastfeeding.

Outbreaks
Several serosurveys conducted in 1939 in central Africa revealed varying levels of anti-WNV positivity, ranging from 1.4% in Congo to 46.4% in the White Nile area of Sudan (103). WNV was later discovered in Egypt in 1942 and in India in 1953. In 2012, the United States faced a severe pandemic that resulted in the death of 286 individuals, with the state of Texas being particularly affected by this viral outbreak.

Vectors: Ornithophilic mosquitos *Cq. Richiardii*, *Culex salinarius*, *Culex pipiens*, and *Culex modestus* are recognised vectors, although, *Anopheles maculipennis Aedes cantans*, and *Aedes triseriatus* can sometimes transfer illnesses.

Vertebrate hosts: wild birds and mammals like chipmunks, and tree squirrels.

Symptoms
Some individuals may have febrile sickness. Approximately 20% of those who get the infection have a rise in body temperature accompanied by other symptoms such as headache, muscle soreness, joint discomfort, vomiting, diarrhoea, or skin irritation. Severe symptoms in a minority of individuals. Roughly 1 out of every 150 individuals who get the infection has a serious condition that affects the central nervous system. The medication for WNV is unknown.

2.9 Usutu Fever
Usutu virus (USUV) is a kind of flavivirus that was firstly obtained from *Cx. neavei* in 1959 from South Africa (104, 105). It is an arising zoonotic arbovirus that is worrisome due to its ability to cause disease in people and its closeness in ecological behavior to other developing arboviruses like the West Nile virus. The virus mostly affects Culex mosquitoes and birds, whereas humans serve as a host that does not contribute to the spread of the virus.

Outbreaks
As of 2019, there have been around 50 documented occurrences of the infection in people, mostly occurring in Europe. USUV has been documented in many African nations, such as the Central African Republic, Nigeria, South Africa, and Uganda (106).

Vectors: *Culex* spp. (*C. perfuscus, C. univittatus*), *Mansonia Africana* and *Coquillettidia aurites* are mostly ornithophilic mosquitoes. *Cx. Hortensis, Cx. pipiens, Culiseta annulata, Cx. territans, Ae. Rossicus, and Ae. Vexans* are the vectors found in Italy.

Vertebrate hosts: Pipistrellus bats, as well as deer, horses, dogs, wild boar, shrews, and rodents, have been reported to possess anti-USUV antibodies. Humans and horses are terminal hosts. Presently, there is no targeted treatment available for Usutu virus infections.

Table 3: Mosquito-borne diseases that affects the Human

Disease	Pathogen	Hosts	Geographical distribution	Impact on wildlife	Impact on Human health
Eastern equine encephalitis (EEE)	Zoonotic Togavirus	Passerines, amphibians, reptiles, and rodents.	East Coast of the US, Northeastern United States	(EEE) is a viral disease that primarily affects birds, and horses in wildlife.	In 1938, thirty children in the Northeastern United States died of encephalitis.
Western equine encephalitis (WEE)	Alphavirus	squirrels, birds, jackrabbit.	United States, California,	The virus affects the bird population, if this disease is widespread in an area.	overall fatality rate of WEE is modest (about 4%).
Venezuelan equine encephalitis	Venezuelan equine encephalitis virus	Horses (with a high viremia level), canids, pigs, birds.	America, Venezuelan state of Zulia, Colombia	VEE outbreaks in wildlife contribute to the amplification of the virus and increase the risk of transmission to horses and humans.	In September 1995, there were 14,156 human cases of Venezuelan equine encephalitis virus during this epidemic, and 26 people died as a result.
Sindbis Fever	Sindbis virus (SINV)	Wild birds, rodents, bats, and amphibians.	UK, Belarus, Bulgaria, Czech Republic, Estonia, Finland, Germany, Spain, and Sweden.	Sindbis virus in wildlife contributes to the broader knowledge of arbovirus ecology and epidemiology.	Region of North Karelia was 81 cases/100,000 people during the 2002 epidemic.
Yellow fever	Flavivirus	Primates	Africa, Brazil, Cuba.	Non-human primates, such as monkeys, serve as natural reservoirs for the yellow fever virus in the sylvatic cycle. They can become infected with the virus and contribute to its maintenance in the forest ecosystem.	World Health Organization (WHO) estimates that 200,000 people contract yellow fever across the globe.
Japanese Encephalitis	Japanese encephalitis virus	Waterbirds, including herons and egrets.	Tokyo, temperate regions of Asia.	Japanese encephalitis in wildlife is important for understanding the dynamics of the virus in natural ecosystems.	There are over 68,000 cases with symptoms and almost 17,000 fatalities of JEV.
Murray Valley encephalitis	Kunjin virus	Wetland birds, particularly egrets	Australia's eastern coast	MVEV in birds may have ecological implications, influencing the abundance and	Viruses can progress to affect the central

				distribution of certain species.	nervous system, leading to encephalitis (inflammation of the brain).
West Nile Fever	West Nile virus	Animals, such as chipmunks and tree squirrels	Uganda, Egypt, central Africa, southern Europe, southwest Asia, and Australia.	West Nile virus can cause severe illness and death in infected birds. Infected birds exhibit neurological symptoms such as tremors, weakness, and lack of coordination.	In US history occurred in 2012, killing 286 people.
Usutu Fever	Usutu virus	Animals such as horses, dogs, deer, wild boar, rats, and shrews.	Austria, Belgium, Croatia, France, Germany.	Usutu fever can have ecological consequences by affecting bird populations, which play crucial roles in ecosystems.	Severe neurological complications, such as inflammation of the brain, and paralysis.

Ticks-borne diseases

2.10 Tick-borne Encephalitis

Tick-borne encephalitis (TBE) is a viral illness caused by a specific virus called Tick-borne encephalitis virus (TBEV) (107). This virus belongs to the Flavivirus genus and is a positive-strand RNA virus. TBEV is transmitted by tick bites and is related to the development of tick-borne encephalitis. The transmission of Tick-borne encephalitis (TBE) virus occurs by the biting of a tick that is carrying the virus. The TBE virus is present in some areas spanning from western and northern Europe to northern and eastern Asia.

Transmission

Hard ticks of the family Ixodidae are responsible for transmitting the tick-borne encephalitis (TBE) virus (108). Ticks acquire the infection when they consume blood from infected vertebrate hosts, especially tiny rodents, and may then transfer the virus during their following blood meal. Ticks may acquire the virus by transtadial transmission (from larva to nymph to adult ticks), transovarial transmission (from adult female tick to eggs), or through co-feeding on infected animals. The peak time of tick activity and risk of transmission is during the warmer months spanning from April to November.

Outbreaks

Each year, an estimated 10,000-12,000 clinical cases of tick-borne encephalitis are officially recorded (109). However, it is widely thought that this number considerably underestimates the true total number of clinical cases.

vectors: Ticks (*I. Ricinus*, and *I. persulcatus*).

Vertebrate hosts: Rodents (*Myodes spp*., and *Apodemus spp*.) and insectivores (*Sorex araneus*, *Erinaceus concolor,* and *Talpa europaea*).

Symptoms

Severe illness often manifests as either encephalitis (infection of the brain) or meningitis (inflammation of the membranes surrounding the brain and spinal cord). Early manifestations may include pyrexia, cephalalgia, emesis, and asthenia. There is no medicine to treat TBE viral infection.

2.11 Omsk Hemorrhagic Fever

Omsk hemorrhagic fever is a viral illness characterized by bleeding, caused by a kind of virus called *Flavivirus*. The virus was discovered in Siberia and subsequently named after a disease epidemic that occurred in the city of Omsk (110). The first documentation of the novel virus emerged between 1940 and 1943. Instances of human OHF infections have been documented in the region since 1941 (111, 112). OHFV mostly infects rodents, with the non-native muskrat being the major host. Omsk Hemorrhagic Fever Virus (OHFV) is first acquired by ticks, which then transfer it to rats via their bites. Humans get infection by tick bites or direct touch with a muskrat. Humans may get infections by coming into touch with the blood, excrement, or urine of a dead or ill muskrat (or any kind of rat). The number of cases in the endemic region ranges from 100 to 200 each year.

vectors: *Dermacentor reticulatus* tick

Vertebrate hosts: Rodents (*Arvicola terrestris*, *Ondatra zibethicus*, *M. oeconomus,* and *Microtus gregalis*).

Symptoms

The individual has symptoms such as chills, headache, discomfort in the lower and upper limbs, and extreme fatigue. Additionally, there is a rash on the soft palate and enlarged glands in the neck. There is no specific medication for OHF, however, supportive care is essential.

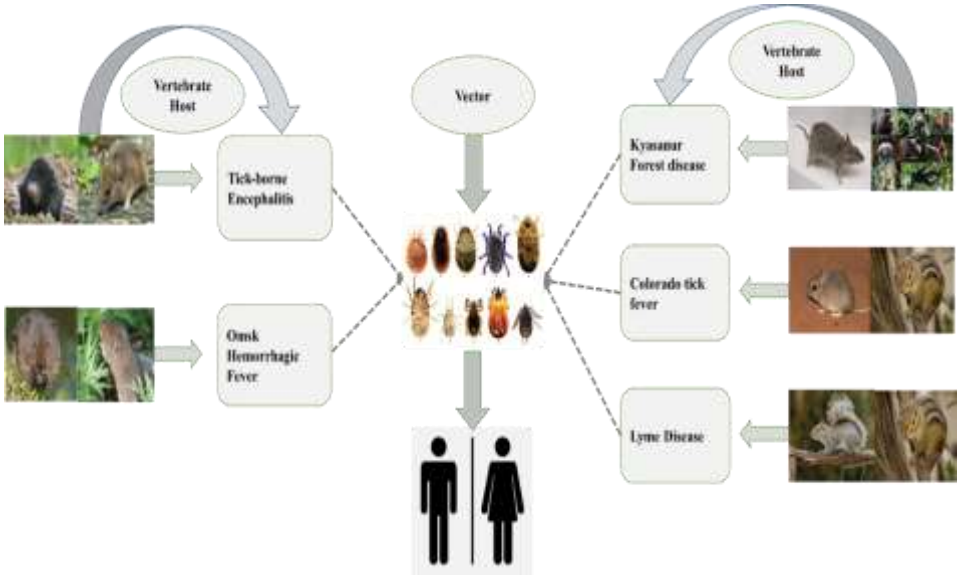

Figure 5: Image shows the patterns of transfer wildlife diseases into human through the Vectors

2.12 Kyasanur Forest disease

Kyasanur forest disease (KFD) is a viral illness transmitted by ticks that is prevalent in the Southwestern region of India (113). The first KFDV strains were obtained from sick individuals, monkeys, and Haemaphysalis ticks during an unexpected epidemic in the tropical Kyasanur Forest near Baragi.

Transmission

Several animal species, such as mice, rats, shrews, porcupines, and squirrels are believed to serve as reservoir hosts for the illness. Monkeys serve as the primary reservoirs for the KFD virus and are susceptible to infection by the virus. bonnet macaque and Northern plains gray langur have a high susceptibility to the Kyasanur Forest Disease (KFD) virus. They experience a significant increase in the amount of virus in their bloodstream and transmit the infection to the ticks. The tick species *Haemaphysalis spinigera*, which is prevalent in woods, is the disease's carrier. Humans get infected

by the biting of tick nymphs. The human is the definitive host for the ticks, and there is no transmission of the disease between humans due to the unsuitability of the human home environment for tick survival.

Outbreaks

India has consistently recorded an annual average of 400-500 cases since 1957, with a case fatality rate ranging from 1% to 3% (114). Between 2003 and 2012, a total of 3263 cases were recorded in these regions, out of which 823 instances were verified via laboratory testing.

Vectors: The tick species include *H. turturis* and *Haemaphysalis spinigera*. ALKV was found in *Hyalomma dromedarii* and *Ornithodoros savignyi*.

Symptoms and Treatment

Symptoms include fever, chills, and headache occur quickly. Kyasanur Forest Disease (KFD) is thought to have a 3–5% death rate. The vaccine for Kyasanur Forest Disease Virus comprises KFDV that has been inactivated using formalin. There is a 62.4% success rate for the vaccination among people who get two doses of the immunization.

Table 4: Ticks-borne diseases that affects the Human

Disease	Pathogen	Hosts	Geographical distribution	Impact on wildlife	Impact on Human health
Tick-borne Encephalitis	Tick-borne encephalitis virus	*Talpa europaea, Sorex araneus, Apodemus and Myodes.*	Northern and western Europe to northern and eastern Asia	Certain wildlife species, including mammals like rodents, can act as reservoir hosts for the TBEV.	Diseases (encephalitis), (meningitis), Fever, headache, vomiting, and weakness affect the human health.
Omsk Hemorrhagic Fever	*Flavivirus*	*Arvicola terrestris, M. oeconomus, Ondatra zibethicus, Microtus gregalis.*	Canada, Siberia, Novosibirsk, Kurgan, and Tyumen	Tick-borne diseases, the presence of infected ticks can potentially lead to negative health effects in wildlife populations.	Enlarged glands in the neck, the appearance of blood in the eyes (conjunctival suffusion), chills, and headaches are severe forms of Omsk Hemorrhagic Fever.
Kyasanur Forest disease	*Haemaphysalis spinigera*	*Rattus blanfordi* and *R. rattus, Funambulus tristriatus*	The southwestern region of India	Cause some serious infections in rodents.	Chills, fever, and headache are the abrupt onset of symptoms that accompany KFD.

3 Conclusion

This extensive investigation of several zoonotic illnesses highlights the crucial significance of being informed, taking preventive measures, and using efficient management tactics. Every illness mentioned presents distinct obstacles, ranging from Eastern Equine Encephalitis (EEE) transmitted by mosquitoes to Tick-borne Encephalitis (TBE) transmitted by ticks, and viral hemorrhagic fevers such as Ebola and Omsk Hemorrhagic Fever. Rabies and Leptospirosis exemplify the many modes of transmission of these illnesses and their consequences for both people and animals. The study highlights the need for wildlife workers, healthcare experts, and the general public to possess comprehensive knowledge on the frequency, transmission, and symptoms of these illnesses. The

recorded occurrences of diseases stand as clear reminders of the persistent danger, requiring constant monitoring and aggressive actions. Preventive measures, such as vaccines, personal precautions, sanitary standards, and immediate medical care, are essential in reducing the hazards involved with handling animals and working in natural surroundings. The many vectors and vertebrate hosts mentioned emphasize the intricate nature of disease transmission cycles, underscoring the need of multidisciplinary approaches to disease treatment. it is essential to implement the following suggestions for efficient disease prevention:

1. Ensure you are well-informed on the zoonotic illnesses that are common in your area and acquaint yourself with their clinical signs.
2. Prioritize acquiring pre-exposure vaccines, with a focus on illnesses such as rabies.
3. Reduce exposure hazards by taking personal safeguards. To protect yourself against disease vectors such as ticks, mosquitoes, and fleas, wear gloves and masks and use efficient repellents.

By following these guidelines, wildlife workers may strengthen their protection against zoonotic illnesses, creating a safer work environment and encouraging the harmonious coexistence of people and animals. This diligent approach not only safeguards the well-being of people but also enhances the general capacity to withstand and maintain ecosystems where interactions between humans and animals take place.

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