Japanese encephalitis (JE) is a newly entrant dreadful mosquito borne viral disease prevalent in India. It is basically an enzootic infection in its natural cycle. Man is an accidental host. In the fifties a few sporadic cases were recorded in the north Arcot district in South India. Prior to this the disease was not known in this country. In 1973 for the first time Japanese encephalitis (JE) occurred in an epidemic form in West Bengal involving an area of about 2000 sq km, attacking 970 people and killing 419 persons (Hati A K 1986). But wherefrom and how the disease has come? What may be the possible reason? Was it introduced accidentally through migratory birds or animal, serving as reservoirs or amplifiers of the virus or was it imported through miscreants, deliberately adding a new medical problem? Was there only zoonotic cycle operating before the disease broke out in humans getting some favourable condition such as optimum vector density, spilling over virus from the animal world to man, extension of agricultural activities, environmental facilities, weather condition etc. It may be mentioned that the disease was a clinical entity in Japan since 1871. It started in epidemic form in India after 100 years of its clinical recognition (Hati A K 1990).

Japanese encephalitis (JE) is a mosquito borne disease caused by a member of group B arbovirus belonging to the family Togaviridae, genus Flavivirus (Huang 1982).


In transmission cycle man serves as a blind end probably because of very short lived viraemia. Mosquito-borne arboviruses can be carried through the winter in hibernating female mosquitoes, although the ecological importance of this remains uncertain. The arrival of virus in migrating birds may set off seasonal transmission if it coincides with high densities of birds biting mosquitoes. Investigations of arbovirus epidemiology are frequently hampered by difficulty of distinguishing and identifying the female of Culex spp., taxonomy of which is based on the morphology of male terminalia in many cases (Gordon work 1976).
The disease was first recognised in Japan in 1871. The virus was recovered from a fatal case in 1934. Major human outbreaks have been reported from Japan, Republic of Korea, China (Taiwan), Thailand and India (Pant 1979 a). Sporadic cases have been reported from Japan, Republic of Korea, Siberia, Taiwan, Malaysia, Indonesia, Singapore, Thailand, Philippines, Sri Lanka and India (Bhattacharya S. 1985).

The annual morbidity exceeds 50,000 cases with highest incidence in temperate and subtropical regions of China, northern Thailand and India, where epidemic occur in summer months coincident with abundance of the peripheral vectors Culex mosquitoes.

Recent outbreaks have occurred in Saipan and Okinawa, the first in over 40 years; Between 1978 and 1992 a total of 24 cases has occurred in United States expatriates visitors living in endemic areas. The vectors breed in irrigated rice fields and the distribution of human cases is linked to the exposure in this ecological setting Culex vector bite preferentially at sunset and sunrise. Pigs and certain birds species are the amplifying viraemic host in the transient cycle and man and equids are incidental dead end hosts (Gordon Cook 1996).

In India, some opine that pigs are the only host and pig mosquito-pig and pig-mosquito-man cycle is only operating. But recent study shows that birds also play a very important and significant role in transmitting the virus in nature as JE antibodies were detected in both young sentinel and wild birds through out the year JE antibodies were also detected in pigs (Hati AK 1990).

Viruses are maintained in nature by mosquitoes vertebrate and mosquito cycle. Restricted interaction between viruses and vector species and vertebrate hosts tend to confine the geographic spread but occasionally a virus escapes its usual ecologic niche and causes wide spread epizootics. Hence infection are seasonal and are acquired in endemic area.

The perennial transmission is going on in nature in its maintenance cycle which spills over to the human population at time, of peak circulation. The peaks may be related to the influx of young non immune birds and new born mammals into the animal population in summer.

In India the disease was recognised in 1955 in Christian Medical College Hospital, Vellore, North Arcot Districts, Tamil Nadu. In the following year 1956; the virus of Japanese encephalitis was isolated from wild caught mosquitoes (Carey et al 1969). The virus was recovered from man in 1958 from brains of encephalitis cases (Webb et al 1964).
and the disease was restricted to Southern India only.

In 1973 a large outbreak of Japanese encephalitis occurred in West Bengal and this was the first recognised epidemic of Japanese encephalitis in India. Before this, there was no record of Japanese encephalitis in West Bengal (Chakraborty et al. 1975, Chakraborty 1981).

The first case of this 1973 malady was reported from Asansol mines Board of Health on 22nd June. In this year ten districts of West Bengal state involving an area about 2000 sq km were affected by Japanese encephalitis which continued up to the end of November 1973 and the percentage of mortality was 34 (Dasgupta AK 1981).

Since then JE with almost annual recurrences, high rate of case fatality and morbidity coupled with unpredictable geographical distribution has been the source of a colossal distress to the public health of West Bengal state (Chakraborty et al 1975, Chakraborty et al 1980a).

In India up to 1980 altogether 39 strains of Japanese encephalitis have been isolated and the virus was isolated from *C. vishnui*, *C. tritaeniorhynchus*, *C. bitaeniorhynchus*, *C. pseudovishnui*, *C. whitmorei*, *C. epidesmus*, *A. barbirostris*, *A. hyrcanus*, *A. subpictus* and lastly from *M. annulifera* from different parts of India (Hati A.K. 1976, Okuno T 1979, Chakraborty MS 1980).

Although Japanese encephalitis virus have been isolated in different species of mosquitoes, belonging to Culex, Anopheles and Mansonia (Hati AK 1981), it is known that the principal vectors are mosquitoes belonging to different species of Culex. Repeated isolation of JE virus from *C. vishnui* in two successive epidemics of 1973 and 1976 in West Bengal suggested the possibility of this mosquito being the vector of JE virus in West Bengal (Hati AK 1976).

The taxonomic status of *Culex vishnui* was not very clear before 1967. Reuben et al. 1969 (Reuben et al. 1969) reported that this species was a complex with *Culex vishnui*, *Culex pseudovishnui* and *Culex tritaeniorhynchus*. But about epidemiology of JE and about this complex in endemic and non endemic areas of West Bengal there was no clear cut answer. Relative importance of the constituent of the complex was not studied. In addition the appearance of JE antibody in the natural system has not been done, which is perhaps a better sero-epidemiological study as it alerts endangered population. Statistical methods can further strengthen the view points for this the study has been conducted.

*Culex vishnui*, a rice field breeder, has emerged out as the vector mosquito as thrice
the vector has been isolated in 1973, 1976 and 1983; (sixteen (16) and twenty six (26)
percent of them can imbibe human blood according to biotopes i.e. cattle sheds and human
habitations respectively)

Besides *C.vishnui*, some almost zoophilic Anopheles species (*A.barbirostris* and
*A hyrcanus*), have been incriminated as natural vector

It may be mentioned that animal-mosquito-animal cycle may be maintained by both
these Anopheles and Culex species and for animal-mosquito-animal cycle *C.vishnui* is
almost entirely responsible. It constitutes about 44% of nocturnal man biting mosquitoes.
Peak per man per night collection was in February. A second peak was in September
when indoor outdoor baits were placed overnight, about 56 percent of mosquitoes were
attracted to human indoor baits. Though man biting activity continues through out the
night 56 per cent of bites occur in the 3rd quadrant. They are attracted more to human
baits in the new moon than in the full moon nights. About 54 per cent of mosquitoes
take shelter in cowsheds and the rest in human habitations ranged from 3.3 to 11.6 percent
and 1.0 to 14.8 percent respectively. 56 percent, 10 percent and 5 percent mosquitoes
were positive for bovine, porcine and avian blood respectively (Hati AK 1990)

Just before an epidemic breakout JE antibody titre in young sentinel pigs may show
stiffness. Another simple indication may be abortion in pigs (Hati AK 1990).

Let us now find out the magnitude of the problem in our state out of 39149 cases and
14246 death in India from 1978-87, 8435 cases (21.5 percent) and 3375 deaths (23 per
cent) occurred in West Bengal

Between 1973 and 1988 no cases occurred in 1974, 1975 and 1983 and cases were
very few in 1977, 1980 and 1981. Why were there such bull periods? What factors were
responsible? Was not vector population adequate? Was not microclimate suitable for
transmission of viruses?

It was observed that in 1980 and 1981, when per man hour density of *Culex vishnui*
in human habitation was 7.5 and above, the disease started but that density was not
obtained in 1983 in the transmission season and no JE case occurred, suggesting that
optimum vector density is of prime importance in transmission dynamics which required
further longitudinal studies (Hati AK 1980) Upto 1978 the disease used to appear in
the months of May and June. But afterwards the appearance was shifted to August -
September mainly. The disease usually disappears between October and November
though in several years stray cases were there even in February. This can be explained
by the fact that because of perennial transmission in the natural host infection in human also occur outside the epidemic episodes observed mainly in the monsoon and post monsoon periods (Chakarborty et al 1980a, Chakraborty 1981)

Microclimate plays a very important role in transmitting the disease, about which very little attention has been paid by researchers Data of ten years extending from 1973 to 1982 show that a significantly large number of cases occurred when maximum temperature was between 34°C and 35°C Ranges of Maximum temperature, rainfall and maximum relative humidity when cases started in different years were 34° - 42°C, 21 - 198 mm and 92 - 100 percent respectively.

It seems that when temperature and relative humidity are high, transmission of JE in man is favoured. The number of male victims is higher than that of female victims. Though no age seems to be immune in the age group between 0-4 years and 5-19 years in 1987 12 and 47 percent of cases provided respectively. About 74 percent of the cases had age group between 0 and 4 years (Hati AK 1990)

In West Bengal the disease did not occur in Darjeeling, 24 Parganas and Calcutta. One peculiar feature is that the disease invariably would start in the district of Burdwan in Memari block I & II then it would spreads to other areas and districts This is still a big enigma Burdwan is the granary of West Bengal with several cropping in a year Irrigation facilities are in abundance The area is more humid. The density of vector mosquito is always adequate in comparison to other areas for perennial transmission in natural hosts So vector climate host interaction is possibly optimum in the District of Burdwan Further study is required in this aspect On the other hand there is no invasion of JE in Calcutta and suburbs There is practically no possibility of occurrence of JE in Calcutta and suburbs because the vector Culex vishnui is non existent, even if obtained is very very low (0-1 9 percent of the total catch) The second explanation is Arbo virus (JE) are grouped according to antigenic character After inoculations of one virus into a fresh animal, not only the homologus antibodies but also heterology antibodies reacting with other viruses of the same group tend to appear. So recovery of an infection by a member of one group of arboviruses may provide some degree of resistance to a subsequent infection by another member of the same group. Accordingly epidemics Murray valley, West Nile and Japanese encephalitis are not reported in the endemic areas of dengue because of cross resistance (Hati AK 1990)

During epidemics for an overt case of JE in apparent infection occurs in 400 - 1000
people, protecting them naturally. But this may be as low as 1.25 as found in American soldiers in Korea. In West Bengal, an apparent infection occurs in about 200 persons per 1 overt case.

Seroconversion in nature has been studied in villages in Burdwan. In 1981-82, in the village Nudipur out of 190 sera tested JE antibody was positive in 15 (7.9 percent). After 2 years, sera of 106 persons previously negative for JE antibody was examined of which 15 (14.2 percent) became positive. This shows on the one hand that JE activity in the area is continuing and on the other hand about 14 percent of the people are becoming naturally protected at the interval of two years. This type of study should be extended.

There are some peculiarities in distribution of JE cases in West Bengal. It is seen that JE is noticeable in three principal rice growing district of West Bengal i.e. Burdwan, Birbhum & Bankura. It can therefore be logically summarised that the geographical belt constitutes the chief epidemic zone of JE in West Bengal. Although such a zonal classification of the epidemiology of Japanese encephalitis in West Bengal is not at all absolute. It is seen that in some areas the disease is prevalent every year whereas there is a lull period in neighbouring area. The mystery is not fully understood. Another feature of JE is the scattered pattern of incidence. On an average 1 to 15 cases occurred per village and there was not more than one case in a household.

The present study has been planned and performed keeping in view of the above perspective which may prove helpful in understanding some interrelation between the animal reservoir, vectors and hosts related to JE in this particular zoo-geographical area.

Object of study

The recurrent epidemic outbreaks of Japanese encephalitis in certain districts of West Bengal, almost assuming an annual event, have posed one of the major public health hazards in recent time, whereas in most of the districts there is no reported case of Japanese encephalitis. So, a thorough understanding of endemicity of the disease process specially the factors related to and responsible for its spread and persistence compared to non occurrence of the disease process in other part.

JE being a vector borne disease with an extra human reservoir and having in complex "eco" system, the task lies to correlate vectors, reservoir and JE cases.
During the present work priority has been given to study the biology of *C. vishnui* in rural West Bengal, though the role of other related species has also been investigated.

**Plan of study**

1. Surveillance of adult *Culex vishnui* complex in endemic and non endemic zones
   a) Distribution and resting preference
   b) Seasonal prevalence
   c) Blood meal analysis
2. Surveillance of immature *Culex vishnui* complex in endemic and non-endemic zones.
   a) Distribution
   b) Seasonal prevalence
   c) Breeding spot searching
3. Correlation between the appearance of Japanese encephalitis antibody within the reservoir host (sentinel chick), vector population build up and appearance of infection in the human population
4. Statistical analysis to find out answer about the competition between vector species in endemic and non-endemic areas in relation to disease prevalence.

To summarise the study to be conducted both in endemic and in a non endemic zone in the same guide lines eg

(1) To study the distribution of 3 members of *Culex vishnui* complex in endemic and non endemic zone
(2) To study the appearance of JE antibody in sentinel chick (monthwise)
(3) To study the occurrence of human cases (monthwise)
(4) To correlate the above 3 findings and to study the member of vectors which is relatively more important for the disease.