5. Black-streaked dwarf (RBSD); pathogen: RBSDV and Southern rice black-streaked dwarf (SRBSD); pathogen: SRBSDV

(continued from Part II, Section 3, Chapter 2, p. 78)

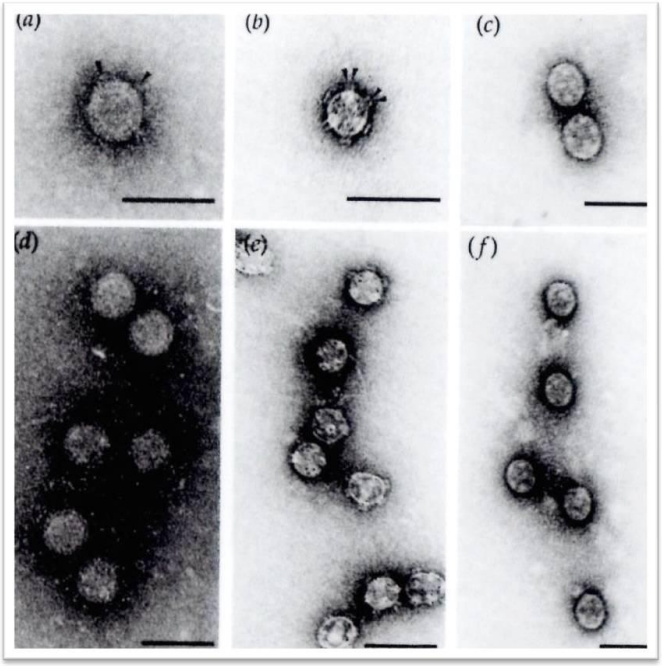
RBSD Fig. 1. A rice plant infected by RBSDV. Source: Masamichi Isogai.

5.1. History and distribution

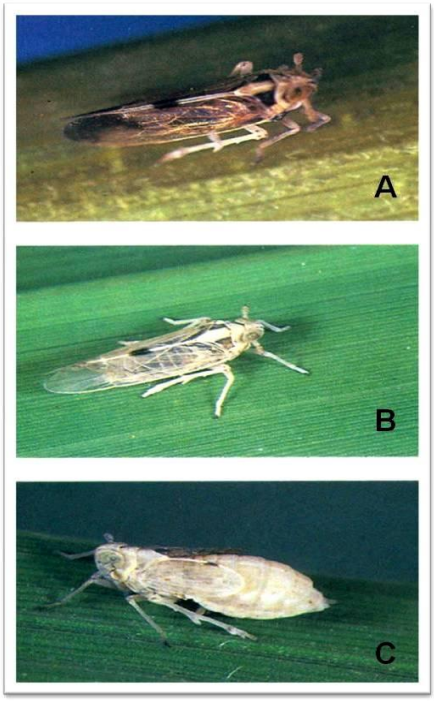
Black-streaked dwarf of rice (**RBSD Figure 1**) occurs in China, Japan, and Korea (Hibino 1992, 1996; Ling 1972; Ou 1985; Shikata 1974). It was first described at Nagano Prefecture, Japan, as a disease transmitted by the small brown planthopper (SBPH), *Laodelphax striatellus* Fallén (**Intro Figure 7**) in 1950 (Kuribayashi and Shinkai 1952). Besides rice, the RBSD agent also infects barley, maize, wheat, and other cereals, and causes “streak dwarf” (**RBSD Figure 2**). In Zhejian, China, RBSD occurred in 1963 and caused serious damage in rice, maize, barley, and wheat in the 1960s and again in the 1990s (Li et al 1979). In Korea, RBSD epidemics occurred at Sonsan in 1973 (Lee et al 1977). In 1969, polyhedral RBSD V (**RBSD Figure 3**) was isolated from RBSD-infected rice plants (Kitagawa and Shikata 1969a,b; Shikata 1969).

In 2001, a disease similar to RBSD occurred in Guandong, China (Zhou et al 2004). After 2005, the disease reached epidemic proportions widely in southern and central China (Zhou et al 2008, Wang et al 2010). The virus agent that differed from RBSDV occurred in China, Japan, and Korea was called southern rice black-streaked dwarf virus (SRBSDV) (Zhang et al 2008, Zhou et al 2008). SRBSDV was efficiently transmitted by the whitebacked planthopper (**RBSD Figure 4**), *Sogatodes furcifera* (Horvath), and less efficiently by SBPH, and shared relatively lower sequence homology with RBSDV isolates in Zhejian and Hubei, China, and in Japan (Zhang et

RBSD Fig. 2. Streaked dwarf symptoms on a maize leaf infected with RBSDV.



RBSD Fig. 3. Electron micrographs on a partially purified RBSDV fraction in uranyl acetate stain showing intact particles (a and d), B-spiked particles (b and e), and core particles (c and f). Single and double arrows indicate A and B-spikes, respectively. Bar scale: 200 nm. Source: Isogai et al (1999).



RBSD Fig. 4. The whitebacked planthopper has a characteristic white band on its thorax. The male (A) occurs only in the long-winged form. The females can be either long-winged (B) or short-winged (C). Source: IRRI.

al 2008, Zhou et al 2008). After 2008, it caused severe yield losses in China and in central and northern Vietnam (Hoang et al 2011), Wang et al 2010). In 2010, it occurred in Kyushu and two nearby prefectures in Japan (Higashi et al 2011, Matsumura and Sakai 2011, Sakai et al 2010).

In Japan, severe infestation of RBSD occurred first in Kanto Region and at Yamanashi and Nagano Prefectures. Probably, RBSD had long been present in central Japan (Shinkai 1957a,b,c). At Nagano, RBSD locally damaged rice in 1941 and during 1947-49 (Kuribayashi and Shinakai 1952, Obi and Kosuge 1956). Major outbreaks of RBSD occurred largely in maize at Yamanashi in 1956-57 (Obi and Kosuge 1956; Shinkai 1957a; Kosuge and Obi 1957a,b); in maize, wheat, and barley at Nagano and Yamanashi in 1961 (Shimizu et al 1961, Kosuge 1962); in paddy and upland rice, maize, wheat, and barley at Saitama and Tochigi in Kanto and at Nagano during 1965-68 (Ishii 1965, Ishii et al 1968, Suzuki et al 1968, Katayama et al 1968, Shinkai et al 1969); and in rice and maize in Kanto and at Yamanashi and Nagano during 1970-71 (Hongou et al 1971) and again in 1976-77 (Masuyama et al 1978).

Meanwhile, epidemics of stripe disease of rice (RS), which shares SBPH as the principal vector with RBSD, was high during 1955-70 and 1977-86 (Hibino 1996). Overlapped infestations in rice with RBSD and RS, as well as with leafhopper-borne rice dwarf and yellow dwarf

occurred frequently (Obi and Kosuge 1958). In the Tohoku Region north of Kanto, RBSDV damaged maize, but not rice, wheat, and barley (Mikoshiba et al 1985). At Asahikawa in Hokkaido Region, north of Tohoku, RBSD occurred in rice and maize, though the incidence was generally low (Uyeda et al 1993, Isogai et al 1995c). Since 1980 in Japan, RBSD incidence has been generally low in rice but often high in maize.

In China, RBSD first occurred in Zhejian in 1963, and reached epidemic proportions in 1965-66 (Wang et al 2009). After the epidemic, RBSD spread to northern, central, and southern provinces, though RBSD incidence was generally low thereafter for three decades. However, after 1996, severe epidemics occurred again in the middle and eastern Yangtze River Basin, and Fujian (Wang et al 2009). In later surveys in China, the disease occurred in Anhui, Zhejian, Fujian, Jiangso, Jianxi, and Shanghai (Xie et al 1994, Xie and Lin 2001).

In a survey in Korea in 1975, RBSD was found occurring widely except in the northern regions (Lee et al 1977). Another survey on rice viruses in Yeongnam area in 1981 (Park et al 1982) reported locally high incidences of RBSD, but low incidences of RS.

RBSD Fig. 5. White- or brown- or black-colored galls or vein swellings on the underside of rice leaves infected with RBSDV. Source: Tadashi Morinaka.

5.2. Symptoms

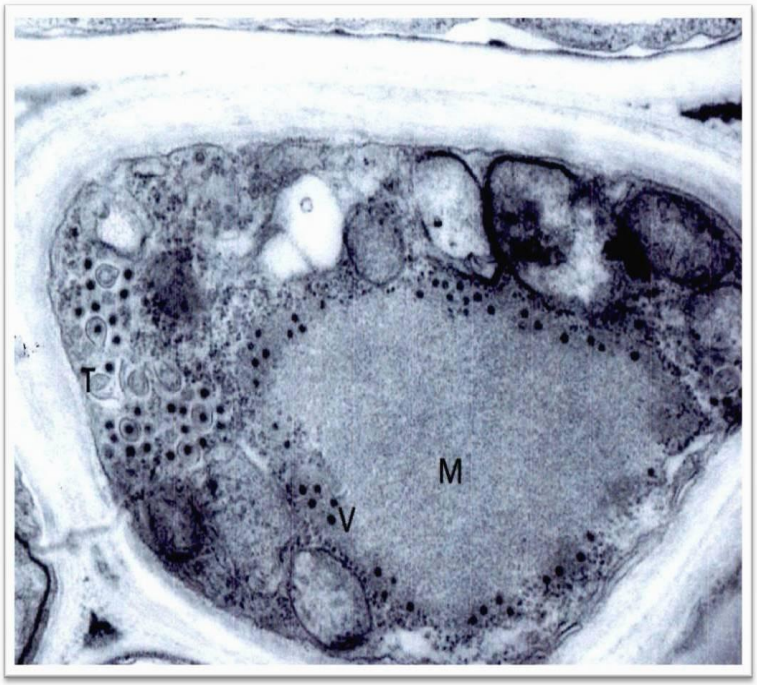
Infected rice plants show pronounced stunting, darker green color, twisting at the leaf tips, splitting of the leaf margin, galls or vein swelling along the major veins on the underside of leaf blades and the outer surface of the sheaths and columns, and develop up-growing root lets on the stem nodes (**RBSD Figure 1**) (Kuribayashi and Shinkai 1952, Shinkai 1962). The galls usually occur on older leaves as waxy white, irregularly elongated protuberance, which later turn brownish or blackish in color (**RBSD Figure 5**). Infected plants generally produce no panicles or, if any, a few panicles with unfilled or discolored grains. Infected plants generally retain green color up to the harvest time. In plants infected at the later tillering stage, symptoms may not

be clear until the maximum tillering stage, where plants show stunting, darkening of leaf color, and galls on leaves.

RBSDV susceptibility of rice varies depends on plant age (Shinkai 1958, 1962). Rice seedlings inoculated by RBSDV-viruliferous SBPH at the 3- to 12-leaf stages have a high percentage infection, while those inoculated at the 13- to 16-leaf stages develop no symptoms in standing rice crops. Seedlings, inoculated after the third tillering stage, have a low percentage infection and less damage (Li et al 1979 Wang et al 2009). Generally, rice plants infected at later growth stages develop milder symptoms and take longer incubation periods for the symptoms to show. In plants infected at the later growth stages, panicle exertion is delayed and often incomplete. The symptoms caused by SRBSDV in rice were similar to those caused by RBSDV. Vein swellings on SRBSD-infected plants might be less conspicuous and up-growing rootlets were not formed on the stem nodes.

RBSDV causes streak dwarf disease in barley, wheat, maize, rye, and oat. Diseased plants of those crops show stunting, darker green color, and swelling of the leaf veins, although the symptoms are milder in wheat (Obi and Kosuge 1963).

Galls on RBSDV-infected rice plants result from hyperplasia and hypertrophy of the phloem tissues (Shikata and Kitagawa 1977, Choi et al 1993b, Kashiwagi 1966). Amorphous inclusion bodies of about 6.5 μm in diameter occur in the cytoplasm of the gall cells (Kashiwagi 1966). The bodies are round or oval in shape, and stained with Giemsa, azur II, pyronin, trypan blue, or acetocarmine.



RBSD Fig. 6. Part of a phloem parenchyma cell of a rice root infected with RBSDV showing a viroplasmic matrix (M) containing virus core particles (V) in darker regions, and tubules (T) with virus particles (x 17.000).

In RBSD-infected rice plants, virus particles are limited in the phloem and gall tissues (Choi et al 1993a, Isogai et al 1999; Shanghai Institute of Biochemistry 1974, Shikata 1974, Shikata and Kitagawa 1977). In RBSDV-infected cells, virus particles of 75-80 nm in diameter are scattered or aggregated in or around viroplasmic matrices in the cytoplasm (**RBSD Figure 6**) (Choi et al 1983, Shikata 1974, Shikata 1969, Shikata and Kitagawa 1977). The

virus aggregates often form crystal-like arrangements. Viroplasmic matrices correspond to the amorphous bodies observed in the gall cells under the light microscopy. In electron microscopy, the matrices have two regions, dark-stained regions that consist of convoluted masses of strings and light-stained regions that consist of masses of intertwined kinky filaments as those reported for other members of the genus *Fijivirus*. (Milne and Lovisolo 1977). Particles of 50-55 nm in diameter, corresponding to subviral particles, are scattered in the dark regions but scarce or none in the light regions. Often, dark regions contain an aggregate of annular particles, which appear to be outer shells of RBSDV particles. Tubules containing single row of virus particles occur in the cytoplasm (**RBSD Figure 6**) and are 170-180 nm in width with a wall of 12-15 nm thickness. Tubules are often short in length with a few or no virus particles or appear as open scrolls with no particles.

In rice tissues infected with SRBSDV, virus particles occur in the phloem tissues (Zhou et al 2008). Virus particles were scattered or aggregated in or around viroplasmic matrices or arrayed in tubules as observed in RBSDV-infected rice tissues. Aggregates of virus particles often arrayed in crystal-like arrangement in the cytoplasm (Hoang et al 2011, Zhou et al 2008).

In RBSDV-infected SBPH, virus particles occur in the salivary glands, ovaries, fat bodies, and intestines (Kitagawa and Shikata 1974, Shanghai Institute of Biochemistry 1974, Shikata 1974, Shikata and Kitagawa 1977, Isogai et al 1999). In infected planthopper cells, virus particles are scattered or aggregated in the cytoplasm and in viroplasmic matrices, or arrayed in tubules in the cytoplasm as those observed in infected rice cells. Viroplasmic matrices are probably the site of virus maturation (Shikata and Kitagawa 1977). Virus-like particles of about 70 nm in diameter were observed in fractions “purified” from RBSDV-free SBPH, indicating SBPH has a native virus of about 70 nm in diameter (Kitagawa and Shikata 1974),

5.3. Virus

RBSDV (Kitagawa and Shikata 1969b, Shikata 1969) is a member of the genus *Fijivirus* of the family *Reoviridae* and contains 10 segments of double-stranded (ds) RNA as the genome. It is an icosahedral double shelled particle, 75-80 nm in diameter (**RSBD Figures 3a,d**) containing cores of about 50-55 nm in diameter (**RSBD Figures 3c,f**) (Isogai et al 1999; Kitagawa and Shikata 1969a, 1974; Shikata 1969; Shikata and Kitagawa 1977). It is likely that the RBSDV particle has 12 projections (A-spikes) on the surface of the outer capsid and 12 projections (B-spikes) on the surface of the inner cores as other viruses belonging to the genus *Fijivirus* (Milne and Lovisolo 1977, Isogai et al 1999). SRBSDV also has 10 dsRNA

segments that migrate similarly to those of RBSDV in gel electrophoresis (Zhou et al 2008).

RBSDV is not mechanically transmissible. Infectivity of virus particles can be assayed by injecting virus suspension into the abdomen of SBPH nymphs through a glass capillary and testing the injected planthoppers for RBSDV infectivity on rice seedlings (Kitagwa and Shikata 1969a). In the injection method, the infectivity was detected up to 10^{-4} dilution in RBSDV-infected rice leaf extracts, while up to 10^{-5} dilution in virus-infected SBPH extracts. RBSDV particles are unstable as other members of the genus *Fijivirus*, of which the outer shell is degraded after treatment with organic solvent or heating at 60°C (Milne and Lovisolo 1977, Hatta and Francki 1977). RBSDV lost its infectivity after incubating at 60°C for 10 minutes or at 4°C for 7 days or treatment with organic solvent including chloroform, n-butanol, ether or amylalcohol, but retained its infectivity after treatment with fluorocarbon (Difron S-3) or carbon tetrachloride (Kitagawa and Shikata 1969a, 1973). Virus particles that lost the outer shell are likely no longer infective (Milne and Lovisolo 1977).

Monolayer cell lines were established from the eggs of SBPH at 6-10 days after laying (Kimura et al 1996). The cell lines on cover slips incubated with RBSDV suspension were infected with RBSDV and stained with fluorescent isothiocyanate labeled with antibody to RBSDV.

RBSDV was easily degraded in phosphotungstic acid (PTA) or uranyl acetate stains, which are commonly used for the negative staining of virus particles for electron microscopy. It loses the outer shell when stained with PTA adjusted to pH 6.5-7.0, but retains the shell when stained with PTA at pH 4.5-5.5 (Isogai et al 1999; Mikoshiba et al 1988a,b). Treatment of RBSDV particles with organic solvents removes the A-spikes and the outer shells to give B-spiked core particles (**RBSD Figure 3b,e**). Treatment of B-spiked core particles with 1.9 M $MgCl_2$ removes the B-spikes from the core particles (**RBSD Figure 3c,f**). The A-spikes were retained when virus preparations were stained with uranyl phosphate, uranyl formate or PTA after fixing them on the grids using glutaraldehyde, similar to what has been observed for related viruses (Milne and Lovisolo 1977, Hatta and Francki 1977).

Purification of RBSDV is difficult, as virus content in infected tissues is low and virus is easily degraded during the purification processes. RBSDV was purified from infected rice leaves by macerating in phosphate buffer (pH 7.0), treating two times with carbon tetrachloride and one cycle of differential and 20-50% sucrose density gradient centrifugations (Kitagawa and Shikata 1969b). The centrifuged tubes had one or two bands. Purified virus fraction recovered from a band had infectivity when tested for virus infectivity by the injection method. RBSDV was also purified from infected rice leaves or SBPHs by macerating in phosphate buffer (pH

7.5) containing EDTA, one cycle of differential centrifugation, and stepwise sucrose density gradient centrifugation (Shikata and Kitagawa 1977). Single band formed after the last centrifugation showed infectivity. The preparations obtained in either purification procedures contained core particles but not intact virus particles, when stained in PTA for electron microscopy. Also, RBSDV was purified from infected maize roots by macerating in phosphate buffer containing ethylenediamine-tetraacetate and L-ascorbate (pH 6.0), treatment with Difron S-3, differential centrifugation, and 20-50% sucrose density gradient centrifugation (Mikoshiba et al 1988a,b). The centrifuged tubes had two bands, the top band with core particles and the bottom band with intact particles.

RBSDV particles contains 10 segments of double-stranded (ds) RNA as the genome, with a total molecular size of about 19 x 10^6 (Isogai et al 1995a, Reddy et al 1975) or 29,132 or 29,141 base pairs (Milne et al 2005). RBSDV has at least six proteins (Mikoshiba et al 1988b, Isogai et al 1999). Three proteins compose core particles and three other proteins compose the outer shell. RBSDV particles have RNA-dependent RNA polymerase activity (Uyeda et al 1987, Toriyama 1984).

Nucleotide sequences of RBSDV genome have been determined for isolates in Japan (Uyeda et al 1990, Azuhata et al 1993, Isogai et al 1999) and in China (Zhang et al 2001a,b; Bai et al 2002; Wang et al 2003). Each of eight dsRNA segments of RBSDV has coding regions for single protein, while segments 7 and 9 each have coding regions for two proteins. Of the 12 proteins coded in RBSDV-dsRNAs, six are structural proteins. In the immunogold labeling on RBSDV-infected rice or SBPH cells, the P7-1 protein coded in genome segment S7 constructs the tubules, while the P9-1 protein coded in S9 is localized in viroplasmic inclusions (Isogai et al 1998, Zhang et al 2008). Ten genome segments of RBSDV have common terminal sequences, AAGUUUUU--- for the 5’ terminal and ---AGGUNNCGUC or AGGUNNUGUC for the 3’ terminal (Azuhata et al 1993). The terminal sequences of maize rough dwarf virus (MRDV), which is closely related to RBSDV, are AAGUUUUUU--- and ---UGUC (Marazachi et al 1991).

SRBSDV has 10 strands of dsRNA with a gel electrophoretic profile similar to that of RBSDV. Nucleotide sequences of SRBSDV isolates in Guandong and Hainan have been determined (Zhang et al 2008a,b: Zhou et al 2008; Wang et al 2010). The size and organization of 10 genome segments of SRBSDV are similar to those of RBSDV and other viruses belonging to the genus *Fijivirus*. The nucleotide sequence homology between the Guandong and Hainan isolates ranged from 97.3 to 99.9%, while that to a RBSDV isolate in Zhejian was lower at from 70.6 to 79.0%. The terminal sequences of SRBSDV are AAGUUUUU--- for the 5’ end and CAGCUGAUGUC---- for the 3’ end.

RBSDV appears variable. Electrophoretic patterns of dsRNAs from individual plants infected with RBSDV differed from each other, even among those collected from one field (Isogai et al 1995a). The strains from field collections differed from a strain maintained in greenhouse. The nucleotide sequence was determined for S7 to S10 of RBSDV isolates on wheat in Hebei and on rice in Zhejian and for S9 and S10 of RBSDV isolate in Hubei (Zhang et al 2001b). The Hubei isolate was highly homologous at from 94 to 99% with other Chinese isolates at from 90 to 95% with RBSDV in Japan and from 85 to 88% with MRDV in Italy.

RBSDV and SRBSDV are closely related to MRDV in Europe and China, to pangola stunt virus (PSV) in the Mediterranean, and to Mal de Rio Cuart virus in South America and northern Australia in the genus *Fijivirus* (Marazachi et al 1995, Milne and Lovisolo 1977, Milne et al 2005). They are serologically related to each other, transmitted by the same vector species SBPH in persistent manners, and have common plant hosts in which they induce similar symptoms. On the other hand, unlike RBSDV, MRDV does not naturally infect rice and is reported to be transmitted via eggs in SBPH (Milne and Lovisolo 1977). MRDV in China and in Japan was not transmitted via eggs. Genomic dsRNAs of RBSDV and MRDV showed similar electrophoretic profiles (Reddy et al. 1975, Isogai et al 1995a). In studies using electrophoresis and nucleic acid hybridization, RBSDV S7 was found to correspond with MRDV S6, and RBSDV S8 to MRDV S7 (Azuhata et al 1993, Isogai et al 1995a). The sequence data on viruses in the genus *Fijivirus* indicated that the genetic distance between SRBSDV and MRDV is smaller than that between SRBSDV and RBSDV (Wang et al 2010). Indeed, RBSDV, SRBSDV, and MRDV are very close to each other. Presently, it is appropriate to leave them as species because of the economical importance of these diseases. RBSDV and MRDV have considerable variation and require further analysis to determine whether they are geographical races of the same species (Marzachi et al 1995, Zhang et al 2001b).

5.4. Host range

RBSDV has wide host range in gramineous plants. Artificial inoculation to cereals and gramineous weeds at the seedling stage, using viruliferous SBPH, showed that 25 RBSDV-infected gramineous species, including rice, wheat, oat, sorghum, maize, *Panicum milliaceu*, *Alopecurus aequalis*, *A. japonicous*, *Beckmania syzigachne*, *Cynosurus cristatus*, *Digitaria adscendens*, *D.violascens*, *Echinochloa crus-galli*, *Echinochloa* var. frumentacea, *E. crus-galli* var. orizicola, *Eragrostis milticaulis, Glyceria acutiflora, Lorium multiflorum, L. perenne, Phleum practense, Poa annua, Setaria viridis*, and *Trisetum birfidium* (Shinakai 1962).

Except in infected *P. annua*, infected plants showed stunting, fewer tillers, darker green leaves with occasional twisting at the leaf tips or splitting at the edges of leaf blades, and few panicles with no seeds. Infected *P. annua* showed milder symptoms and developed panicles with seeds. Galls or vein swelling were formed in the underside of leaves in infected maize, *S. italica, B. Syzigaachne, P. annua, A. aequalis,* and *A. japonicus,* but were not evident in infected plants of other species.

In Hainan, China, plants of maize, *E. crusgalli*, *Juncellus serotinus*, and *Pennisetum flaccidum* near SRBSD-affected fields showed plant stunting, leaf rolling, and dark-green leaves and gave positive reactions in reverse-transcriptase polymerase chain reaction (RT-PCR) targeting RBSDV (Zhou et al 2008). Inoculated plants of *Pennisetum flaccidum* gave positive RT-PCR but did not show symptoms.

5.5. Transmission by vectors

RBSDV is transmitted in a persistent manner by SBPH (**Intro Figure 7**) (Kuribayashi and Shinkai 1952) and two other planthopper species, *Unkanodes sapporona* Matsumura (Shinkai 1966) and *U. albifascia* (*Rihautodelphax albifascia)* Matsumura (Shinkai 1967, Hirao 1968). *U. sapporona* and *U. albifascia* are not important vectors of RBSDV in rice. SBPH acquired RBSDV within 15-30 minutes of access feeding. SBPHs that acquired RBSDV became infective after an incubation period of 7 to 35 days and retained the infectivity until they died (Shinkai 1962, Hirao 1968). SBPH transmits RBSDV to rice seedlings via access feeding within 5-10 minutes.

The proportions of infective SBPHs in populations that were exposed to RBSDV-infected rice plants varied depending on vector populations. The percentages were 20-88% for SBPH, 34-45% for *U. sapporona*, and 29-73% for *U. Albifascaia* (Shinkai 1962, Hirao 1968). RBSDV propagates in SBPH (Kitagawa and Shikata 1974) but it is not transmitted from female adults to their progeny via eggs (Shinkai 1962). SBPHs that fed on defrosted rice leaves infected with RBSDV acquired and transmitted the virus (Li et al 2011).

SRBSDV in Hainan, China was efficiently transmitted in a persistent manner by WBPH (**RBSD Figure 4**) and by SBPH although the efficiency was less than that by WBPH (Zhou et al 2008).

5.6. Diagnosis and serology

RBSD and SRBSD are diagnosed based on the characteristic symptoms on infected plants including galls, pronounced stunting, and darker green leaf color in the field. In mildly infected plants, diagnosis based on the symptoms is inconclusive as symptoms somewhat similar to these viruses are developed by infection with other viruses such as rice bunchy stunt virus (RBSV), rice dwarf virus (RDV), rice gall

dwarf virus (RGDV), and rice ragged stunt virus (RRSV). For conclusive diagnosis, serology or transmission tests using virus-free SBPH nymphs for RBSD and WBPH nymphs for SRBSD are adapted. RBSDV in infected rice extracts can be diagnosed by injecting extracts of diseased plants into the abdomen of SBPH nymphs and testing them later for their infectivity (Kitagawa and Shikata 1969a,b). Reverse transcription polymerase chain reaction (RT-PCR) detects RBSDV in rice or maize tissues or in a single SBPH (Li et al 2011). RBSDV and SRBSDV can be differentiated by RT-PCR using specific primers (Zhou et al 2008).

RBSDV is a good immunogen. Antisera to RBSDV have been prepared by injecting purified virus preparations into rabbits (Kitagawa and Shikata 1969a, Shikata and Kitagawa 1977, Woo and Lee 1987, Ishikawa et al 1987). Antisera reacted with RBSDV in infected plant or vector extracts, or in purified preparations in the ring interface precipitine test (Luisoni et al 1973), enzyme-linked immunosorbent assay (ELISA) (Woo and Lee 1987, Ishikawa et al 1987, Takahashi et al 1991), latex test (Omura et al 1984, Takahashi et al 1991), passive hemagglutination test (Takahashi et al 1991), and immunofilter paper assay (Cabauatan et al 1994). ELISA provides the most reliable detection for RBSDV. Diagnoses based on dot blot hybridization using cDNA clones of RBSDV-RNA as probes are sensitive and reliable (Isogai et al 2001). RBSDV and MRDV can be differentiated by dot blot hybridization with cDNA clones for S5 of a RBSDV strain H in Japan.

RBSDV is serologically related to MRDV, MRCV, PSV and *Cereal tillering disease virus* (Luiosoni et al 1973, Milne and Lovisolo 1977, Milne et al 2005). Most probably, RBSDV is also related to SRBSDV. Antibodies to double-stranded RNA were also produced in antisera when plant viruses of *Reoviridae* were used as immunogens (Ikegami and Francki 1973). Although there is no information so far, antisera to RBSDV likely also contain antibodies to dsRNA. The titer against dsDNA in antisera to fijiviruses is generally low and does not disturb ordinary serological diagnosis using the antisera to virus particles if they are diluted more than 10 times.

5.7. Disease cycle and epidemiology

In Japan, RBSDV naturally infects rice, maize, wheat, barley, oat, sorghum, and *Alopecurus aequalis* (Shinkai 1962). Infected cereals, except maize and weeds, serve as the reservoir for RBSDV and also SBPH during the winter season (Ishii and Yoshimura 1973). Infected maize plants do not serve as a virus source because SBPH does not prefer feeding on maize. After rice or maize is harvested in RBSDV epidemic areas, infective SBPHs move to grassy weeds in levees or nearby fields and then move on to newly planted wheat and barley, transmit RBSDV, and oviposit (Obi and Kosuge 1963, Ishii and Yoshimura 1973). Nymphs appear in September or

October and overwinter as diapausing nymphs at the fourth instar. During the winter season, nymphs stay at the basal part of the winter cereals or weeds or in soil cracks (Obi and Kosuge 1963). Infection of wheat, barley, and *A. sequalis* with RBSDV occurs generally at the seedling stage in October or November and symptoms in those infected plants may appear in November or December, but generally in March (Ishii and Yoshimura 1973). Infection of these plants may also occur in March if the growth of these plants is delayed because of an abnormal climate.

A. sequalis plants developed RBSDV symptoms at 0.5-2.0% in RBSD-affected rice fields with sparse vegetation with *A. sequalis* but zero in rice fields with dense vegetation (Shinkai 1962). Adults of the over-wintered generation appear in March to May. In central Japan, adults of the overwintered generation generally stay on weeds and winter cereals and oviposit in late April to early May. The overwintering generation survives and retains virus infectivity until late May or early June (Shinkai 1957c, 1962). The first generation stays on the winter cereals and acquires RBSDV on infected plants at the nymphal stage, and moves to newly planted rice or maize at the adult stage in late May to June (Shinkai 1957c, Obi and Kosuge 1863, Ishii and Yoshimura 1973). Rice planted in May to early June has higher infection but crops planted in mid- or late June have less infection. In maize, incidence is high in fields planted in late May to mid-June or in fields planted during late July to early August, indicating infection by second- and third-generation adults (Ishii and Yoshimura 1973). The RBSDV epidemics during 1961-65 in central Japan were attributed to the introduction of early rice planting (Kiritani 1983). Intermingled planting of early rice and middle or late rice in one area created favorable conditions for vector planthoppers, thus, for RBSD and RS. High RBSDV incidences in barley and wheat in 1961 were attributed to higher temperatures in November and December 1960 (Kosuge 1962).

In Zhejian, China, adults of the overwintering SBPH generation appeared in March and generally oviposited in barley or wheat (Li et al 1979, Ruan et al 1981, Wang et al 2009). Migration of the first-generation adults from the winter cereals to rice fields starts in April and increases after the harvest of the winter cereals in May to June. Parts of the second generation and large proportions of the third generation move from early rice to the second crop or late-planted rice (Ruan et al 1981). RBSDV incidence is generally correlated to the densities of SBPH first-generation adults (Bae et al 1992).

In maize, RBSDV incidence is generally low in fields planted before 15 May, high in fields planted during 15 May-15 July, while low in fields planted after 15 July (Li et al 1979). In the 1990s and 2000s in Zhejian, China, RBSD incidence was high in rice and winter cereals in indica-japonica rice double clopping areas where the

indica rice was transplanted in mid-March and harvested at the end of July and the japonica rice was transplanted in May and harvested by October (Wang et al 2009). The incidence is higher in the late japonica rice than in the early indica rice. RBSD incidence in rice is generally correlated to the incidence in winter wheat. The incidence in the early rice is correlated to the percentage of infective SBPH in overwintering populations.

At Yeongnum, Korea, in 1984, first-generation adults were first observed in rice fields on 29 May. In a 13 June counting, the maximum density of SBPH obtained was 4.9 to 19.6 hoppers per hill, depending on rice cultivars. The density was generally higher in fields transplanted earlier than in fields transplanted late. RBSDV infection in the nursery beds was not observed in fields transplanted in May but was high at 14.2% in fields transplanted on 20 June. RBSDV incidence was higher in fields transplanted in May than in fields transplanted in June. The incidence was even lower in fields transplanted in July. The development of RBSDV infection was faster in fields transplanted on 20 and 30 May than in fields transplanted on 10 May or in June. The proportion of SBPHs carrying RBSDV in populations was 13.6% for the second generation (Kim 1985).

Indeed, barley, wheat, and some weed hosts serve as reservoirs for RBSDV and its vector SBPH. Generally, the overwintering SBPH generation stays on the winter cereals. However, in areas where rice is planted early, the overwintering generation directly moves to rice fields and disperses RBSDV (Wang et al 2009). Generally, the first-generation SBPH acquires RBSDV from winter cereals and disperses it to newly planted rice or maize fields (Shinkai 1957, Obi and Kosuge 1963, Ishii and Yoshimura 1973, Li et al 1979, Ruan et al 1981). Many of the second-generation adults are winged and capable of flying long distances. The first generation is highly prolific. The SBPH population greatly increases in rice at the second generation but does not increase in maize, which is not a good host of SBPH. SBPH shows less preference and higher mortality in maize than in rice (Ishii and Yoshimura 1973, Li et al 1979). Fewer SBPHs acquire the virus on maize than on rice (Ishii and Yoshimura 1973). Proportions of RBSDV-infective SBPHs in overwintering populations were as high as 10-20%. It reached 30-40% in extreme cases (Shinkai 1962, 1971; Shinkai et al 1969).

RBSDV is generally endemic in many locations in China, Japan, and Korea, and often can reach epidemic levels locally. Generally, the epidemics occur suddenly either on rice, winter cereals, or maize or sometimes on both rice and maize. In each area, the epidemics generally lasted 1 or 2 years. The disease cycle for RBSDV might be hardly completed. Since 1980 in many locations in Japan and Korea, RBSDV occurred sporadically in localized areas and the incidence has been

generally low. In China, RBSD incidence was very low for almost 3 decades after the epidemic in the 1960s and before the second epidemic after 1996. It is known that SBPH has the ability to migrate long distances with the monsoon winds even across eastern China or the Huanhai seas (Kishimoto 1979). Some of planthopper immigrants most likely carry RBSDV, RSV, and SRBSDV. Long-distance migration of SBPH might have important roles in some epidemics of those diseases.

SRBSD, which reached epidemic proportions widely in southern China and Vietnam after 2005, was transmitted principally by WBPH (**RBSD Figure 4**) and also by SBPH (Zhou et al 2001). SRBSD emerged in Kyushu, Japan, in 2010. WBPH is an important rice pest in Asia, inhabits widely in the Asian tropics and subtropics, and does not overwinter in the temperate regions. It has a strong ability to migrate long distances from Vietnam through southern China to central, northern China, Japan, and Korea and migrates every year with the monsoon winds in the late spring to early summer from Vietnam through southern China to central and northeastern China, Japan, and Korea (Kishimoto 1976**;** Otuka et al 2008, 2009). On the other hand, SBPH inhabits and overwinters in central and northern China, Japan, Korea, and the Primorskiy area of Russia. SBPH density is generally low or zero and RBSD is not known in southern China except in Fujian (Xie et al 1994, Xie and Lin 2001). Apparently, WBPH has a principal roll in SRBSD epidemics whereas SBPH has a limited role (Zhou et al 2008). Indeed, SRBSD is a potential threat widely in the Asian subtropics and the temperate regions, as well as in the Asian tropics.

5.8. Loss

Rice, wheat, and barley plants infected with RBSDV at the seedling stage produce no grains. In severe cases, percentage infection in rice fields reaches 70-80% and total crop damage is also encountered (Ishii and Yoshimura 1973). When infected at later growth stages, plants show milder symptoms. Plants that showed 60% reduction in height had no yield while those with 16% reduction resulted in 35% reduction in yield (Ishii and Yoshimura 1973). Rice plants infected with RBSD before the 11-leaf stage produced no grains, but those infected at the 13-leaf stage did not show symptoms and no yield loss (Shinkai 1962). In rice, the damage caused by RBSDV was generally associated with damage caused by RSV, which also has SBPH as the principal vector. Plants infected with both RBSDV and RSV show severer symptoms (Obi and Kosuge 1958). In Zhejian, China, yield loss was correlated with RBSD incidence in infected rice fields as follows (Wang et al 2009):

y=0.80 (+-0.014)x – 0.03(+-0.147) in early indica rice
y=0.92(+ - 0.087)x – 0.65(+ - 0.269) in late japonica rice

Yield reduction amounts to 30-80% in barley and 20-80% in wheat (Obi and Kosuge 1963). Maize plants infected with RBSDV before the five-leaf stage produce no grains; those infected at the 6-leaf stage result in yield loss at 20%; and those infected at the 7-leaf stage have loss at 50% (Obi and Kosuge 1959). In maize fields where infection occurred at the later growth stages, plants showed leaf splitting and mild stunting and yield loss at 20-70 % (Kosuge and Obi 1957a).

5.9. Variety resistance

Rice varieties have been evaluated for resistance to RBSD in the field under natural infection (Yasu et al 1966, Ishii et al 1969, Ishii and Yoshimura 1973), in fields transplanted with seedlings that have been exposed in a cage to RBSDV-viruliferous SBPHs (Morinaka and Sakurai 1967), or in the greenhouse by artificial inoculation (Morinaka and Sakurai 1967, 1968; Choi et al 1983c; Xie and Lin 1982). Scoring is based on the percentage infection for the field tests and on a disease severity index for the greenhouse tests (Morinaka and Sakurai 1967).

No commercial varieties exhibit a high level of resistance to RBSD. No rice varieties so far tested is immune to RBSDV. All japonica varieties tested were susceptible (Morinaka and Sakurai 1967, 1968; Ishii and Yoshimura 1973), while a number of indica varieties and indica-japonica intermediate-type varieties showed strong resistances to RBSD in field and greenhouse tests. Many of those resistant varieties show low percentage infection, high percentage infection but tolerance to RBSD with milder symptoms, or both. The resistance in variety Te-tep is controlled by the major gene *Bs* (Morinaka et al 1969a,b).

In Fujian, China, 219 varieties were tested in the greenhouse for their resistance to RBSD infection (Xie and Lin 1982). Varieties Chikuaiai 3 and Baotaiai showed high levels of resistance. In Korea, indica-japonica crosses (Tongil varieties) showed higher resistance to RBSDV than japonica varieties (Choi et al 1983). In Zhejian, China, indica hybrid rice varieties showed resistance to SRBSD, milder symptoms, or both, while japonica varieties were more susceptible (Wang et al 2009). The susceptibility in japonica rice is not likely due to their susceptibility to SBPH. In 2010, when SRBSDV occurred in southern Japan, some of the high-yielding indica-japonica hybrids that showed especially high infection suppressed the WBPH population. It is known that plants of many japonica rice varieties show strong ovicidal activities against WBPH as a result of the plant's reaction to planthopper oviposition, while many of indica varieties show weaker reactions (Sogawa 1991; Suzuki et al 1993, 1996). These hybrids showed low ovicidal activities.

Rice lines with immunity to RBSD were obtained when transforming rice plants by introducing a fragment of a 500-base pair virus gene encoding viroplasm

component protein (Shimizu et al 2011). Some maize varieties showed resistance to RBSDV infield and greenhouse evaluations (Ishii and Yoshimura 1973). All the resistant cultivars had high infection rates but showed milder symptoms.

5.10. Control

Commercial varieties with high levels of resistance to RBSD are not available. RBSDV control is either by insecticide application to reduce the vector population or by cultivation practices. Since RBSDV shares SBPH as a common vector with RSV and the two virus diseases often occur together, insecticide application was often practiced for both diseases in combination.

In double-cropping rice areas in China, insecticide application in wheat and barley for the overwintering SBPH generation in March and in transplanted rice fields for the first generation in May effectively reduced the vector population and subsequently reduced RBSDV incidence in rice (Ruan et al 1981). In single rice cropping areas in Japan, broad application of insecticides using helicopters in wheat, barley, and rice fields in the late May to June reduced SBPH density in rice and maize fields and RBSD and RS incidences (Yanagi et al 1962, Matsumoto et al 1967).

Application of insecticides in nursery beds for late rice effectively reduced incidences of rice diseases including RBSD (Yanagi et al 1960). Application of insecticides in seedling boxes for machine transplanters was also effective in reducing seedling infection by RBSDV and other diseases after transplanting (Bae et al 1992). Insecticide application for third-generation nymphs and adults may also give effective control. In Japan, planting of rice early in May or late in July effectively reduced RBSD incidence. Winter plowing of rice fields reduced the vector population and RBSD incidence.

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