Review Article
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Japanese Encephalitis Virus: A Zoonotic Arbovirus in the One Health Context

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Abstract

Japanese encephalitis (JE) is a mosquito-borne viral zoonosis. The disease is caused by the Japanese encephalitis virus (JEV) of the genus *Flavivirus* and the family *Flaviviridae*. It is the leading cause of encephalitis, mostly among children in Asia. Host, environment, vector and climatic conditions play a major role in the emergence of JE. Wading birds act as reservoirs, whereas pigs act as amplifying hosts for the virus. The virus is transmitted mostly by the *Culex* species of mosquitoes, especially *Culex tritaeniorhynchus*. However, the virus can be transmitted without the involvement of a vector from pig to pig via direct contact. Human infection may happen without the presence of pigs in nearby areas. Humans act as a dead-end host for the virus. The prevention and control of the disease are primarily aimed at vector control, increased surveillance, vaccination, and stamping-out strategies. One health strategy is of paramount importance with respect to JE in attaining optimal health for humans, animals, and the environment. In this review, we have focused on the transmission cycle of JEV; the importance of vectors, reservoirs, and amplifiers in disease causation; diagnostic approaches and control measures; and the importance of the One Health approach in the mitigation of the risk of the disease.

Keywords: Japanese encephalitis, Zoonoses, One health, Climate change

Introduction:

Japanese encephalitis is caused by mosquito-borne flavivirus prevalent in Asia, Australia, and the Western Pacific (Simon et al., 2025). The disease was first recognized in Japan in 1924 (Mackenzie et al., 2006), and the virus (JEV) was first isolated in 1935 from the brain of a patient with fatal encephalitis (Mitamura et al., 1936; Lewis et al., 1947). In India, the virus was first recognized in the state of Tamil Nadu in 1955 (Carey et al., 1968), but the first epidemic occurred in 1973 in the state of West Bengal (Sengupta et al., 1974). An outbreak of the disease in India during 2005 resulted in 1700 deaths, mostly among children (Parida et al., 2006). The virus is transmitted particularly by *Culex* species of mosquitos, especially *Culex tritaeniorhynchus*. The habitat of the mosquito vector of JEV includes pools, puddles, small streams, rice fields, and human water containers. Ardeid wading birds, such as egrets and herons, serve as natural reservoirs of the virus. However, pigs act as amplifying hosts for the disease during outbreaks in human populations (Turtle and Solomon, 2018; Mulvey et al., 2021). Agricultural areas with irrigated paddy fields and pig husbandry provide breeding grounds for *Culex* species mosquitos and can also attract migratory wading birds, resulting in maintenance of the natural transmission cycle of the virus (Mulvey et al., 2021; Yang et al., 2006). However, the disease may occur in urban areas in the presence of predisposing factors. The majority of infections are asymptomatic; some develop symptoms such as fever, headache, disorientation, coma, tremors, and mental status changes due to cerebral inflammation. The mortality rate is high among the patients developing clinical signs; at least one in four patients dies (Simon et al., 2025). Children are most commonly affected and most people residing in endemic areas develop immunity by adulthood. There is no treatment for the disease except for some supportive care. Vaccination is the most effective method to prevent the disease in endemic areas. Elimination of mosquito breeding sites and prevention of mosquito bites reduces the probability of the disease occurrence to a greater extent. Only 1% of patients infected with the virus will progress to encephalitis. Whereas, 20-30% mortality is seen in patients developing encephalitis. The patients who survive the infection will have significant neurological and psychiatric sequelae in 30-50% of the cases.

Virology:

Japanese encephalitis virus belongs to the genus *Flavivirus* of the family *Flaviviridae*; the genome is single-stranded, positive-sense, non-segmented RNA in nature (Mulvey et al., 2021). The virus genome is 11 kb in size, which codes for a single open reading frame (ORF). The ORF codes for a single large polyprotein, which is subsequently proteolytically cleaved into three structural proteins pre-membrane, core, and envelope (E) and seven non-structural proteins (NS1, NS2A, NS2B,

NS3, NS4A, NS4B, and NS5). The E protein is the largest structural protein and helps in the entry of the virus into the host cell; it is also the main target for humoral immune response. The virus is enveloped, spherical in shape, and approximately 50 nm in diameter. JEV has been classified into five distinct genotypes based on the nucleotide sequence of the envelope (E) protein gene: GI, GII, GIII, GIV, and GV (Schuh et al., 2013). Human diseases are mostly caused by clade b of genotype I (GI-b) and genotype III (GIII) (Schuh et al., 2013, 2014). The neutralizing antibodies generated by all five genotypes are cross-reactive; hence, there is only one serotype of the virus.

Transmission ecology and host dynamics:

At least 14 mosquito species have been identified as vectors for the virus and 11 more species have been reported to transmit the disease experimentally (Auerswald et al., 2021). However, the disease is mainly transmitted by the bite of *Culex* mosquitos due to their zoophilic feeding pattern. Most commonly, *Culex tritaeniorhynchus* is involved in the transmission of the disease (Simpson et al., 1970). The natural cycle of JEV circulation involves a range of animal species, including *Culex* mosquitos, wading birds, and pigs (Konno et al., 1966; Dhanda et al., 1977; Soman et al., 1977). Ardeid birds such as egrets and herons act as wildlife reservoirs for JEV and play an important role in the transmission of the virus (Mulvey et al., 2021). Pigs act as principal amplifying hosts in JEV epidemics and as maintenance hosts in disease endemic areas (Gresser et al., 1958; Konno et al., 1966). Pigs develop significant viremia and sustain transmission of the virus, leading to epizootic spillover. Areas with dense pig populations have an increased risk of JE (Le Flohic et al., 2013). Outbreaks may occur in the absence of piggeries in the vicinity of human habitat if present within the flight range of vector mosquitos, or in the presence of viremic wild and migratory birds such as eagles, egrets, plovers, and redshanks (Ting et al., 2004; Yap et al., 2019; Mulvey et al., 2021). Humans are a dead-end host for JEV infection and do not develop high enough viremia to be infectious to feeding mosquitos (Lord et al., 2015). Similarly, other domestic animals (cows, dogs, chickens, goats and horses) and wildlife (flying foxes, frogs, snakes and ducks) also act as dead-end hosts for the virus. Since the amplifying hosts, such as the wading birds, are present around agricultural lands, most incidences of the disease occur in rural areas. Although mosquitoes act as vectors in transmitting the majority of infections, recent studies suggested that pigs, which are the principal amplifying host for JEV, can transmit the virus via the oronasal route through direct contact with naive pigs without the involvement of vectors (Ricklin et al., 2016a); infected hosts may also shed infectious virus. Vector-free transmission explains the rapid seroconversion of newborn piglets (Ogasa et al., 1977). Primary replication of JEV takes place in pig tonsils, where the virus persists for at least 6 weeks (Ricklin et al., 2016b). Vector-free transmission of JEV among pig populations requires further study to fully understand the epidemiology of the disease.

Clinical features:

JEV mainly affects children (< 14 years) in endemic areas; however, adults also may be affected (Campbell et al., 2011). The incubation period of the disease ranges from 4 to 15 days. In the case of humans, less than 1% of the infections are symptomatic (Turtle and Solomon, 2018). Mostly human cases are asymptomatic (99% of cases) or cause mild flu-like symptoms that last for 5-15 days, characterized by fever, headache, nausea, vomiting, diarrhea, and myalgia, which may last for several days (Amicizia et al., 2018; Turtle and Solomon, 2018). In a small number of cases, the symptoms turn to acute encephalitis, which is the most common neurological manifestation, characterized by altered consciousness, agitation, confusion, seizures, and psychosis (Basumatary et al., 2013; Turtle and Solomon, 2018). Adults often suffer from headaches and meningismus, while children develop seizures. Mortality rate is as high as 30% in persons developing encephalitis; the recovered may have lifelong mental impairments and physical ailments such as difficulty in speaking and moving (Ghosh and Basu, 2009; Turtle and Solomon, 2018; Vaughn and Hoke, 1992).

In pigs, an outbreak of JE may go unnoticed since the infection is mostly asymptomatic or mildly symptomatic (Konno et al., 1966; Ricklin et al., 2016b; Zimmerman et al., 2019). Young pigs generally suffer from the neurotropic disease, usually encephalitis; however, it is less common, maybe due to the presence of maternal antibodies, which can protect the piglets for up to 6 months (Scherer et al., 1959; Yamada et al., 2004; Zimmerman et al., 2019). In piglets, initially there is fever, anorexia, and depression, followed by neurological signs such as hind limb tremors and ataxia (Park et al., 2018, 2021). Stillbirth, mummification, abortion, and birth of weak piglets are characteristic of JEV infection in gestating gilts and sows (Takase et al., 1987; Lindahl et al., 2012; Zimmerman et al., 2019). JEV infection of boars is associated with infertility (Ogasa et al., 1977; Yamada et al., 2004; Zimmerman et al., 2019). Pigs develop high viremia following primary infection for 3-5 days, sufficient to infect biting *Culex* species mosquitos. Seroprevalence in endemic areas is very high in pigs, ranging from 98% to 100%. There are two amplification cycles in pigs, the first when infected mosquitoes bite pigs, leading to ~ 20% infection rates. The second cycle involves mosquitoes feeding on these viremic pigs and

transmitting the infection to the naive pig population, resulting in 100% seroconversion among the pig population (Konno et al., 1966; Van Den Hurk et al., 2009).

Epidemiology and geographic distribution:

Factors affecting the ecology and epidemiology of JEV are complex, involving hosts, vectors, amplifiers, and human behaviour. Nearly 68,000 cases of Japanese encephalitis are reported each year, of which 75% occur in children under 14 years of age (Campbell et al., 2011). Severe disease occurs in about one in 250 infections (Kulkarni et al., 2018). There is seasonal transmission of the virus; the disease occurs from summer to fall in temperate climates and throughout the year in tropical climates (Turtle and Solomon, 2018). Mosquitos generally bite between dawn and dusk. The disease is endemic in at least twenty-four countries in Southeast Asia and the Western Pacific. Changes in the climate, including global warming, changes in precipitation, and increased flooding and wind patterns, have a significant impact on the distribution of vectors, reservoirs, amplifiers, and the genotype of the virus. Climatic alterations change the breeding pattern, size, and life span of mosquito vectors. It also impacts the dispersal pattern of aquatic birds. Changes in wind patterns may lead to the dispersion of mosquitos to a relatively longer distance and the introduction of the disease in new areas (Kay and Farrow, 2000; Ritchie and Rochester, 2001; Huestis et al., 2019).

JEV is one of the major causes of viral encephalitis in India. In the country first human case was reported from North Arcot district of Tamil Nadu in 1955. Initially the disease was mostly confined to the Southern States, then first major outbreak resulting in 42.6% fatality rate was reported in the Burdwan and Bankura districts of West Bengal in 1973 (Bandopadhyay et al., 2013). JE is now endemic in several states of India with approximately 597 million people living in JE endemic regions and 1,500 to 4,000 cases are reported every year (Kabilan et al., 2004). A total of 24 States/UTs are affected with JE but the State of Uttar Pradesh (UP) contributes more than 75 per cent of total JE cases. JEV strains of genotype III are the predominant genotypes in India, however genotype I is also reported in some cases (Parida et al., 2005; Fulmali et al., 2011). JEV vaccination campaign for children of 1-15 years of age was started in India in 2006. In 2013 it was integrated into the routine immunization schedule in endemic areas (Vashishtha et al., 2015). Different factors such as water logging, flooding, animal husbandry, high temperature and humidity etc. plays an important role in incidences of the disease in India.

Diagnosis and control strategies:

Several serological tests are available for the detection of the diseases, such as the neutralization test, agar gel immunodiffusion test (AGID), single radial haemolysis (SRH), complement fixation test (CFT), and hemagglutination inhibition test (HI) (Kumar, 1999). These tests are usually performed on paired sera acute and convalescent—taken 14 days apart, and a fourfold rise in titer to JEV is indicative of recent infection. Isolation of the virus from blood is often unsuccessful, considering the short period of viremia; however, it can be isolated from brain tissues at necropsy or from CSF (Solomon et al., 2000). Polymerase chain reaction has been employed in the detection of JEV infection, but it is not very sensitive, and a negative result should not rule out the disease (Yang et al., 2004; Swami et al., 2008; Yeh et al., 2010; Patel et al., 2013).

Due to a lack of effective treatment, prevention of the disease is important. Community awareness is important in the control of Japanese encephalitis. Avoiding mosquito bites by wearing protective clothing such as long sleeves, long pants, socks, and closed-toe shoes. Mosquitoes may bite through thin clothing; therefore, clothes may be treated with repellent such as permethrin, DEET, etc. to reduce risk. Transmission of the virus occurs most commonly between dawn and dusk, so outdoor activities should be avoided during this period. Mosquito nets should be used to prevent bites during sleep. Despite the huge disease burden, the surveillance is lacking in different Asian countries due to a lack of diagnostic capabilities and misdiagnosis due to similar symptoms to other viral infections. Increased surveillance measures are required to ascertain the true burden of the disease.

Vaccination is paramount in the prevention of the disease in endemic areas. Inactivated Vero cell culture-derived JEV vaccine (IXIARO) and a mouse brain-derived vaccine (JE-VAX) have proven to be effective in the control of the disease. However, the mouse brain-derived vaccine was later discontinued as it was associated with acute disseminated encephalomyelitis (Turtle and Solomon, 2018). Presently, three vaccines are licensed for JE: inactivated Vero-cell-derived vaccine (based on Beijing-1 or SA 14-14-2 strains), live attenuated vaccine (JEV SA14-14-2), and live chimeric vaccine (ChimeriVax-JE) (Chen et al., 2015; Hegde and Gore, 2017; Hills et al., 2019). The live attenuated SA 14-14-2 vaccine manufactured by Chengdu Institute of Biological Products (CDIBP), China, is immunogenic, induces long-term immunity, and requires few doses in childhood to provide protection (Hennessy et al., 1996). This vaccine was selected for vaccination in India based on its success in the neighbouring country, Nepal (Bista et al., 2001). A safe and effective recombinant live attenuated SA 14-14-

2 JEV vaccine is also available (Chokephaibulkit et al., 2016). In India the JE vaccination campaign started in 2006 as a one-time mass immunization program for all children in the age group of 1-15 years living in high-risk districts using live attenuated SA 14-14-2 vaccine. Later it was integrated into the immunization schedule in endemic areas during 2013. The disease has been eliminated from countries like Japan, Korea, and Taiwan by immunization and eradication programs (Yang et al., 2006).

Though pigs suffer from neurological and reproductive disorders caused by JEV and also act as a source of epizootic spillover, acting as amplifying hosts, there is still no licensed vaccine for use in pigs. Some regionally approved vaccines in Japan, China, and Korea are the AT222, ML17, and Anyang 300 vaccines, respectively (Fujisaki et al., 1975; Lee et al., 2012; Fan et al., 2013; Nah et al., 2015). However, immunization of pigs cannot completely eliminate the risk of human JEV infection due to the rapid increase in the population of pigs, the high cost involved with the vaccination program, and the involvement of wildlife reservoirs of the virus. Though vaccination of pigs can reduce the disease occurrence in pigs, its impact on human infection is relatively minimal. Further, maternal antibodies from sows vaccinated with live attenuated vaccine render vaccination of piglets less than 3 months of age ineffective (Scherer et al., 1959). Removal of piggeries from areas of high human density is another proven way of minimizing the occurrences of the disease (Solomon, 2000; Wong et al., 2008; Ladreyt et al., 2019).

One health perspective:

Emergence of arthropod-borne viral infections such as JE involves a complex interplay between vector, host, environment, climate, and anthropogenic factors (Impoinvil et al., 2012). It is a major public health problem, especially in Asian countries, and is the leading cause of mosquito-borne viral encephalitis, affecting mainly children. Understanding the disease dynamics and mitigating the risk of JE, the One Health approach is of paramount importance. An interdisciplinary approach emphasizing the interaction of pathogen and environment to explore a new understanding of health, biodiversity, and ecology is required.

Conclusion:

JE is an important vector-borne viral disease of humans, mostly affecting children in Asia. Mosquitos of the *Culex* species, especially *Culex tritaeniorhynchus*, act as vectors for the disease. Wading birds of the family Ardeidae and pigs act as reservoirs and amplifying hosts, respectively. Though, infection may occur in the absence of pigs, directly from the reservoirs via mosquito vectors. Infection in pig results in neurological disorders such as encephalitis in young pigs; in gestating gilts and sows, there is a reproductive disorder characterized by stillbirth, mummification, abortion, and birth of weak piglets. In humans, children are mostly affected and suffer from acute encephalitis. Humans act as dead-end hosts for the virus. There is no effective treatment for the disease. Hence, control strategies such as surveillance, vector control, and vaccination are important for mitigating the risk of human as well as animal infection. There is a complex interplay between different environmental factors, vectors, hosts, and the pathogen in transmission dynamics of the disease. Hence, a One Health approach comprising routine surveillance for vectors, routine serological surveillance in wild animals, surveillance of domestic animals, and diagnostic confirmation of encephalitis cases in humans and animals is important. Changes in environmental parameters, lack of adequate surveillance strategies in endemic countries, and poor community awareness of JEV transmission pose a threat of further expansion of the infection among the human population (Wood et al., 1991, 1992; Hanna et al., 1999). Low vaccination rates and a lack of vector control programs in most of the endemic countries make control of JE challenging. Changing climate poses the risk of introducing JEV to regions that have never experienced the disease before. The enzootic cycle of JEV is not completely understood in terms of transmission and maintenance of the virus.

Conflicts of interest:

The authors declare that there is no conflict of interest.

Authors' contribution:

All authors contributed equally in writing of this article.

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