Section 3. Virus and Phytoplasma Diseases

Chapter 6. Phytoplasma diseases

1. Orange leaf (ROL); pathogen: ROLP

1.1. History and distribution

Orange leaf of rice (ROL) characterized by orange discoloration and leaf wilting (**ROL Figure 1**) occurs in South and Southeast Asia and southern China. In China, it occurs in southern provinces including Jianxi, Fujian, Guandong, Guanxi, Hainan, and Yunnan (Xie et al 1994).

ROL was first observed in northern Thailand in 1960 (Ou 1963). It was widely endemic in the Philippines (Rivera et al 1963). In 1962, ROL once reached an epidemic proportion locally in Laguna, Philippines. ROL is transmitted in a persistent manner by the zigzag leafhopper, *Recilia dorsalis* Motschulsky (Rd) (**Intro Figure 6**) (IRRI 1963, Rivera et al 1963). The occurrence of ROL was confirmed by transmission tests using ***R. dorsalis*** in Indonesia (Saleh et al 1977), Malaysia (Singh 1971, Saito et al 1976), the Philippines (Rivera et al 1963, Duan and Hibino 1988, Hibino et al 1987), Sri Lanka (Abeygunawardena 1969, Abeygunawardena et al 1970), and Thailand (Wathanakul and Weerapat 1969) in the Asian tropics and in Yunnan and Fujian, China (Lin et al 1983). The presence of a rice disease showing orange leaf-like symptoms was reported in northern Australia but no further information has come forth (Chapman 1976). In 1976, mycoplasma-like organisms were found in ROL-diseased rice plants in Thailand (Saito et al 1976), Indonesia (Saleh et al 1977) and the Philippines (Duan and Hibino 1988, Hibino et al 1987).

ROL Fig. 1. A rice plant infected with orange leaf disease.

ROL has been suspected to be a virus disease as the putative virus is transmitted by the leafhopper vector like several virus diseases of rice. Based on symptomatology, association of mycoplasma-like bodies, and relation with the Rd vector, ROL is considered as a candidate species belonging to phytoplasma. However, in Fujian, China, phytoplasma-like bodies were not found in rice plants showing ROL-like symptoms (Lin et al 1983). Instead, 15-nm particles were found in such plants (Lin et al 1983).

1.2. Symptoms

Rice plants infected with ROL show orange discoloration beginning at the lower leaves and starting from the leaf tip (**ROL Figure 1**). The discolored leaves roll longitudinally and eventually wilt. Rice seedlings inoculated by ROL-viruliferous Rd show orange stripes on

the outer margin or on one side of the leaf blade near the tip of the leaf 14 to 21 days after the inoculation. Leaves of infected plants show one or more well-defined orange stripes parallel to the midrib or along the veins and turn from light green or orange to nearly yellowish brown starting at the tip. As the disease progresses, the orange color becomes pronounced. The leaves gradually roll inward and dry. Affected plants tend to develop less tillers and poor root systems. Plants show stunting and generally wilt before the flowering stage. Plants infected at the later growth stages develop panicles with high sterility. Infected plants may show diseased tillers and normal looking tillers on one plant (Abeygunawardena 1969, Abeygunawardena et al 1970, Ling 1972, Ou 1985, Rivera et al 1963, Singh 1971).

In the Philippines, seedlings of rice cultivar Taichung Native 1 were inoculated in screenhouses at 7 to 10 days after soaking. The first symptoms appeared at 8-20 days after the inoculation. The emergence of new leaves was delayed in infected seedlings (Duan and Hibino 1988, Hibino et al 1987). Such leaves were short and often showed chlorotic stripes, tip twisting, and ragged blades. Infected seedlings were stunted and their leaves showed orange discoloration progressing downward from the tips, inward rolling, and eventual desiccation. All infected seedlings died 2-3 weeks after the development of symptoms. When 8 rice cultivars were inoculated with ROL, the ragged leaf symptoms appeared in IR8, IR20, and TN1 (Duan and Hibino, 1988). The symptoms are especially pronounced in TN1. Two japonica cultivars, Fukumasari and Reiho, showed stunting, yellow discoloration, rusty spots on leaves, and wider leaf angles, somewhat similar to those caused by grassy stunt (Duan and Hibino 1988).

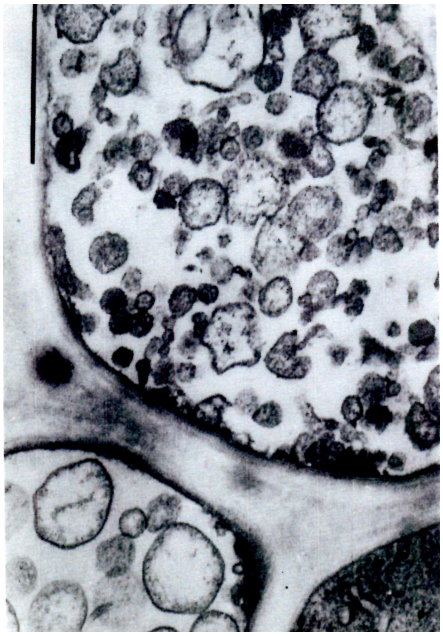
In orange leaf-infected rice plants, phytoplasma bodies occur in the phloem tubes (**ROL Figure 1**) (Duan and Hibino 1988, Hibino et al 1987, Saito et al 1976, Saleh et al., 1977).

1.3. Phytoplasma

Rice plants infected with ROL have phytoplasma bodies in the sieve elements (**ROL Figure 2**). The bodies are bounded by a triple layered membrane and contain ribosome granules and DNA-like fibrils. The bodies are pleomorphic and variable in size from 50 to 1,100 nm in diameter (Duan and Hibino 1988, Hibino et al 1987, Saito et al 1976, Saleh et al 1977). In rice tissues artificially infected with ROL in Fujian, China, spherical particles of 15 nm in diameter occurred in the phloem cells (Lin et al 1983).

Association of phytoplasma bodies with ROL has been reported in Indonesia, Malaysia, the Philippines, and Thailand (Duan and Hibino 1988, Hibino et al 1987, Saito et al 1976, Saleh et al 1977). Since rice yellow dwarf phytoplasma (RYDP) distributes widely in the Asian tropics, ROL-infected plants may also have infection with RYD phytoplasma. In these experiments, seedlings infected using ROL-viruliferous ***R. dorsalis*** have been used and possible contamination of test materials with RYDP has been eliminated.

Specific diagnosis using DNA hybridization or PCR amplification of phytoplasma DNA has not



ROL Fig. 2. Sections of the phloem of an orange leaf-infected leaf vein showing numerous phytoplasma bodies (x 32,000). Source: Hibino et al (1987).

been developed for ROLP. A DNA probe derived from plasmid of aster yellow phytoplasma hybridized with ROLP-DNAs, indicating ROLP carried plasmid related to aster yellow phytoplasma (Nakashima and Hayashi 1995).

In Fujian, China, a rice disease was identified as ROL based on symptomatology and its relation to Rd (Lin et 1983). However, phytoplasma-like bodies were not found in the rice tissues infected with the disease. Instead, particles of about 15 nm in diameter occurred in the phloem cells. When treated with tetracycline, ROLP-infected rice plants did not show sign of recovery from severe infection (Lin et al 1983). The disease in Fujian may be a virus disease but this has not been confirmed.

Phytoplasma bodies have not been isolated in pure form from ROD-infected plants and the Koch's postulates for phytoplasma etiology have not been fulfilled.

1.4. Host range

Other than rice, no other crop species is known to be infected by ROLP.

1.5. Transmission by vectors

Rd is the only known vector of ROL (Abeygunawardena 1969, Abeygunawardena et al 1970, Lin et al 1983, Rivera et al 1963, Singh 1971). ROL is transmitted in a persistent manner by the leafhopper vector. It is likely that ROLP multiplies in Rd but is not transmitted from Rd females to their progeny via eggs. Rd acquires ROLP in a 3-hour access feeding and become infective after an incubation period of 19-27 days at 17°C, 13-23 days at 25.7°C, and 6-15 days at 28.5°C (Lin et al 1983). ROLP-viruliferous Rd transmitted ROLP to rice seedlings in a 30-min access feeding. In Rd populations exposed to ROL, 8 to 68.9% of planthoppers become infective of ROL.

1.6. Diagnosis and serology

Diagnosis of ROL in the field is primarily based on symptoms but it is not always conclusive since yellow orange discoloration of plants may also be caused by infections with other diseases including tungro, grassy stunt, transitory yellowing, and RYD. ROL is generally diagnosed based on a transmission test using Rd population collected from affected fields. Diagnosis based on DNA hybridization or PCR specific to ROLP-DNA has not been adapted. DNA probes derived from plasmid of aster yellow phytoplasma hybridized with ROLP-DNA and phytoplasmas of aster yellow, onion yellow, and paulownia witches' broom, indicating that ROLP and these three phytoplasmas in Asia contain plasmids related to that of aster yellow phytoplasma (Nakashima and Hayashi 1995).

Serology is not available for ROL.

1.7. Disease cycle and epidemiology

ROL is generally sporadic and does not cause conspicuous yield loss. Epidemiology of ROL and ecology of ***R. dorsalis*** in relation to ROL are not known.

1.8. Cultivar resistance

Seedlings of cultivar Kalu Dahanala inoculated by using ROLP-viruliferous Rd showed lower percentage infection than other tested cultivars with milder symptoms (Abeygunawardena et al 1970).

1.9. Control

ROL is generally sporadic in the field and the yield reduction it causes is negligible. Control measures are not taken specifically for ROL.

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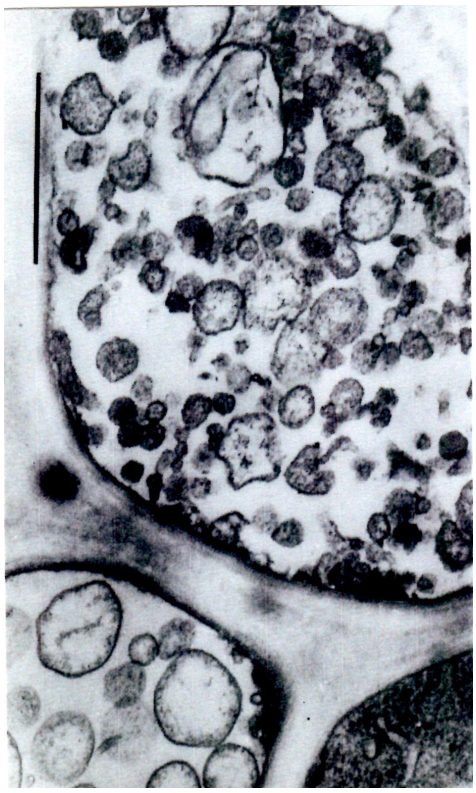
2. Yellow dwarf (RYD); pathogen: RYDP

2.1. History and distribution

Yellow dwarf of rice (RYD) characterized by chlorosis of leaves and plant stunting (**RYD Figure 1**) occurs widely in Asia (Ling 1972, Ou 1984). In China, it occurs in Fujian, Hainan, Guandong, Guangxi, Hubei, Jianxi, Chejian, Sghanghai, Jiangso, Anhui, and Yunnan (Xie et al 1994, Lin et al 1985). It is generally sporadic and occasionally reaches epidemic proportions in areas under intensive cropping systems (Hibino 1992, Iida 1969, Ling 1972, Ou 1984).

RYD Fig. 1. A rice plant infected with yellow dwarf.



RYD Fig. 2. Sections of phloem tubes of an orange leaf-infected leaf vein showing numerous phytoplasma bodies (x 32,000). Source: Hibino et al (1987).

RYD was first recognized in the 1910s at Kochi Prefecture in southwestern Japan where double cropping of rice was practiced (Iida 1969). In 1943, the Kochi Experimental Station reported possible transmission of RYD by the rice green leafhopper ***Nephotettix cincticeps*** Uhler (Nc) (**Intro Figure 2**). A later report (Enjoji 1948) further indicated involvement of Nc as a vector for RYD. Transmission of RYD in a persistent manner by Nc was demonstrated precisely (Iida and Shinkai 1950, Shinakai 1962). ***N. virescens*** (GLH) (**Intro Figure 4**) (Shinakai 1959) and ***N. nigropictus*** (Nn) (**Intro Figure 5**) (Ouchi and Suetsugu 1963, 1964; Shinkai et al 1962) also transmit RYD.

In Japan, RYD was initially localized in the coastal areas in southwestern Japan for about half a century. After the mid1950s, RYD spread widely to southern, southwestern, western, and central regions with intensification of rice cultivation by the introduction of early rice and early planting of middle rice (Hirao and Inoue 1978, Iida 1969, Kiritani 1983). During 1962-64, RYD incidence was especially high (Hirao and Inoue 1978, Kiritani 1983). Areas affected with RYD covered 199,000 hectares in 1962, 182,000 in 1963, and 125,000 in 1964. RYD incidence greatly decreased in southern Japan after 1968, while it gradually decreased to a low level in central Japan by the end of 1980s (Kiritani 1983). On the southern islands of Okinawa, RYD was sporadic since 1956 and became epidemic in 1961-62 (Shinkai et al 1963). RYD incidence is low in western and southwestern Japan, while sporadic or none in central Japan. It is absent in northwestern and northern Japan.

In Taiwan, RYD has been known since 1925 and has caused considerable damage (Kurosawa 1940; Chiu 1964, 1966). The occurrence of RYD was confirmed by transmission tests using Nc, GLH, or Nn in Hainan (Hashioka 1952) and Fujian, China (Lin and Xie 1985),

India (Raychaudhuri et al 1967a,b), Indonesia (Satomi et al 1978), Malaysia (Lim 1970, Lim and Goh 1968), the Philippines (IRRI 1963, Palomar and Rivera 1967), Sri Lanka (Abeygunawardena et al 1970), and Thailand (Wathankul and Weerapat 1969). It occurs widely in rice–growing areas of Asia except in areas at high latitudes and altitudes.

As RYD is transmitted in a persistent manner by leafhoppers as do rice dwarf virus and other rice viruses, it has long been thought to be a virus and efforts to identify the virus agent were unsuccessful. In 1967, soon after the discovery of mycoplasmalike organisms (MLO) in several witches' broom and yellowing diseases of plants (Doi et al 1967), similar studies on RYD-infected rice plants showed pleomorphic bodies in their sieve tubes of (Shikata et al 1968). Similar bodies were also found in the salivary glands and midguts of RYD-viruliferous Nc (Nasu et al 1967). Tetracycline antibiotics effectively reduced the severity of symptoms on RYD-infected rice plants (Ishiie et al 1967; Sakurai and Morinaka 1968; Sugiura et al 1968, 1969b). The association of phytoplasma bodies with RYD-infected rice plants and RYD-viruliferous leafhopper vectors, and suppression or delay of the symptoms expression in infected plants by the antibiotics indicated that RYD is of phytoplasma origin, although the Koch posturation has not been fulfilled. Rice yellow dwarf phytoplasma (RYDP) has not been cultured in artificial media.

2.2. Symptoms

Rice seedlings infected with RYD showed symptoms 27-40 days after the inoculation (Goto 1952, Iida 1969, Iwahashi and Goto 1964, Kiryu 1952, Kurata 1945, Ling 1972, Ou 1985, Shinkai 1962). Infected plants showed general chlorosis, pronounced stunting, and profuse tillering. Chlorotic leaves are uniformly pale green or pale yellow (**RYD Figure 1**). The discoloration first appears on newly developing young leaves and all the succeeding leaves show chlorosis, become soft, and droop slightly. Profuse tillering and plant stunting appear on plants 60-90 days after the inoculation (Nakashima and Hayashi 1995a). Infected seedlings are generally alive until maturity. Infected plants produce no panicles or, if produced, plants bear a few, small panicles with mostly unfilled grains. After the rice harvest, infected ratoons develop new leaves with chlorosis and yellowing symptoms. In temperate regions, infected ratoons do not overwinter except in warm regions in warm years. .

Development of the symptoms is delayed in plants infected at later growth stages or in plants grown under lower temperatures (Iwahashi and Goto 1964, Shinakai 1962). The Incubation period for the symptoms in infected rice plants is about 1 month in plants inoculated at the 10- to 13-leaf stages. Plants infected at 2 months after seeding or later did not develop symptoms on standing plants, but developed symptoms after the harvest on ratoons. In another study, plants inoculated at younger than the 13-leaf stage developed symptoms, while plants inoculated at the 14 leaf stage or later did not develop symptoms on standing plants, but do on ratoons (Shinkai 1962).

In the field in Japan, all plants inoculated during 1 April to 15 August developed symptoms. In India, seedlings of cultivar MR-277 infected with RYD at 1 to 1.5 months before the harvest did not show clear symptoms, but developed tillers with symptoms from lower nodes (Muniyappa 1981).

In Taiwan, seedlings of TN 5 and Taichung 186 infected with RYD at 30 days or younger stages showed distinct stunting and severe reduction in yield, while seedlings inoculated at 50 days or later showed a little reduction in height and in yield (Chen and Ko 1975).

RYD-Infected rice plants have pleomorphic phytoplasma bodies in the sieve tubes of leaf blades, sheaths, stems, and roots (**RYD Figures 2** and **3**) (Chen 1978, Chen and Liu

1974, Lin and Xie 1985, Nasu et al 1967, Lin and Xie 1985, Saleh et al 1978, Satomi et al 1978, Singh et al 1970, Sugiura et al 1968). The bodies are bounded with a cell membrane but have no cell wall. They are 40-1200 µm in diameter, are spherical and irregularly ellipsoidal or elongated in shape, and have ribosome particles along with the membranes and DNA strands in the central portions. Bodies in young leaf tissues are generally smaller than those in aged leaf tissues, and filled with ribosome granules (Chen 1978). In infected rice plants, phytoplasma bodies are most abundant in the third youngest leaves, but limited in the first and second youngest leaves (Chen 1978). Bodies often occur abundantly near the sieve plates and in or around the sieve pores (Chen and Liu 1974). In RYD-infected plants cured with tetracycline antibiotics, phytoplasma bodies are scarce (Chen and Liu 1974).

Phytoplasma bodies similar to those found in infected rice tissues occur in the salivary glands, midguts, Malpighian tubes, and mycetomes of RYD-viruliferous Nc and Nn (Chen 1978, Nasu et al 1967, Sugiura et al 1969a).

Fat bodies of Nc exposed to RYD-infected plants show nucleus larger than those in unexposed leafhoppers at 10-15 days after the exposure (Takahashi and Sekiya 1962).

Symptom development on RYD-infected rice plants are delayed and milder when they are grown under the presence of tetracycline antibiotics (Chen 1978; Chen and Liu 1974; Galvez and Shikata 1969; Lin and Xie 1985; Muniyappa and Ramakrishnan 1976a; Sakurai and Morinaka 1968, 1970; Singh et al 1970; Sugiura et al 1968, 1969b). ***N. cincticeps*** exposed to RYD-infected rice plants showed a longer incubation period when leafhoppers were fed on or injected into the abdomens with tetracycline antibiotics.

Attempts to isolate in pure form rice yellow dwarf phytoplasma (RYDP) from infected rice plants or RYD-viruliferous Nc, or to culture in media were not successful (Katsuhara et al 1991, Nasu and Sugiura 1974).

RYDP in infected rice plants and leafhopper vectors is quantified by dot hybridization using peroxidase labeled RYDP-specific DNA probes derived from chromosomal DNA based on absorbance values on the dot spots (Nakashima and Hayashi 1995a). RYDP was detected in RYD-infected rice leaves first at 23 days after the initiation of 3 days acquisition access feeding. Relative amounts of RYDP increased rapidly to 33 days and leveled off to 43 days up to 83 days. There was no definite correlation between the amount of RYDP and the level of chlorosis determined by assessing chlorophyll contents. In Nc that was exposed to RYD-infected rice plants for 3 days, RYDP was first detected at 13 days after the initiation of the exposure and increases the amount at 33 to 43 days. Nc gave strong signals in the abdomen and the thorax at 28 days, while give signals in head parts at 53 days.

Tetracycline antibiotics including acromycin, aureomycin, and tetramycin, suppressed or delayed symptom expression in RYD-infected seedlings, when applied to rice seedlings through the roots shortly after the inoculation with RYD (Asuyama and Iida 1973; Chen 1978; Chen and Liu 1974; Galvez and Shikata 1969; Lin and Xie 1985; Muniappa and Ramakrishnan 1976a; Sakurai and Morinaka 1970; Singh et al 1970; Sugiura et al 1968, 1969b). RYD-diseased plants developed greener leaves and showed signs of recovery when treated with tetracycline, but the symptoms reappeared after stopping the application. Treatment of RYD-diseased plants with the antibiotics may mitigate the symptoms, but did not cure RYD in plants. Treatments of RYD-infected plants at the roots for 1 day at 0, 5, 10 or 15 days after the infection delayed the development of symptoms, while treatment at 25 days did not affect symptom development (Sugiura et al 1969b). Continuous treatments of RYD-infected plants at the roots at 0 or 5 days after the inoculation and thereafter suppressed the symptom development for 70 days. Foliar application of antibiotics on alternate days had certain effects on symptom development. When Nc was injected with the antibiotics 1 to 9 days after the acquisition-access to RYD-infected plants, the incubation period in Nc for infectivity was prolonged. When Nc were fed on sucrose

solution containing tetracyclines through a membrane after an access to RYD source plants, fewer Nc became infective in comparison to Nc fed on water (Sakurai and Morinaka 1970).

Symptom development in RYD-infected plants was suppressed and transmission rates of RYD-viruliferous Nc were reduced under high temperature (Takasaki et al 1970).

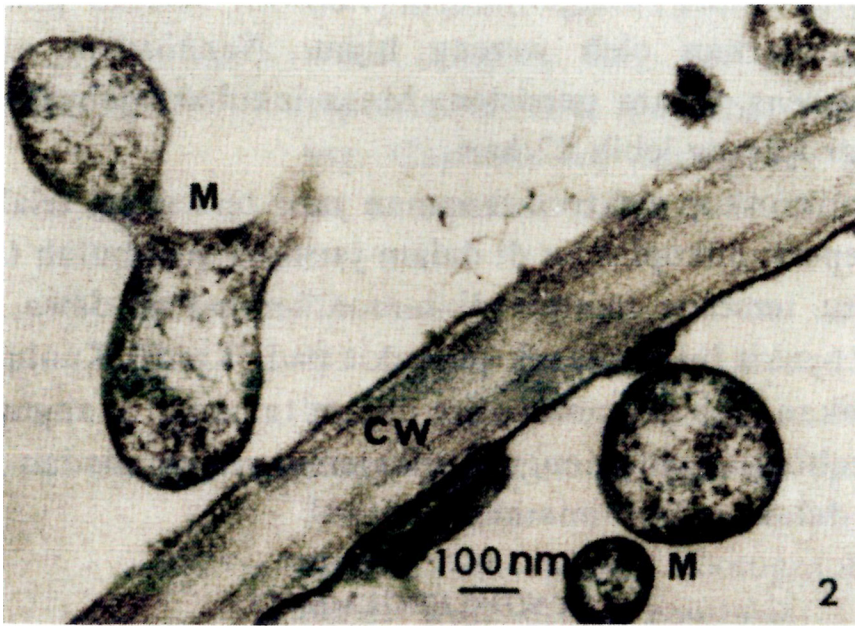
2.3. Phytoplasma

RYDP is the type member of the rice yellow dwarf group in the classification schemes based on restriction fragment length polymorphism (RFLP) analysis on 16S ribosomal RNA and ribosomal protein gene sequences (Davis and Sinclair 1998; Lee et al 1998a,b, 2000; Namba et al 1993a).

RYDP is pleomorphic with an average diameter ranging from 200 to 800 µm and bounded with unit membranes 8 nm in thickness (**RYD Figure 3**). RYDP bodies contain ribosome granules and DNA-like strands, and appear to arise in binary fission (Chen 1978, Chen and Liu 1974, Nasu et al 1967, Lin and Xie 1985, Saleh et al 1978, Satomi et al 1978, Shikata et al 1968, Singh et al 1970, Sugiura et al 1968).

Infectivity of phytoplasma in leaf sap can be assayed by injecting the sap into the abdomen of phytoplasma-free Nc nymphs through glass capillaries and testing the injected leafhoppers for RYDP infectivity (Kawakita et al 1991; Sugiura et al 1968, 1969a). The infectivity in RYDP-infected rice leaves and its extracts can be preserved in phosphate buffer solution containing 0.03 M $MgCl_2$, Na_2SO_3 and sucrose in liquid nitrogen (Kawakita et al 1991). Infectivity in RYDP-infected Nc and extracts from RYDP-infected plants can also be preserved similarly in glycine buffer containing $MgCl_2$ and sucrose.

RYDP can be maintained for a certain period in culture media with no definite signs indicating multiplication of RYDP (Nasu and Sugiura 1974). Phloem sap was collected from RYD-infected rice plants by cutting with a laser beam the stylets of brown planthoppers which were feeding on rice culms (Kawabe et al 1980, 1991). The sap obtained contained fluorescent particles of 20-30 nm in diameter in 4′, 6′-diamidino-2-phenylindole (DAPI)



RYD Fig. 3. Sections of phloem tubes in a yellow dwarf-infected leaf vein showing phytoplasma bodies (x 90,000). Source: Satomi et al (1978).

stain specific for animal mycoplasma. In PCR amplification using mollicute-specific or phytoplasma-specific primer sets, the presence of RYDP in the sap was confirmed (Sato et al 1993, 1997). The presence of RYDP in culture media containing the sap was also confirmed after two passages of cultivation (Katsuhara et al 1991).

RYDP-DNA was separated from host DNA after repeated bisbenzimidazole-CsCl equilibrium density gradient centrifugations (Nakashima et al 1991). Fragmented DNAs obtained were cloned, labeled with peroxidase, and used as probes in hybridization analysis. Two specific DNA proved one for RYDP chromosomal DNAs and one for extrachromosomal DNAs were used in detecting RYDP in rice and leafhopper vectors and differentiating RYDP from other phytoplasmas (Nakashima and Hayashi 1995a,b; Nakashima et al 1991). Extrachromosomal DNA was circular with a major length at 1173 nm and with the size of 4 kb, indicating that the DNA is a plasmid (Nakashima and Hayashi 1995b). In dot hybridization, extrachromosomal DNA probes for RYDP isolates in Japan and Thailand hybridized with DNAs from Bermudagrass white leaf phytoplasma in Thailand and weakly with DNAs from phytoplasmas of sugarcane white leaf, Brachiaria white leaf, and Dactyloctrnum white leaf in Thailand (Nakashima and Hayashi 1995b). Extrachromosomal DNA probes for sugarcane white leaf phytoplasma in Thailand hybridized with DNAs from RYDP in Japan and Thailand. Also, Chromosomal and extrachromosomal DNA probes from sesame phylody phytoplasma hybridized with RYDP in Japan and Thailand (Nakashima et al 1995). RYDP isolates from Japan, the Philippines, and Thailand harbored extrachromosomal DNAs with high sequence homology to each other (Nakashima and Hayashi 1995b). Biological functions of these DNAs are not known.

Symptomatic strains are not known for RYDP. Reactions of six rice cultivars to RYD isolates from Indonesia, Japan, and the Philippines did not show evidence indicating the presence of different strains (Asaga and Furuta 1974, Asaga et al 1977, Morinaka and Sakurai 1970). On the other hand, recent studies on the RYDP genome show that RYDP is diverse. Extrachromosomal DNA probes from a RYDP isolate from Japan hybridized with DNAs from RYDP isolates from different regions in Japan, and isolates from the Philippines and Thailand (Nakashima and Hayashi 1995b). The electrophretic mobility of extrachromosomal DNAs was variable among RYDP isolates collected from different locations, even among isolates collected from plots in about 100 m^2 (Nakashima and Hayashi 1995b). Whereas DNA products obtained using chromosomal DNA probes did not show any variation among RYDP isolates tested. Chromosomal DNA probes for RYDP isolate from Japan detected RYDP-DNAs from rice tissues infected with a Philippines isolate up to 60 µg (Nakashima et al 1992).

Based on sequence data on the 16S ribosomal RNA gene, phytoplasmas were divided into 4 groups. Primer sets for analysis by polymerase chain reaction (PCR) were obtained for on mollicutes, phytoplasmas and specifically for phytoplasma Group I or Group III (Namba et al 1993a,b). RYDP-DNA can be differentially amplified by combining two primer sets either for Mollicutes and for phytoplasma or for group III.

Tetracycline antibiotics including acromycin, aureomycin, and tetramycin, suppressed or delayed symptom expression in RYD-infected rice seedlings, when applied to rice seedlings through the roots shortly after the inoculation with RYD (Asuyama and Iida 1973; Chen 1978; Chen and Liu 1974; Galvez and Shikata 1969; Lin and Xie 1985; Muniyappa and Ramakrishnan 1976a; Sakurai and Morinaka 1970; Singh et al 1970; Sugiura et al 1968, 1969b). When Nc exposed to RYD-infected rice plant were injected with or fed on antibiotics through membranes, the percentage of Nc that became RYD infective was reduced and the incubation period for infectivity was prolonged (Sakurai and Morinaka 1970).

2.4 Host range

2.4.1. Japan. In Japan, seedlings of 30 gramineous plants are inoculated by RYDP-viruliferous Nc (Shinkai 1962). Beside rice, ***Oryza cubensis***, ***Alopecurus aequalis***, and ***Glyceria acutiflora*** can be infected with RYD and developed yellowing or chlorotic symptoms after long incubation periods of from 45 to 71 days. Using RYD-free Nc, RYDP was recovered from those infected weeds to rice seedlings. Two hundred plants each of ***A. aequalis*** collected from two fields infected with RYD were grown in the greenhouse for the symptoms and recovery tests to rice seedlings (Shinkai 1962). Eight ***A. aequalis*** plants from one field showed symptoms and gave a positive recovery. ***A. aequalis*** plants that were inoculated with RYDP and transplanted in fields developed symptoms in April to June. In other tests, RYDP was not recovered from ***A. aequalis*** plants showing RYD-like symptoms (Ishii et al 1969, Komori et al 1972). Instead, all the plants showing RYD-like symptoms were found to be infected with ***Sclerophthora macrospora*** (Komori et al 1972). Infection of ***A. sequalis*** with RYDP might occurred on rare occasions.

2.4.2. Taiwan. In Taiwan, seedlings of several wild rice species were individually exposed to RYD-viruliferous Nc. Five lines of ***O. barthii***, and two lines each of ***O. nivara*** and ***O. stapfii*** had infection with RYD and developed symptoms (Chen and Ko 1976a,b). One line of ***O. nivara*** had latent infection with RYD. In another test, 24 gramineous weeds were tested for RYD infection by RYD-viruliferous Nc (Chen 1977). Plants of ***A. aequalis***, ***Imperata cylindrical***, ***Leersia hexandra***, and ***Panicum repen***s had infection, showed yellowing symptoms, and gave positive recovery to rice seedlings by using Nc. Inoculated ***P. dilatatum*** plants showed similar yellowing symptoms, but gave negative recovery. In field surveys, plants showing yellowing symptoms were found for ***Digitaria setigera***, ***F. koizumiana***, ***Leptochlea chinesis***, and ***Paspalum distichum*** (Chen 1977). By the recovery test on these plants, natural infection of ***L. chinesis*** and ***P. distichum*** was confirmed. Natural infection of weeds with RYD likely occurs only rarely and appeared not important in the RYD infection cycle in Taiwan.

2.4.3. India. In India, plants of ***Oryza barthii, O. glaberrima, O. longistaminata***, and ***O. rufipogon*** exposed to RYD-viruliferous GLHs developed symptoms (Muniyappa and Raychaudhury 1988, Muniyappa and Ramakrishnan 1976b, Muniyappa and Raju 1981).

2.5. Transmission by vectors

RYDP is not transmitted through seeds in rice or through the soil. RYD is not mechanically transmissible in rice. RYD was transmitted in persistent manners by Nc (Iida and Shinkai 1950, Shinkai 1962), Nn (Ouchi and Suenaga 1963, 1964; Shinkai et al 1963), GLH (IRRI 1963; Shinkai 1959, 1962; Shinkai et al 1963), and ***N. malayanus*** (Nm) (Hirao and Inoue 1978). RYDP propagates in the leafhopper vectors, but is not transmitted from female adults to their progeny via eggs (Shinkai 1951). Nc is the principal vector in the temperate regions, Nc and Nn in semitropical regions, and GLH, while Nn in the tropical regions. Nc, Nn, and GLH acquire and transmit RYDP generally in high efficiencies with some local differences in their efficiencies (Asaga and Furuta 1974, Asaga et al 1977).

The efficiencies are 25-100% for Nc (Asaga et al 1977; Iida and Shinkai 1950; Chen 1970; Lin and Xie 1985; Ouchi and Suetsugu 1963, 1964; Shinkai 1962), 24-93% for Nn (Chen 1970; Ouchi and Suenaga 1963, 1964; Palomar and Rivera 1967), and 26-96% for GLH (Lim 1970; Lin and Xie 1985; Palomar and Rivera 1967; Shinkai 1959, 1962). Leafhoppers acquired RYDP minimum in a 10-min. access feeding, and transmited it minimum in a 1-3 min.-access feeding after a long incubation period of 20-47 days. Phytopathic effects of RYDP on the leafhopper vectors are not known.

2.6. Diagnosis and serology

Diagnosis based on characteristic symptoms is widely practiced in the field. Diagnosis by the transmission test using vector leafhoppers collected from affected fields has been adapted widely. When RYD-infected plants were cut back at 5-10 cm from the ground level, new leaves showed clear symptoms. Diagnosis can be done on newly developed leaves after cutting back the stems. Molecular-based detection methods by DNA dot hybridization and PCR amplification have become available. PCR using primer sets specific for the amplification of 16S ribosomal RNA genes for molicutes, phytoplasma or phytoplasma group III effectively amplified RYDP-DNAs (Namba et al 1993a,b). Two cycles of PCR using the universal primer set for mollicutes and the primer set specific for phytoplasma group III (step 2) was adapted on DNA extracts from RYD infected rice tissues. Amplified PCR products were detected in agarose gel electrophoresis as a composite band.

By DNA hybridization, DNA probes labeled with peroxidase detected RYDP-DNA in RYD-infected rice tissues and in RYD-viruliferous Nc. (Nakashima and Hayashi 1995a,b; Nakashima et al 1991, 1992). Probes for extrachromosomal (plasmid) DNA detected RYDP at a minimum 7 µg of infected leaf tissues, while probes for chromosomal DNA detected RYDP at a minimum of 31µg of tissues. The reactions of host DNAs with the DNA probes were very weak. By the dot hybridaization, the relative amount of RYDP can be quantified by measuring the strength of reactions using the colorimeter (Nakashima et al 1995).

Serological diagnosis is not available for RYDP. Since early days when the RYD agent was not known, attempts to purify the disease agent from infected rice plant or ***N. cincticeps*** and to produce antiserum have been done with no comprehensive success (Komori 1966, Seki and Onizuka 1965, Takahashi et al 1964, Takahashi and Kuroiwa 1965).

2.7. Disease cycle and epidemiology

RYD is generally sporadic in the field. It requires a very long incubation period in rice plants for symptom expression and also in the leafhopper vectors to gain infectivity especially in cool seasons. So, dispersal of RYD from primarily infected rice plants to surrounding plants is very slow in the field. The disease cycle of RYD is rather fragile and easily interrupted either in single or double cropping rice fields.

RYDP is transmitted by leafhopper vectors in the field. The principal vector for RYD is Nc in the temperate regions, Nc and Nn in the semitropics, and GLH and Nn in the tropics. In temperate regions, leafhopper vectors that acquired RYDP on RYD-infected rice plants, rice stubble, or voluntary rice plants overwinter as nymphal diapauses and serve as the RYD source for newly planted rice fields. Ratooned rice crops infected with RYD develop new leaves after the harvest, serve as a disease source in the early winter season and are generally wilted during the winter season (Hirao and Inoue 1978). In the semitropics and tropics, infected rice plants including rice stubble and voluntary rice plants are the primary sources for the new crop season. The weed ***A. aequalis*** is an important host of Nc during the winter season and is naturally infected with RYD (Chen 1977, Shinkai 1962). Infected ***A. sequalis*** plants overwinter and develop symptoms in May or June, although the incidence is very low. It is likely that ***A sequalis*** has a limited role in RYD epidemics (Chen 1977, Komori et al 1972, Shinkai 1962). Leafhopper vectors overwinter on ***A. sequalis*** and other weeds in fallow rice fields, in rice fields planted for ***Asttragalus sinicus***, or other manure crops on levees or along irrigation canals (Hirao and Inoue 1978, Ishii et al 1969). At Ibaraki, central Japan, the density of overwintered Nc population had a high correlation with density of ***A. sequalis*** (Kimizaki and Takano 1969).

During the winter season in Japan, Nc feeding on RYD-infected rice stubble in October to December became infective in late April to early May. In RYD-infected fields,

the percentage of RYDP-infective Nc in overwintering populations was generally higher in April and May than in earlier months (Hasuko et al 1965, Komori et al 1972, Yasuo et al 1963). In RYD epidemic areas, proportions of RYD-infective Nc in overwintered populations were 1-10% at Kagoshima in 1963 (Itoga and Babaguchi 1963), 4-26% at Ibaraki in 1963 (Matsunaga et al 1973), 1-21% at Saitama in 1963 (Yasuo et al 1963), and 5-39% at Miyazaki in 1964 (Iwahashi et al 1964). Adults of the overwintering generation appeared in March to April, moved to early-season rice, and survived until early or mid-June. Major infection of rice occurred in transplanted fields by the overwintering generation in April in warm regions and early May to late June in cooler regions, by the second generation in July to August, and by the third generation in August to September (Itoga and Babaguchi 1963, Komori 1966, Komori et al 1972, Miyahara and Yamaguchi 1965, Mori et al 1965, Shinkai et al 1963, Yasuo et al 1963). RYD symptoms appeared in July in plants infected in early May, in July to August in plants infected in June to July, while on ratoons after the harvest in plants infected in August to September (Goto et al 1965, Komori 1971). In traditional cropping systems, rice is transplanted in June and therefore the disease cycle for RYD is hardly completed and RYD incidence is generally sporadic or zero.

RYD incidence is generally high in early-season rice, while low in late-planted rice (Kimizaki and Takano 1969, Komori et al 1972, Takano et al 1963). At Ibaraki, central Japan, early planting on 9 May had a 52% infection with RYD in standing rice plants, semi-early planting on 30 May had 16% infection, while late planting on 26 June had no infection. The formula for predicting RYD incidence was obtained at Miyazaki in southern Japan and at Ibaraki in central Japan based on the percentage of viruliferous leafhoppers in overwintering populations and RYD incidences scored on ratoons of the previous crop (Goto et al 1975, Hirao and Inoue 1978, Komori 1964, Sato and Sugino 1965).

In Okinawa, Japan, the principal vector is Nn (Shinkai et al 1963). RYD occurred around 1956, increased in incidence by 1961, and reached an epidemic proportion in 1962. In 1962, RYD incidence was 20% in standing rice crops transplanted in January, 36% in rice transplanted in mid-June to early July, and 10% in plants transplanted in late August to early September.

In Taiwan, Nc, Nn, and GLH occur in rice fields throughout the year (Chen 1977, Chen and Ko 1978). Leafhoppers density in the field was high in May to July and December to January. RYD-infective leafhoppers occurred throughout the year in the field. The proportions of RYD-infective leafhoppers in overwintering populations were from 9 to 34% (Chen 1977). Infected rice stubble and voluntary rice plants also overwinter, although some infected stubble is wilted during the cool months. Infected rice stubble can serve as disease sources for the first crop rice. Infection also occurs in rice if planted very early in the winter season. Rice stubble and ***A. aequalis*** served as important hosts for Nc and Nn during the winter season (Chen 1977). ***A. aequalis***, ***L. hexandra*** and ***L. cylindrica***, are sensitive to RYD in greenhouse tests, though natural infection of these weeds in the fields is limited. These weeds might have limited role in RYD epidemics.

In southern China, leafhopper vectors overwinter largely in fallow rice fields on rice stubble and ***A. sequalis***, which serve as important hosts of Nc and Nn during the winter season.

In the tropics, RYD incidence is generally low or zero. Infected plants rarely develop symptoms in standing crops, but can develop symptoms after the harvest on ratoons.

RYD is generally sporadic and causes a little loss. However in Japan, the incidence increased to a high level in 1953 in southern Japan with the increase in areas planted for early season rice (Samejima 1967, Shintome and Itoga 1962). Thereafter, severe infestation of RYD expanded to north and inland. After 1958, severe infestation occurred widely in central and western Japan and total areas affected with RYD in Japan reached 199,000

hectares in 1962, 182,000 in 1963 and 125,000 in 1964 (Hirao and Inoue 1978, Kiritani 1983). Areas affected in Kanto region of central Japan alone were 105,000 hectares in 1962, 83, 000 in 1963, and 66,000 in 1964 (Ishii et al 1969).

The RYD epidemics during 1960-80 was attributed to the introduction of early planting of middle rice and early-season rice, allowing leafhoppers, which acquired RYD in infectedfields in the previous crop season or after the harvest, overwinter and moved directly to newly transplanted early season rice (Kiritani 1983, Shinkai 1962). Early planting of rice increased rice yield and ensured rice production by avoiding damage due to typhoons in September and October. The epidemics after 1991 have been largely attributed to reduction in insecticide applications, lack of manpower to manage rice fields, and increase in abandoned fields**.**

2.8. Losses

Yield losses in rice due to RYD infection vary greatly with symptom severity on rice plants (Ishii et al 1969). Generally, the loss is greater in plants that develop symptoms at earlier growth stages (Mori et al 1965). Yield losses in plants with severe or moderate infection were 57 to 97%. In warm areas of central Japan, the losses were 26% in plants that developed symptoms at the tillering stage, 30% in plants developing symptoms at the panicle formation stage, 12% in plants developing symptoms at the booting stage, and 11% in plants developing symptoms at the maturing stage. In cooler areas, the losses were 37.4% in plants developing symptoms before the heading stage, 25.6% in plants developing symptoms at the early yellow-ripe stage, and 16.2% in plants developing no symptoms but developing symptoms on ratooned crops (Komori 1965, Komori et al 1972).

In fields transplanted at one plant per hill, yield losses in plants that developed symptoms in standing crops was 64.5-100%, and those in plants that developed no symptoms in standing crops but developed symptoms on ratoons was 31.5%, while in fields transplanted at 2-3 plants per hill, yield losses in plants that developed symptoms in standing crops was 23.6-47.7% (Iwaki et al 1969).

Yield loss (Y) of plants with a percentage of RYD-infected stems (X) is formulated as $Y=0.7250 + 0.2976 X$ (Kureha et al 1974). Y in plants with percentage infection (X) in ratoons is formulated as $Y= 1.520 + 0.2716 X$, and Y in plants with percentage of RYD-diseased panicles (X) as $Y= -2.4072 + 0.2644 X$ (Kureha et al 1974).

Under the subtropical conditions in Taiwan, percent yield reduction (Y) has been estimated based on plant age (days after transplanting) (X) when the symptoms appear as $Y=777.0593 – 12.2015 X +0.04855 X^2$ for the first crop and $Y=37.26395 + 4.96484 X – 0.00058 X^2$ for the second crop (Chen et al 1977).

In another estimate, yield loss (Y) is obtained based on plant age (days after seeding) (X) as $Y= 59.9803 – 0.5001 X$ in the first crop and $Y=104.07 – 1.24 X$ in the second crop (Hsieh 1976).

Under the tropical conditions in India, yield loss was 100% in plants infected 25 days after transplanting, while it was 10.7% in plants infected 100 days after transplanting (Muniyappa 1981).

2.9. Cultivar resistance

As early as 1932 and 1946 in Taiwan, cultivar resistance to RYD was tested in the field (Kurosawa 1940, Hashioka 1952). Rice cultivars from Japan were mostly susceptible, while a number of indica cultivars and cultivars from Taiwan showed resistance to RYD. In field tests in Japan, japonica glutinous cultivars Kagura Mochi, Nagazane Mochi, and Saitama Mochi 10; nonglutinous cultivar Oou No. 263; and foreign cultivars Kaladumai, Loktjan,

Pe-bi-fun, Russia No. 33, Tadukan, and Te-tep showed resistances to RYD (Asaga et al 1976, 1977; Komori and Takano 1964; Komori et al 1972; Morinaka and Sakurai 1969, 1970).

In the 1970s, a greenhouse screening system for cultivar resistance to RYD was adapted in Japan (Asaga et al 1976, 1977; Morinaka and Sakurai 1970; Sakurai 1969). Rice seedlings at the 2-leaf stage in containers were exposed to RYD-viruliferous Nc in cages, transplanted in trays to grow for 4 weeks, the stems cut back to 5 cm high from the base, and scored for infection based on the yellowing symptoms on newly developed leaves 7-10 days after the cutback (Morinaka and Sakurai 1970, Sakurai 1969). The results obtained in the greenhouse tests showed certain correlations with the results obtained in the field tests. In the greenhouse tests, Te-tep, Russia No. 33, and glutinous cultivars Kagura Mochi, Mangetsu Mochi, Naozane Mochi, Saitama Mochi 10 and Shinano Mochi 3 showed resistances (Asaga et al 1976, 1977; Morinaka and Sakurai 1970). None of japonica nonglutinous and japonica upland rice cultivars tested showed resistance. Many of rice cultivars that were scored as resistant in earlier tests (Hashioka 1952, Komori and Takano 1964) fell into susceptible categories. When cultivar reactions to RYD isolates from Indonesia, Japan, and Philippines were compared, cultivar reactions were basically similar, although the incubation period in infected plants was somewhat different among isolates (Asaga et al 1977). The resistance in Saitama Mochi 10 was controlled by a dominant or incomplete dominant major gene (Morinaka et al 1970).

In Taiwan, cultivar resistance to RYD was conducted in the greenhouse using Nc or Nn (Chen 1975; Chen and Ko 1976a,b; Chen et al 1972; Chiu et al 1970; Lin et al 1975, 1980). Among 70 cultivars tested, Taipei 131 and Norin 49 had no infection, while Taipei 310 had a low percentage of infection with mild symptoms (Chiu et al 1970). In another test using 1,430 cultivars and lines, 113 showed high levels of resistance (Chen et al 1972). Further evaluation on selected cultivars in the field showed that C_4-63 from Thailand, Firooz-1 from Iran, and Kabara from Sierra Leone showed strong resistances—Ian-chu-tzu (A), Ian-chu-tzu (B), Firooz-4, 581A6-545, 4bs-b-1, Blue-bell, Meher (1), Gangala, and an upland line showed high levels of resistance in the field, whereas moderate resistances in the greenhouse testsSeveral cultivars were tolerant of RYDP, developed symptoms only on ratooned crops and suffered a little reduction in yield. Kagura Mochi, Saitama Mochi 10, Belle atus, Blue Bonnet, Te-tep, and Yang-sien-tao, which scored as resistant in Japan did not show resistance in Taiwan. In another test using 2,610 cultivars including ***Oryza nivara, O. barthuii and O. stafii***, cultivars Kabara, C_4-63A, Blue-bell, Faya, IR1487-194-5-3-2, 4bs-6-1, B581A6-545, 1531R-22(SI), MTU 1, IR Early 773, IR994-102-2-3-2, and two lines of ***O. nivara*** showed high levels of resistance to RYD (Chen and Ko 1976a). In the greenhouse and field, none of Firooz-1 plants inoculated with RYD developed symptoms, although some of them gave positive recovery of RYD to rice seedlings by Nc (Chen and Ko 1976b).

Selected cultivars having resistances to RYD and/or Nc were tested for feeding behaviors of Nc on rice plants (Chen and Ko 1976b). Cultivars Firooz-1 and Kabara having resistance to RYD showed nonpreference to leafhoppers, while resistant cultivars Ilan-chu-tsu A and Dee-chuch-chu-tsu show preference to leafhoppers. Vector-resistant cultivars Te-tep and Koa-sen-yu 12 were susceptible to RYD, while vector-resistant cultivars H 105 and MTU 1 were moderately susceptible to RYD. The correlation between resistance to Nc and to RYD in the greenhouse and field tests appeared to be limited (Chen and Ko, 1976b). The resistances to RYD in Firooz-1 and Kabara were controlled by single dominant genes (Lin et al 1975). Breeding lines having resistances from Firooz-1 or Kabare with good agronomic and grain qualities were obtained (Lin et al 1980**).**

In greenhouse tests using GLH for RYD resistance in India, cultivars MR-363, Gamasolu, and HY-256 had no infection (Muniyappa and Ramakrishnan 1976, Muniyappa and Raju 1981). ***Oryza alt*, *O. ausraliensis, O. brachyantha, O. eichingeri, O. grandiglumis, O. latifolia,***

O. minuta, O. nivara, O.officinalis, O. puntata, O. ridleyi also had no infection. In another test in India, IR62, IR64, and IET 7492 having resistance to GLH had low levels of infection and developed the symptoms after long incubation periods (Rao and Narayanasamy 1989. In a greenhouse test using RYD-viruliferous GLH in the Philippines, several IR cultivars had low levels of infection (Daquioag and Hibino 1985**).**

2.10. Control

RYD has been primarily controlled by applying insecticides to reduce vector population and cultivation practices. In Japan, major infection of rice with RYD was caused by the overwintering Nc generations and by the second generations, which newly acquired RYDP on infected rice stubble in the field (Hirao and Inoue 1978). Insecticide application in fallow rice fields to reduce overwintering Nc populations gave effective control of RYD (Hashizume 1963; Komori 1966, 1971; Komori et al 1972; Mori et al 1965; Samejima 1967; Shimoyama and Shibamoto 1966). The critical levels for applying insecticides for RYD control in late autumn or early spring was percentage RYD infection at 5% on ratoons of previous crops or percentage RYD carrying Nc in overwintering populations at 5% (Komori 1971).

Malathion was intensively used in the early 1960s. After development of resistance to malathion in Nc, carbamates and phosphorus insecticides were used. To increase the efficiency of control in an area, cooperative control by area-wide application of insecticides was practiced in severely infected areas (Iwaki and Matsumoto 1968). Aerial spray using helicopters has been widely practiced to manage leafhopper- or planthopper-borne viruses and RYD all together in rice (Hayashi and Komatsu 1964, Komori et al 1972, Miyazawa and Hayakawa 1964, Miyazawa et al 1961, Nakamura et al 1962, Takahashi et al 1968, Toyoda et al 1964).

Since the 1990s, aerial application of chemicals was diminished with the elevation of concerns on the effects of the chemicals on the environment. On the other hand, use of transplanting machines became popular after the 1960s and application of insecticides in seedling boxes for the transplanters become popular after the 1970s (Takai and Inou 1975, Yamazaki and Hariya 1978, Yasuzaka et al 1979). Aerial application and nursery box application of insecticides have also been practiced for controlling RYD and other rice diseases in Taiwan.

Plowing of fallow rice fields during the winter season have been widely practiced in Japan to reduce vector populations and controlling rice viruses and RYD (Kiritani 1983).

In Japan, areas affected with RYD gradually decreased after 1966 and declined to a very low level by 1980. The incidence increased again in northern parts of central Japan in 1991 to 2004. The incidence is generally low in southwestern Japan and very low or zero in central Japan.

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