Spacio-temporal Aspects of Epidemiology in Insect Borne Rice Virus Diseases

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General sketch of current status of virus disease of rice

The number of species known as vectors of rice viruses or mycoplasma amounts to 11 in total and all of them belong to either leafhopper or planthopper except chrysomelid beetle, *Sesselia pusilla* which vectors yellow mottle virus disease. Each hopper species is able to vector more than one viruses (hereafter the word virus is used as including mycoplasma) and each virus is able to be vectored by more than one vector species.

The distribution or presence of vector is a prerequisite for the occurrence of any insect borne virus disease. In temperate areas, e.g. Japan, the occurrence of virus diseases is mainly confined to the southern part of the distribution range of their vectors. Rice dwarf and yellow dwarf both vectored by the green rice leafhopper, *Nephotettix cincticeps*, and stripe by the smaller brown hopper, *Laodelphax striatellus*, are prevalent only in the southern part of Japan, i.e. south of the Kanto district, while, the green rice leafhopper and the smaller brown planthopper are distributed north to Tohoku and Hokkaido, respectively. The reason for this apparent gap in distribution range between virus diseases and vectors will be explained later.

Although the occurrence of rice dwarf virus disease transmitted by the green rice leafhopper was reported in Japan as early as 1833 from Shiga Prefectural Agricultural Experiment Station, outbreaks of the disease were rather infrequent local problems until post-World War. Outbreaks of virus diseases, e.g. grassy stunt vectored by the brown plant hopper, *Nilaparvata lugens*, and tungro vectored by leafhoppers, *Nephotettix* spp. were first reported in the 1970's in Southeast Asia as a result of the outbreaks of vectors induced by a large scale introduction of new high-yielding varieties of rice (Kiritani, 1979)[10].

Similarly, the sign of epidemy of the virus diseas of rice in Japan appeared in the later half of the 1950's when early planting of rice became prevalent. The epidemic outbreaks of rice dwarf, yellow dwarf and stripe (Table 1) occurred during the period of 1960–1965, as a result, the epidemics extended infected areas to northern Japan or Kanto district.

The spread of early planting rice caused a decrease in acreage of winter crops, particularly wheat and barley, because the early planting rice prevented winter cropping in paddy fields. As a result, area of winter-fallow paddy fields was increased, where winter hosts such as foxtail, a graminaceous weed, grew abundantly, for overwintering leafhopper nymphs. Thus, the cultivation of early planting rice caused rice dwarf epidemics not only by ensuring earlier build up of a dwarf inoculum source, but also by increasing reproduction rate of leafhoppers. In fact, the epidemic of rice dwarf began around 1965 when the acreage of wheat and barley decreased to about one half that of around 1955, and this epidemic status has continued up to present in southern Japan (Nakasuji, 1979)[18].

Table 1. Biological performance of rice virus diseases in Japan

Disease	Casual agent	Distribution	Principal vector	Transmission type	Congenitally infective insects (%)	Incubation period (weeks)		Feeding acquisition (%)	Max. active transmitters (%)
						rice	insect		
Black-streaked dwarf	Virus	Temperate	*L. striatellus*	Persistent	0	2–3	1–3	100	32
Stripe	Virus	Temperate	*L. striatellns*	Persistent & transovarial passage	42–96	2–3	1–2	20–30	52(5–14)*
Grassy stunt	Virus	Tropics	*N. lugens*	Persistent	0	2–3	2–4	—	50
Ragged stunt	Virus	Tropics	*N. lugens*	ditto	0	—	—	—	—
Transitory Yellowing	Virus	Taiwan Okinawa	*N. cincticeps* *N. virescens*	ditto	0	1–2	3–5	30–75	71
Waika	Virus	Kyushu	ditto	Semipersistent	0	4–5	0	40–60	—
Yellow dwarf	Mycoplama	Temperate-tropics	ditto	Persistent	0	3–13	3–6	88–96	78(30–40)*
Dwarf	Virus	Temperate	*N. cincticeps*	Persistent & Transovarial	32–100	1–4	2–4	20–30 (Max. 69)	20(4–14)*

* Normal range of active transmitters in epidemic areas.

It should be noted, however, that decreased acreage of wheat and barley brought about a decreasing incidence of rice stripe in the same regions. This is because the smaller brown planthopper, the vector of stripe, reproduces on these winter crops early in the spring.

Abrupt outbreak of *waika* disease, a tungro-like disease, occurred in 1967 in Kyushu Island. The outbreak reached its peak in 1973, covering 25,000 ha of rice, when the acreage of a recommended rice variety, 'Reiho', shared one half of the total rice acreage in Kyushu Island. The epidemic, however, was suppressed in 1976 by intensive control of the green rice leafhopper and use of varieties more tolerant to *waika* disease than Reiho (Kiritani, 1979).[10]

In view of the fact that the brown plant hopper immigrates into Japan by long distant migration over the sea, the occurrence of tropical virus diseases vectored by the insect, i.e. grassy stunt and ragged stunt, could be expected in Japan. Actually, the occurrence of the former was reported from all prefectures in Kyushu Island in 1978 (Iwasaki, & Shinkai, 1979)[8]. In the following year, ragged stunt was also observed in Kagoshima Prefecture (Shinkai, Nakano & Iwasaki, 1980)[24]. No doubt, such recent discovery of virus diseases of tropical origin in Japan may be related to epidemic prevalence of these virus diseases resulting from the outbreaks of the brown planthopper in tropical Asia.

Essential components of the epidemics of rice virus diseases are virus sources, susceptible variety of rice and vectors. The intensity of epidemics depends on the extent of these components. It should be mentioned that there is no reason to expect any cyclic periodicity in plant virus epidemics. There is no reported cases of immunity of plants to virus diseases. In addition, such an annual crop as rice is grown every year by newly introducing virus-free plants. This nature forms a contrast to such an animal virus disease as influenza. Therefore, there is no biological base to expect cyclic periodicity of virus epidemics inherent to the interaction system between rice and virus.

The term 'vector' here refers to insects which share the same host plant, i.e. rice plant, with viruses (Kisimoto, 1972)[12]. Consequently, the term 'vector' in the strict sense excludes those insect species which are demonstrated experimentally to be able to transmit virus. For instance, three species of planthoppers other than the smaller brown hopper are experimentally capable of vectoring stripe virus to rice, but they are never vectors of stripe under natural conditions, because all of them are not able to reproduce on rice (Kisimoto, 1972)[12].

Factors responsible for virus epidemics

1) Susceptibility of rice

Abundant virus source provided by virus-infected rice plants is one of the prerequisites for an epidemic of rice virus disease. Generally speaking, rice varieties grown in Japan are more or less susceptible to virus diseases, and no varieties are known to be highly resistant. Both lowland and upland rice varieties are susceptible to black-streaked dwarf (Ishii & Yosimura, 1973)[7]. Although upland rice and most of the indica type lowland rice are resistant to stripe, all Japanese lowland varieties are susceptible. The only commercial lowland variety being resistant to stripe is 'Mineyukata (Chugoku, No. 46)' which was bred by incorporating a resistant gene from indica rice (Toriyama & Sakurai, 1969)[25].

Although there exist a great variation in the degree of susceptibility to dwarf among lowland rice varieties, there is no highly resistant variety. All upland varieties are known to be susceptible (Ishii et al., 1969)[5]. Japonica lowland rice is also susceptible to yellow dwarf. As a result, Mineyukata is the only commercial variety available in Japan for the use against virus diseases (Shimura, 1977)[21].

The susceptibility of rice plant to virus infection is closely related to the age of plant. The younger the rice plant, the more suscep-

tible it is to virus disease with more serious symptom and a shorter incubation period (the duration from infection to disease emergence). Consequently, the infection at a younger stage causes greater yield loss. High temperatures also contribute to shorten the incubation period in rice plant (Shinkai, 1962[22]; Hirao, 1969[1]; Nakasuji, 1974[18]; Kisimoto, 1972[12]; Uehara & Tozaki, 1975[26]).

2) Interaction between virus and vector
(1) Transmission cycle in relation to distribution.

The transmission cycle of rice viruses vectored by either leafhopper or planthopper can be categorized either persistent or non-persistent including semi-persistent. Some of the persistent viruses are transmitted transovarially from one generation to the next (Table 1).

Major differences in transmission cycles are as follows (Ling, 1975)[14]. The non- and semi-persistent viruses need diseased plants to complete the cycle, because the viruses do not persist in vectors throughout their life. The vectors become infective almost immediately after acquiring the virus by feeding on diseased plants. The infectivity is lost by molting. An infective insect retains infectivity for one week at the longest. The infectivity, however, declines with the time. The existence of retention period of virus in the vector is the reason why the virus is designated as semi-persistant (Saito, 1977[20]; Hirao & Inoue, 1978[2], 1979[3]). In Japan only *waika* disease belongs to this type of transmission cycle.

On the contrary, with persistent viruses, the vector becomes infective after a long incubation period (in weeks) after acquiring the virus. Its infectivity is not affected by molting and remains infective throughout its life. The difference between transovarial, e.g. dwarf, and non-transovarial transmission cycles, e.g. yellow dwarf, is that in the former group, the vectors retain their infectivity through successive generations without access to a diseased rice plant, while the vectors in the latter group need to acquire the virus

every generation to complete the transmission cycle (Ling, 1975))[14]. In temperate areas, the transmission cycle of yellow dwarf is cut off unless overwintering nymphs acquire the virus by feeding on tillers regenerated from stubbles of diseased plants.

Table 1 gives differences in biological performances among virus diseases recorded in Japan. A salient point shown in this table is that the geographical distribution of rice virus diseases is closely related with their type of transmission cycle. The distribution of non- or semi-persistent virus is confined to the tropics where rice is grown all the year round. This type of virus will not survive without the continuous presence of diseased plants including wild hosts. One of the major reasons of rapid decline of *waika* disease epidemic in Kyushu Island is the absence of susceptible wild hosts during the winter.

On the contrary, persistent type virus is able to survive the winter without diseased plants, if overwintering vectors could acquire the virus before entering hibernation. The situation is same for off-season of rice in the tropics. As a result, this type of viruses extends their distribution range from the tropics to temperate areas.

Further north in temperate areas, the duration of off-season of rice is much longer so that vectors have to pass one generation after overwintering on host plants other than rice. The transovarial transmission of virus is essential to the survival of the virus under such conditions, because only few wild hosts of the vector become infective when they are fed by infective insects. The fact that this type of virus has been reported only from temperate areas supports this hypothesis.

Grassy stunt and ragged stunt which are persistent and transmitted by the brown planthopper were discovered recently in southern Japan. They are unlikely to complete their transmission cycles in Japan. Absence of overwintering population of the brown planthopper and probable lack of suspected winter hosts of the viruses may be stated for this reason.
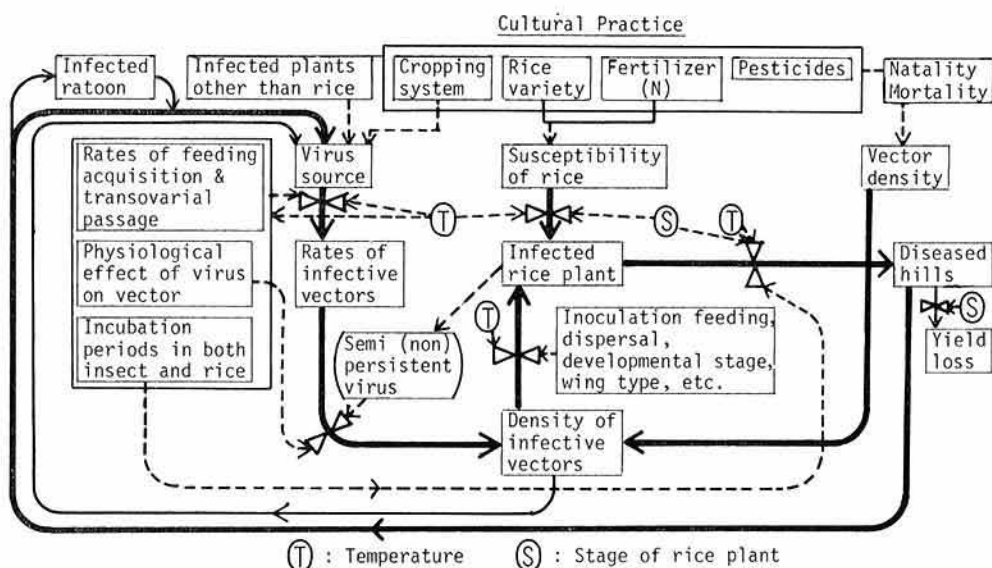
Cultural Practice

Fig. 1. Epidemiological cycle of rice virus diseases.

(2) Rate of infective insects

The rate of infective insects is determined by a complex set of factors (Fig. 1): amount of virus source in terms of infected rice hills, rate of feeding acquisition, developmental stage of vectors, type of transmission cycle, durations of incubation period of virus in both vector and rice plant, physiological effect of virus on vector, atmospheric temperatures, dispersal ability of vectors, etc.

The green rice leafhopper is not able to acquire dwarf virus by feeding at a temperature below 15°C (Kimura, 1962[9]; Ishii et al., 1970[6]). The rate of feeding acquisition becomes greatest at 25°C, followed by 30°C and 20°C in decreasing order (Nakasuji & Kiritani, 1970a[15]). In the case of *waika* disease, the insect needs only less than 3 hr for virus-acquisition feeding at 20–25°C, while it takes 12 hr at 15–20°C. Transmissive ability of infective insects at 15°C decreases to one third that of at 25–30°C, while the retention period of the virus in adult males is prolonged from 1.5 to 6–7 days with decreasing temperatures from 30°C to 15°C (Inoue & Hirao, 1980[1]).

Experiments on the relationship between the smaller brown planthopper and either black-streaked dwarf or stripe showed that the optimum temperature range for feeding acquisition and transmission was 20–25°C, while the minimum temperatures for these activities were at around 15°C and 10°C, respectively (Ishii et al., 1970[6]; Ishii & Yoshimura, 1973[7]). Shinkai (1962)[22] reported that the rate of feeding acquisition of the viruses decreased with the advance of developmental stages of the planthopper. The highest transmissive ability, however, was recorded in mature nymphs and newly emerged adults, and the ability decreased with aging of adults (Kisimoto, 1967)[11]. The incubation period of the viruses in the vector becomes short at a high temperature (Shinkai, 1969[23]).

Deleterious effects of dwarf virus on physiological traits of the green rice leafhopper were observed by Nasu (1963)[19] and Nakasuji & Kiritani (1970b)[16]. They observed shorter life span and lower fecundity of infected females as compared with non-infective ones particularly at high temperatures. Decline in the rate of infective leafhoppers during summer is considered to be related to the low reproductivity of infective females (Nakasuji & Kiritani, 1971[17]. No apparent difference, however, has been observed between

infective and non-infective insects for stripe virus in the smaller brown planthopper (Kisimoto, personal communication).

As evident from the preceeding account and Fig. 1, the epidemic cycle of rice viruses depends greatly on temperature. This means that the revolution rate of epidemic cycle becomes very low, if not stopped, under prevailing low temperatures. This would result in a gradual decline of virus epidemic even if it happened to occur in relatively cool area. The fact that the distribution range of virus diseases is confined to southern part of the distribution range of vectors could be explained by this temperature hypothesis.

Differentiation in the mode of transmission of virus

Kono (1966)[13] proposed a simple mathematical model to describe the inter-generation change in the rate of infective insects for insect-borne rice virus diseases of persistent type.

$$P_{n+1}=P_n r(1-w)+w \quad \cdots\cdots\cdots\cdots\cdots(1)$$

Where w is the feeding acquisition rate of non-infective individuals by feeding upon a diseased plant, r is the rate of transovarial passage expressed in terms of the rate of congenitally infective insects in the progeny produced by an infective parent female. P_n and P_{n+1} refer to the rates of infective insects or active transmitters in nth and (n+1)th generations.

The equilibrium rate of infective insects can be calculated by assuming

$$P_{n+1}=P_n=\overset{*}{P}.$$

$$\overset{*}{P} \frac{w}{1-r(1-w)} \quad \cdots\cdots\cdots\cdots\cdots\cdots(2)$$

The equation (2) indicates that when the value of w and r remain constant for the vector population, P_{n+1} eventually becomes stable at the value of $\overset{*}{P}$.

Fig. 2 shows a series of isoclines of $\overset{*}{P}$ value at different combinations of r and w. For case studies, we take up blackstreaked dwarf,
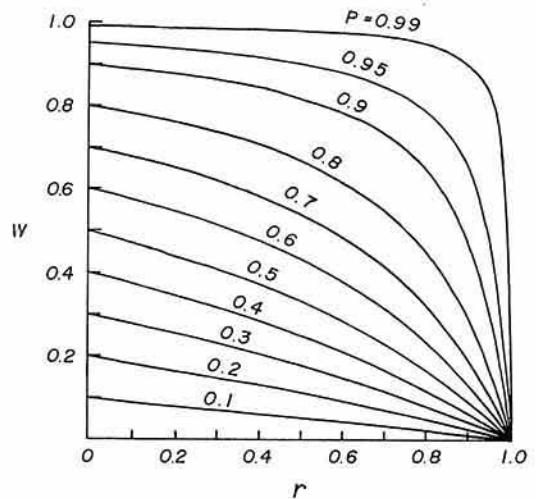

Fig. 2. Equibrium rate of active transmitters (P) at different combinations of r and w. r = rate of transovarial transmission, w = acquisition rate

yellow dwarf, stripe and dwarf (Table 1). The latter two persistent viruses have transovarial passage, while they are usually acquired by only 20–30% of non-infective insects. Although there is a great variation in the value of r depending on the populations, r values reported for stripe were 95% (Kono, 1966)[13], 90–100% (Shinkai, 1969)[23] and 83% (Kisimoto, 1972)[12].

A hypothetical virus with $w=0.8$ and $r=0.8$ will approach rapidly to an equilibrium rate of $\overset{*}{P}=0.95$. For example, starting from $P_n=0.1$, it reaches to 0.82 in the following (n+1) generation and 0.93 in the (n+2) generation and so on. However, in case of actual viruses the rate of infective insects of natural populations varies within the range of 0.1 to 0.4 even when the virus diseases are in the state of epidemics (Table 1). Under epidemic conditions, all the rice hills will be completely or partially infected by the causal agents without insecticidal applications.

If epidemics prevail to such an extent that causes complete destruction of rice plants in paddy fields, the situation is detrimental not only to vectors, but also to viruses. Natural selection would have operated against it. Kisimoto (1972)[12] found three different races

Table 2. Comparison of stripe races with different virulency (Kisimoto, 1972)

Characteristics	Race		
	Wild	Low virulency	High virulency
Symptom in rice	Strong	Weak, infected plants survive up to heading stage	Very strong resulting in a rapid death of ptant
Symptom in wheat	Weak	Strong	Very strong
Acquisition by vector	Easy	Easy	Difficult
Transovarial transmission rate	High (82.9%)	Very high (96.5%)	Low (18.8—71.3%)

in virulency of stripe (Table 2). An interesting point which emerges from this table is that the highly virulent race is not only difficult to be acquired by the smaller brown planthopper, but also has a very low rate of transovarial transmission as compared with other less virulent races. In addition, the highest transovarial transmission rate is observed for weak virulent race.

Kisimoto (1967)[11] reported that the feeding acquisition rate of stripe virus by the smaller brown planthopper is controlled genetically. He succeeded in establishing strains with high and low acquisition rates after successive selection of wild insects. Fifty to sixty percent of the individuals of high acquisition strain became infective by feeding upon diseased plants, while 0 to 10% for the low acquisition strain. Two alleles were found to be responsible for controlling the acquisition rate.

Genes responsible for the low rate are incomplete and complete dominance over those responsible for high rate. Accordingly, a high transovarial transmission rate of stripe virus would be counterbalanced by natural selection which works to keep the rate of acquisition at a relatively low level corresponding to the transovarial transmission rate. Probably the mechanism of this genetically controlled acquisition rate will be analyzed in terms of balanced polymorphism in due course. There is no study on the heredity of acquisition rate of the green rice leafhopper. As described earlier, infective females with dwarf virus have a short life span and a less fecundity when compared with noninfective ones (Nasu,

1963[10]; Nakasuji and Kiritani, 1970b[10]). It seems not questionable that this physiological handicap on the part of infective individuals operates as one of the important mechanisms in keeping the rate of infective leafhoppers at an appropriate low level under natural conditions.

The foregoing account suggests that there is a differentiation in the evolution of transmission cycle of persistent viruses. With persistent virus having no transovaria passage natural selection works in favor of high acquisition rate (w), while a higher transovarial transmission rate (r) is selected with persistent virus with transovarial passage. In the latter case, however, the acquisition rate (w) is determined at a certain balancing level in relation to the value of r. Of course, if $w=0$, the virus will not survive in the long run unless $r=1$. By contrast, as evident from Fig. 2, even a small increment in the value of w at a high level of r will result in a high percentage of infected rice plants endangering survival of virus as well as vector. The above argument is made assuming the virulency of persistent virus remains as it is. If there were a virus which shows high rates of transovarial transmission (r) and of feeding acquisition (w), the virulency of the virus would be relatively weak.

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