

Section 3. Virus and Phytoplasma Diseases

Chapter 3. Other insect-borne virus diseases

1. Giallume (RG), synonym: Enrochat; pathogen: RGV

1.1. History and distribution

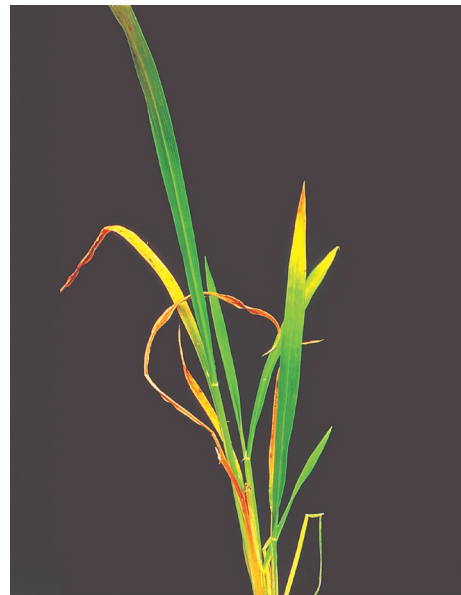
"Giallume" (yellowing in Italy) of rice (RG), which is characterized by leaf yellowing and plant stunting, occurs in Italy (**RG Figures 1 and 2**) (Belli et al 1974, Osler 1984). A similar disease called "enrochat" occurs in Spain (Betalla 1969, Jorda et al 1987). Similar disease also occurs in Hungary (Pocasal et al 1985).

RG was first observed in northern Italy in 1955 (Corbetta 1967). It occurred infrequently at low incidences for about a decade. Since 1966, RG has occurred more extensively and more frequently, especially in the major rice growing areas in northwestern Italy (Baldacci et al 1970, Belli et al 1974). RG is considered as one of the most serious diseases in rice in Italy. RG is transmitted in a persistent manner by *Rhopalosiphum padi* L. and some other aphid species (**RG Figure 3**) (Osler et al 1974). RG was postulated to be a virus disease and electron microscopy was used extensively in identifying the causal virus agent (Amici et al 1970, Amici and Favalli 1972, Bardacci et al 1970, Belli 1969, Pellegrini et al 1969). Once, RG was connected to mycoplasma-like organisms as membrane-bound bodies were observed in the phloem companion cells of RG-infected rice plants and in clarified rice extracts. Later, however, similar membrane structures were also found in rice plants free of RG. Besides the membrane bodies, isometric virus particles were found in the phloem cells of infected rice plants (Amici et al 1974). Similar particles were also found in weed plants naturally or artificially infected with RG (Amici et al 1975).

Based on the association of small isometric virus particles and relations of the particles to aphid vectors and rice cells, RG was identified as a disease caused by a virus related to barley yellow dwarf virus (BYDV) (Burnett 1990, Rochow and Duffs 1981). BYDV has a wide distribution in the world and infects many cereals and grasses. It is noteworthy that BYDV is not known in rice in many rice-growing countries where BYDV occurs in other cereals or grasses. BYDV naturally infects plants under relatively cool temperatures during the spring. Rice is grown under higher temperatures in the summer season in the temperate zone.

1.2. Symptoms

The symptoms in RG-infected rice plants are variable depending on cultivars, the age when plants are infected, and other environmental conditions (Baldacci et al 1970, Belli



RG Fig. 1. A rice plant infected with RGV (Courtesy R. Osler).



RG Fig. 2. A giallume-infested field showing RGV-infected plants in yellow patches (Courtesy R. Osler).



RG Fig. 3. Aphid *Rhopalosiphum padi*, a winged form adult (2.2-2.5 mm) and nymphs (Courtesy M. Miyazaki).

et al 1974, Osler 1984). The symptoms are generally clear under strong sunlight but their development requires lower temperatures than that required for rice growth. Infected rice seedlings develop symptoms 12-15 days after infection (Belli et al 1975). Plants infected with RG at the seedling stage show yellow to orange discoloration of leaves, starting at the tips or main veins, and serrated leaf edges (**RG Figure 1**). Leaves are small and erect and often show necrosis. Infected plants show stunting and reduced tillering, develop poor roots, and produce small panicles that are often sterile. In severe cases, infected plants show premature death. Rice plants infected with RG at younger growth stages show greater yield losses (Moletti et al 1990).

In rice fields, RG symptoms appear about 20 days after foliation of rice during the beginning of June (Belli et al 1974). Infected plants occur sporadically or in small patches, often along the banks (**RG Figure 2**). RG-infected grass, *Leersia oryzoides*, with mild or faint yellowing symptoms often occurs in the middle of such patches. The patches enlarge in size and fuse each other to cover large areas in the field. In July, the occurrence of RG in the field is easily identified from a distance based on the yellow-orange-colored patches. In August, many of the RG-infected plants are wilted or covered by tillers from adjacent healthy plants, and those patches become less conspicuous. Infected plants may develop normal leaves at the later stage of growth.

In the phloem tissues of RGV-infected rice or other gramineous plants, cells are often necrotic and contain virus particles of about 20 nm in diameter scattered or aggregated in the cytoplasm (Amici et al 1974, 1975, 1978). Infected cells have degenerated mitochondria and clusters of vesicles with fine filaments (Amici et al 1978, Faoro et al 1978). Infected cells occur infrequently in the phloem tissues.

1.3. Virus

RGV is included in the BYDV complex of the genus *Luteovirus* based on particle morphology, restricted occurrence in the phloem cells in small quantities (Amici et al 1974; Faoro et al 1978, Rochow and Duffus 1981) and its relations to the aphid vectors (Osler et al 1973, 1974). RGV was first grouped in the category of BYDV-PAV (transmitted by *M. avenae*, *R. padi*, and *S. graminum*) strain of BYDV (Osler 1984, Rochow 1969). In addition to the above traditional characteristics, members of the BYDV complex are icosahedral or spherical of about 25-28 nm diameter, composed of one major and one minor protein component and a single-stranded RNA genome (King et al 2012, Fattouh et al 1990). Virus particles stained with uranyl acetate appear 26-28 nm in diameter (Belli et al 1986) and about 20 nm in thin sections (Amici et al 1974, Osler et al 1974).

Virus concentration in infected plant tissues is very low and its purification is difficult. RGV was purified basically following a procedure reported for BYDV (Takanami and Kubo 1979). Frozen tissues of RG-infected oat plants were cut into small pieces, crushed in liquid nitrogen with mortar and pestle, and then homogenized in a blender with 900 ml of 0.1 M phosphate buffer, pH 6 containing 0.1 % thioglycolic acid and 0.5 % Driselase (Belli et al 1986). The extract was stirred for 60 min at 20-25°C and then overnight at 4°C. After filtration through a cheesecloth, the filtrate was emulsified with 50 ml of butanol and chloroform (1:1) mixture for 30 min and centrifuged at low speed. The aqueous layer collected was added with polyethylene glycol (PEG 6000) to 8% and NaCl to 0.2 M. The mixture was incubated at 4°C for 60 min and then centrifuged at low speed. The supernatant obtained is subjected to two cycles of differential and 10-40% sucrose density gradient centrifugations. A single peak obtained was diluted with the buffer and centrifuged at high speed. The final pellet containing virus particles was suspended in 0.01 M phosphate buffer, pH 7.2. The extinction coefficient at 260 nm of purified virus suspension is 6.0. Virus yield was 100 µg /100 g tissues.

Antiserum was obtained by immunizing rabbits with the purified virus had a titer of 1/1024 times in the agar gel diffusion test.

Purified RGV reacted with antisera to BYDV-RPV (*R. padi* transmitted) and BYDV-MAV (*Macrosiphum avenae* transmitted) (Francki et al 1985, Rochow and Duffs 1981). The reaction was much stronger with RPV than with MAV. Thus, RGV was grouped in the category of BYDV-PAV (transmitted by *M. avenae*, *R. padi* and *S. graminum*). However, based on analysis using enzyme-linked immunosorbent assay (ELISA), RGV was grouped in *Beet western yellows virus* (BWYV)-group (Casper 1988). Based on serological relations and cytopathological differences, the Luteoviruses were grouped into two: Group I includes

MAV, PAV, and SGV (*S. graminum* transmitted) while Group II includes RPV, RMV (*R. maidis* transmitted), and RGV (Francki et al 1985, Gill and Chong 1979). The classification of Luteo-virus is not well established.

1.4. Host range

RGV appeared to have a narrow host range in gramineous species. It naturally infects *Avena byzantina*, *A. sativa*, *Hordium vulgare*, *Leersia oryzoides*, *Panicum dichotomiflorum*, and *Triticum vulgare*. Seedlings of these species and *A. bizantina*, *Echinochloa crusgalli*, *Holcus lanatus*, and *Lorium perenne* were experimentally infected by using *R. padi* that fed on RG-infected rice plants (Amici et al 1975, 1978). Infection of those plants with RGV was confirmed based on the presence of 20 nm particles in infected cells or on the transmission test using *R. padi* to rice or *A. byzantina* seedlings.

The perennial weed *L. oryzoides* acts as a reservoir for RGV and *R. padi* during the winter season and serves as disease sources for newly planted rice seedlings (Osler et al 1980). In RG-endemic areas, RG-infected plants appear sporadically or in patches in newly planted fields. The patches often have a RG-infected *L. oryzoides* plant in the middle of the patches. *L. oryzoides* has become widespread in the 1970s in Northern Italy where rice is grown. Most likely, the increase in *L. oryzoides* densities in the 1970s was one reason for the epidemics of RG (Osler et al 1980a).

1.5. Transmission by vectors

In preliminary transmission tests using *R. padi* (RG Figure 3) and leafhopper and planthopper species collected from RG-affected fields, only seedlings confined with *R. padi* showed yellowing symptoms (Osler et al 1973, 1974). Seedlings that showed the symptoms were examined for the presence of virus particles by thin sectioning. All seedlings that showed the symptoms had BYDV-like virus particles. In 1981, the transmission of RG by *R. padi* was confirmed by using antisera to BYDV-MAV (Rochow and Duffus 1981). RGV is also transmissible by *Metopolophium dirhodum* and *Sitobion avenae* (Osler 1980b, 1984). *R. padi* is the most efficient vector, followed by *S. avenae* and then *M. dirhodum* (Osler 1984). The proportion of RG-infective *R. padi* in populations that was exposed to RG-infected plants was as high as 78% (Osler et al 1980). The transmission efficiency often reached higher than 70% in young *R. padi*.

RGV is transmitted by *R. padi* in a persistent manner (RV Figure 3; Osler 1984). Aphids feeding on a RGV source plant for longer than 1 or 2 hours became infective after a measurable incubation period and retained infectivity until they died. The incubation period in *R. padi* for RGV-infectivity is 2 days, but 9 to 23 days in *S. avenae* and *M. dirhodum* (Osler 1984). *R. padi* that become RG-infective after an access feeding to RG-infected plants retain 50% of the infectivity at 10 days after the access at 20°C. Apparently, RGV is not propagative but circulative in the vectors.

Virus-like particles were found in the gut of *R. padi* previously fed on RG-infected rice plants (Belli et al 1974, Faoro and Thornaghi 1983).

1.6. Diagnosis

Diagnosis is based primarily on the symptoms including yellow-orange discoloration and plant stunting. Diagnosis based on transmission tests using *R. padi* was also adapted on some occasions. After electron microscopy was introduced, diagnosis based on the presence of virus particles on thin sections of phloem tissues had been frequently used for conclusive diagnosis. In 1986, antiserum to RGV was produced making serological diagnosis available (Belli et al 1986).

RGV is a good immunogen. Antiserum to RGV was obtained by immunizing rabbits with purified virus fractions (Belli et al 1986). Antisera detected RGV in agar gel diffusion test (Belli et al 1986) and ELISA (Osler et al 1988, 1995). When serological tests gave negative results, extracts of diseased plants were partially purified before the test as virus concentration in infected plant tissues is limited (Casper 1988).

1.7. Disease cycle and epidemiology

An important disease source in newly planted rice fields is the perennial weed *L. oryzoides* and the vector *R. padi* (Osler 1980, 1984; Osler et al 1980a,b). *L. oryzoides* became more common in rice fields in northern Italy in the 1970s. Weed plants infected with RGV develop mild stunting and yellowing symptoms and overwinter in the field. *L. oryzoides* is also a good host of *R. padi*. After the rice harvest, *R. padi* colonize on *L. oryzoides* plants. In direct-seeded rice fields during spring time, RG-infected *L. oryzoides* plants develop new leaves ahead of rice. Such plants contain RGV particles in the leaf and root tissues and serve as fairly efficient RG sources. Wingless form of *R. padi* acquires RGV on infected *L. oryzoides* plants and disperses the virus to surrounding rice plants.

RG symptoms appear sporadically about 20 days after the emergence of rice seedlings (Belli et al 1974). RG is dispersed from the sources to surrounding rice seedlings to form patches (**RG Figure 2**). One *L. oryzoides* plant is frequently observed in the middle of each patch (Osler et al 1980). *R. padi* collected from rice fields at this stage are infective of RG when tested on rice seedlings. During the summer season, *R. padi* gradually move back from rice to *L. oryzoides*. Winged forms of *R. padi* appear to perform a limited role in intra-field dispersal of RG.

Avena byzantina also serves as a good host of RGV (Osler et al 1980a,b). The transmission efficiency of *R. padi* for RGV is highest from *A. byzantina* to *A. byzantina*, and decreases, in order, from infected rice to rice, from infected rice to *A. byzantina* or vice versa, from naturally infected rice to rice or *A. byzantina*, and then from *L. oryzoides* to rice or *A. byzantina*, or vice versa.

1.8. Losses

In Italy, yield losses due to RG were estimated to be from 5% to almost 100% (Osler 1984). In 1984 in Italy, areas planted to rice covered 184,000 hectares producing 648,000 tons of rice. Losses corresponding to 5% of rice produced were equivalent to 32,000 tons.

Remote sensing techniques were applied to estimate areas affected with RG in Italy (De Carolis and Leehi 1974).

1.9. Cultivar resistance

Screening for RG resistance started in 1974 at the University of Milan and Ente Nazionale Risi in Milan (Osler 1984). For screening, virus-free *R. padi* colonies were reared on rice seedlings of cultivar Balilla in cages (Osler and De Carolis 1976). Aphids at 20-30 per plant were allowed an acquisition feeding for 3 days on RG-infected plants at 19-21°C. Hundreds of seeds of each cultivar were raised in a container. At the 12- to 15-leaf stages, seedlings in containers were separately exposed for inoculation for 1 day to RG-viruliferous aphids. Inoculated seedlings were transplanted in tanks for the symptoms (Osler and De Carolis 1976).

Of 25 Italian rice cultivars tested, 6 cultivars including Roma, Originario, Raffaello, Baldo, Ribe, and Stirpe 136 showed resistance to infection. In other tests, seedlings of commercial cultivars were inoculated in greenhouse using RG-viruliferous *R. padi* (Moletti and

Osler 1978, Moletti et al 1979). Inoculated seedlings were transplanted in the fields and scored for percentage infection based on symptoms 2 months after inoculation. Cultivars Arborio, Navile, Nero, Veneria and Vialone nano showed resistance to RG infection, and also showed high performances in field tests.

When seedlings of five cultivars with different levels of resistances were inoculated with RGV-viruliferous *R. padi* at 1, 3, or 9 per seedling, resistant cultivar Arborio showed no infection. Moderately resistant cultivar G. Marchetti developed symptoms at 17 days after the inoculation while susceptible cultivars developed the symptoms at 12 days (Osler and Moletti 1982). Infection rate on cultivar G. Marchetti was 10% when inoculated at 1 per seedling, 31% at 3 per seedling and 50% at 9 per seedling. In field tests, infection rates obtained on cultivar G. Marchetti was less than 50%. No dosage effect was observed on symptom severity.

Seedlings of 11 Italian rice cultivars were inoculated at 10 days (first leaf stage) or 25 days (early tillering stage) after soaking by viruliferous *R. padi* at 3 per seedling (Moletti et al 1990). Grain yield reduction was 85-98% in plants inoculated at 10 days but 33-65% in plants inoculated at 25 days. Cultivars S. Andrea and Roma suffered the least damages.

Seedlings of 44 important world rice genotypes were inoculated with RG and scored based on symptoms and on ELISA using antiserum to BYDV-PAV (Osler et al 1995). Cultivar Arborio from Italy and MR 7, MR77 and MR103 from Malaysia had no infection. Seven cultivars did not develop symptoms but showed positive ELISA in few plants.

In artificial inoculation using *R. padi* in the greenhouse, cultivar Arborio appeared to carry one incompletely dominant resistance gene for RG (Baldi and Moletti, 1990). Seedlings of F_1 hybrids and 26 of the third backcross lines obtained from the crosses Cripto/Navile and Radon/Veneria were inoculated with RG and tested in ELISA for infection (Osler et al 1988). Of 66 lines tested, 18 did not show symptoms. Among the symptomless lines, 26 that were positive in ELISA were tolerant to RGV while 22 lines showed resistance to infection. Breeding lines have been selected in the field for RG resistance (Baldi et al 1988).

1.10. Control

Control of RG is based on application of insecticides to reduce vector population, use of herbicides to reduce perennial weed hosts in fallow rice fields, and use of resistant cultivars (Belli et al 1975).

Two applications of insecticides on 21 May and 30 May effectively reduced RG incidences and give significantly greater yield (Osler et al 1977).

RG incidence in Italy increased during the 1970s (Moletti et al 1990). Thereafter, the incidence in RG-epidemic areas declined to low levels because of planting less susceptible cultivars, eradication of the weed *L. oryzoides* in the rice fields, and relatively low population density of the main vector *R. padi*. In 1986, the density of *R. padi* and RG incidence were high.

References

- Amici, A., Ballesteros, R., Batalla, J. A., Belli, G. 1970. Corpi riferibili a microplasma in piante di riso spagnoles affetto da "enrochat" (in Italian). Riv. Pathol. Veget. Ser IV, 61:248-253.
- Amici, A., Faoro, F., Osler, R., Tornachi, R. 1978. The "giallume" disease of rice in Italy: new natural hosts of the viral agent, a strain of barley yellow dwarf virus. Riv. Pathol. Veget. Ser. IV, 14:127-135.
- Amici, A., Faoro, F., Tornaghi, R. 1975. Subcellular location in different hosts of the virus causing the "giallume" disease of *Oryza sativa*. Il Riso XXIV (4):353-358.
- Amici, A., Favalli, A. 1972. Thymidine-3H labeling of mycoplasma-like bodies in root of rice plants affected by a yellows-type diseases. Protoplasma 75:37-43.

- Amici, A., Osler, R., Belli, G. 1974. An isometric virus associated with "giallume" disease of *Oryza sativa*. *Phytopathol. Z.* 79:285-288.
- Baldacci, E., Amici, A., Belli, G., Corbetta, G. 1970. Il giallum del riso: osservazioni e ricerche su sintomatologia, epidemiologia ed eziologia. *Il Riso* 19(1). 9 p.
- Baldi, G., Moletti, M. 1990. Inheritance of resistance to giallume in rice. Pp. 429-433 in: P.A. Burnett, ed., *World Perspectives on Barley Yellow Dwarf*, CIMMYT, Mexico.
- Baldi, G., Moletti, M., Osler, R. 1988. Selezione per la resistenza al "giallume" nel riso. Pp. 796-803 in: *Atti Ufficiali, 10th Convegno Internazionale Sulla Riscoltura*, Vercelli.
- Belli, G. 1969. Mycoplasma-like particles in clarified extracts of diseased rice plants. *Riv. Pathol. Veg. Ser. IV.* 5:105-113.
- Belli, G., Amici, A., Corbetta, G., Osler, R. 1974. The "giallume" disease of rice (*Oryza sativa* L.). *Mikrobiologia* 11:101-107.
- Belli, G., Cinquanta, S., Prati, S., Soncini, C. 1986. Purification of an isolate of barley yellow dwarf virus causing rice giallume. *Rev. Patol. Veget. S. IV.* 22:81-86.
- Belli, G., Corbetta, G., Osler, R. 1975. Ricerche e osservazioni sull'epidemiologia e sulle possibilità di prevenzione del "giallume" del riso. *Riso* 24:359-363.
- Betalla, J. A. 1969. La enfermedad del arroz llamada "Roja" o "Rocha". *Arroz* 31:4.
- Burnett, P. A. 1990. **World Perspectives on Barley Yellow Dwarf**. P.A. Burnett, ed. CIMMYT, Mexico. 511 p.
- Casper, R. 1988. Luteoviruses. Pp. 235-258 in: *The Plant Viruses*, Vol. 3, R. Koenig, ed. Plenum Publishing Corporation.
- Corbetta, G. 1967. La nuova malattia che colpisce il riso. *Il Riscoltore* 11:3.
- De Carolis, C., Leehi, G. M. 1974. Rilievi da terra e dall'aereo nell'infrarosso fotografico e termico su campi di riso affetti da "giallume". 1. rilievi da terra. *Il Riso* 23:19-41.
- Faoro, F., Tornaghi, R. 1983. Subcellular location of a barley yellow dwarf virus strain in the intestinal epithelium of the aphid *Rhopalosiphum padi*. Pp. 155-156 in: XVIII Italian Congress on Electron Microscopy.
- Faoro, F., Amici, A., Tornaghi, R. 1978. Cellular ultrastructural alterations as a means of characterizing the virus causing the "giallume" disease of rice. XI Italian Congress on Electron Microscopy. *J. submicroscopic Cytol.* 10: 126.
- Fattouh, F. A., Ueng, P. P., Kawata, E. E., Barbara, D. J., Larkins, B. A., Lister, R. M. 1990. Luteovirus relationship assessed by cDNA clones from Barley Yellow Dwarf Virus. *Phytopathology* 80:913-920.
- Francki, R. I. B. Milne, R. G., Hatta, T. 1985. Ruteovirus Group. Pp. 137-152 in: *Atlas of Plant Viruses*, Volume 1, CRC Press, Boca Raton, Fla.
- Gill, C. C., Chong, J. 1979. Cytopathological evidence for the division of barley yellow dwarf virus isolates into two subgroups. *Virology* 95:59-69.
- Jorda, C., Medina, V., Garcia-Jimenez, J., Alfalo, A. 1987. Incidence of barley yellow dwarf virus on rice in Spain. *Phytopathol. Medit.* XXV:11-14.
- King, A. M. Q., Adams, M. J., Carstens, E. B., Elliot, J., Lefkowitz, E. J. 2012. *Virus Taxonomy*, 8th Eighth Report of the International Committee on Taxonomy of Viruses, International Union of Microbiological Sciences. Virology Division, Elsevier Academic Press, San Diego.
- Moletti, M., Osler, R. 1978. Determinazione della resistenza al "giallume" delle più importanti varietà di riso italiane mediante inoculazioni sperimentali con l'afide *Rhopalosiphum padi*. *Il Riso* XXVII:33-40.
- Moletti, M., Osler, R., Baldi, G. 1979. Valutazione della resistenza al giallume mediante inoculazioni artificiali con l'afide *Rhopalosiphum padi* in linee pure di riso migliorate. *Il Riso* XXVIII:53-61.

- Moletti, M., Risi, E. N., Osler, M. R., Villa, B. 1990. Some agronomic traits affected by rice giallume virus inoculated at two phonological stages in 11 italian rice cultivars. Pp. 464-467 in: P.A. Burnett, ed., World Perspective on Barley Yellow Dwarf, CIMMYT, Mexico.
- Osler, R. 1980a. Occurrence of *Sitobion avenae* and *Metopolophium dirhodum* in rice fields of northern Italy and transmission efficiency of the barley yellow dwarf virus strain causing the rice "giallume". Pp 113-118 in: Proceedings, Third Conference on Virus Diseases of Graminae in Europe, Harpenden, UK.
- Osler, R. 1980b. Il giallume del riso: attuali conoscenze sul ciclo di infezione del virus, con particolare attenzione alle piante *Oryza sativa* e *Leersia oryzoides* e all'afide *Phopalosiphum padi*. Il Riso 29:217-221.
- Osler, R. 1984. Rice giallume, a disease related to barley yellow dwarf in Italy. Pp.125-131 in: P.A. Burnett, ed., Perspectives on Barley Yellow Dwarf. CIMMYT, Mexico.
- Osler, R., Amici, A., Belli, G., Corbetta, G. 1973. Ricerche sull'epidemiologia del giallume del riso. I. Dimostrazione della presenza del vettori alati. Il Riso 22:105-110.
- Osler, R., Amici, A., Belli, G. 1974. Transmission of rice "giallume" by an aphid *Rhopalosiphum padi*. Riv. Pathol. Veg. Ser. IV. 10:3-15.
- Osler, R., Amici, A., Longoni, C. E. 1980 *Leersia oryzoides*, a natural host and winter reservoir of the rice "giallume" strain of barley yellow dwarf virus. Phytopathol. Z. 97:242-251.
- Osler, R., Carraro, L., Moletti, M., Baldi, G. Valdevit, G., Villa, B. 1988. Reazioni sintomatologiche e all'ELISA di linne di riso inoculate artificialmente con il virus del "giallume". Pages 232-239 in : 10th Atti Ufficiali, Convegno Internazionale Sulla Riscoltura, Vercelli.
- Osler, R., De Carolis, C. 1976. Prove preliminary di determinazione della resistenza al "giallume" di varietà italiane di riso mediante infezione artificiale. Riso 25:59-63.
- Osler, R., Faoro, F., Belli, G. 1977. Prove preliminary di lotta contro il vettore del "giallume" del riso. Riso 26:59-63.
- Osler, R., Moletti, M. 1982. Response of four cultivars of *Oryza sativa* inoculated with different numbers of the aphid *Rhopalosiphum padi* infected with the rice "giallume" strain of barley yellow dwarf virus. Phytopathol. Z. 105:51-60.
- Osler, R., Moletti, M., Lol, N., Petrot, I., Gludic, M. L. 1995. Evaluation of the resistance to BYDV of a group of important world rice genotypes. Pp. 69-73 in: J. Chataigner, ed., Rice Diseases in the Mediteranean Region and Breeding for Resistance: Proceedings of the Montpellier Seminar.
- Pellegrini, S., Belli, G., Gerola, F. M. 1969. Mycoplasma-like bodies in rice plants infected with a yellows-type disease. Giornale Bot. Ital. 103:395-399.
- Pocasal, E., Kiss, S. L., Basky, Z., Dezs'ery, M. 1985. Az a' rpa sa'rga torpese'g virus felle'pe'se rizsen. Nove'nyve'delem 21:308.
- Rochow, W.F. 1969. Biological properties of four isolates of barley yellow dwarf virus. Phytopathology 59: 1580-1589.
- Rochow, W. F., Duffs, J. E. 1981. Lueoviruses and yellows disease. Pp. 147-170 in: E. Kurstak, ed., Handbook of Plant Virus Infections and Comparative Diagnosis. Elsevier/North Holland, Amsterdam.
- Takanami, Y., Kubo, S. 1979. Enzyme-assisted purification of two phloem-limited plant viruses: tobacco necrotic dwarf and potato leafroll. J. Gen. Virol. 44:153-159.

2. Yellow mottle (RYM), pathogen: RYMV; synonym: rice pale yellow mottle virus

2.1. History and distribution

Yellow mottle of rice occurs in Africa. Rice plants characterized by yellow or orange discoloration, stunting, and panicle sterility (**RYM Figures 1 and 2**) were observed since 1966 near Kisumu along the shore of Lake Victoria in Kenya. In 1970, the disease was first described as a virus disease associated with isometric virus particles (Bakker 1970, 1974, 1975).

The causal virus, *Rice yellow mottle virus* (RYMV) (**RYM Figure 3**), is readily transmitted mechanically unlike other known rice viruses. Also, RYMV is transmitted in a semipersistent manner by many chrysomelid beetles belonging to the subfamily *Criocerinae*. RYM was once called “rice pale yellow mottle” in Nigeria (Raymundo and Buddenhagen 1976). During 1976-83, RYM was found in many rice-growing countries in Africa, including Burkina Faso, Ghana, Côte d’Ivoire, Kenya, Mali, Niger, Nigeria, Sierra Leone, and Tanzania (Awoderu 1991; Bakker 1974; Banwo et al 2001; Fauquet and Thouvenel 1977; Fomba 1988; John et al 1984; Raymundo and Konteh 1980; Rossel et al 1982a,b; Taylor et al 1990; Traore et al 2001). In 1989, it was found in Madagascar (Reckhaus and Randrianangaly 1990). RYMV might have long been present in these countries. RYM has not been found outside Africa.

RYM is endemic in irrigated areas and frequently reached epidemic proportions locally in such areas. RYM incidence is generally high in newly introduced rice cultivars as they are mostly susceptible (Rossel 1986). RYM is suspected to be a newly emerged disease that originated in gramineous weeds and spread rather recently in cultivated rice fields. The rapid and intense spread of the disease was associated with changes in cropping practices, including introduction of susceptible high-yielding cultivars and intensive cropping systems with transplanting (Calvert et al 2003, Rossel 1986).

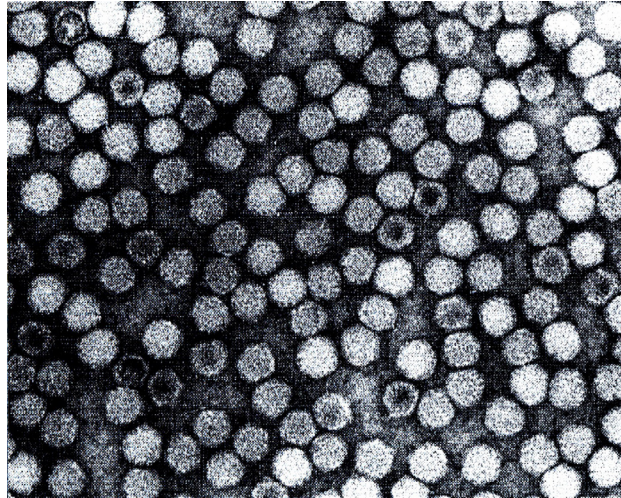


RYM Fig. 1. Rice plants infected with RYMV (Courtesy Hajime Kato).



RYM Fig. 2. A ratooned plant infected with RYMV (Courtesy Hajime Kato).

In Africa, rice cultivars (*Oryza sativa* L.) from rice-growing countries in Asia and other regions and lines of the native rice (*O. glaberrima* Steud) are being planted. Lines of *O. glaberrima* are grown in the flood plains of the Sahel and Sudan zones of West Africa where water is largely uncontrolled (Purseglove 1972). Revolutionary change in the preferences of West African consumers has created a wide and growing imbalance between regional rice supply and demand (GRiSP 2013). Although rice has been grown in many countries in East and Southern Africa for more than 500 years, it has only been in the last 2 decades that rice consumption has increased significantly (GRiSP 2013). Across Africa, the area planted to rice has increased substantially: 2.8 million hectares in 1960, 3.9 million in 1970, 4.7 million in 1980, 5.4 million in 1990, 7.5 in 2000, and 10.5 in 2010. In contrast, the average yield has been rather stagnant in the range of 1.12-1.16 t/ha (1960-90) and 2.31-2.46 t/ha (1990-2010) (World Rice Statics 1985, GRiSP 2013).



RYM Fig. 3. Purified RYMV. Source: Bakker (1974).



RYM Fig. 4. Ratooned rice plants infected with RYMV (Courtesy Hajime Kato).

2.2. Symptoms

Infected rice plants show yellow or orange discoloration of the leaves, plant stunting, and panicle sterility (Bakker 1970, 1974, 1975) (**RYM Figures 1 and 2**). Rice seedlings inoculated at the 3- to 4-leaf stage generally develop small yellow spots at the base of newly developed leaves 5-7 days after the inoculation. Yellow spots increase its number with the development of leaves, fuse each other with the leaf veins, develop mottling, streaking and crinkling, and eventually cover the whole leaf. New leaves delay in emergence and may show twisting and spiral appearance especially in the greenhouse. Leaves developed at later growth stages show yellow discoloration but no malformation. Leaf sheaths of infected plants often show mottling. Infected plants show stunting, form fewer tillers, and produce abnormal panicles with malformed small spikelets carrying unfilled grains. Infected plants may die prematurely. Infected seedlings of some cultivars show partial recovery after development of the typical symptoms. After the harvest of rice, infected rice stubble develop new leaves with symptoms (**RYM Figures 2 and 4**). Such ratoons survive certain periods and serve as disease sources.

When seedlings of the susceptible cultivar Sindano were inoculated at the 3- to 6-leaf stage, symptoms appeared on newly developed leaves 5 to 7 days after inoculation (Bakker 1974). Seedlings inoculated at 34 days developed the symptoms 7 days after

inoculation; those inoculated at 64 days developed symptoms 11 days after, while those inoculated at 93 days did 20 days after (Bakker 1974). The symptoms appeared at 4 or 5 days after inoculation at 30°C or higher, at 6 days at 25°C, at 7-8 days at 20°C, and at 10 to 12 days at lower than 20°C. In resistant cultivar Basmati 217, the symptoms were faint and difficult to discern under the sunshine. The faint symptoms appeared at 7 days on seedlings inoculated at 25 days, at 17 days on seedlings inoculated at 53 days, while no symptoms appeared in seedlings inoculated at 81 days or later.

Under the light microscope, infected leaf cells are yellowish-green and contain reduced number of chloroplasts (Bakker 1974). In infected cells, RYMV particles are aggregated or scattered in the cytoplasm of epidermal and mesophyll cells. Often, virus particles are arrayed in crystalline arrangements. Aggregates of fine fibrils occur in association with virus aggregates in the cytoplasm. Bundles of long flexuous tubules occur in the cytoplasm of infected cells. Microbody-like structures with electron dense centers have been observed in the cytoplasm of RYMV-infected plants. RYMV particles have also been observed in the epidermal cells, mesophyll cells, and in bundle sheath and vascular parenchyma cells (Opalka et al 1998). A large amount of RYMV has been reported in the xylem parenchyma cells and vessels.

2.3. Virus

RYMV is a member of the genus *Sobemovirus* and contains single-stranded (ss) - RNA. It is isometric, about 28 nm in diameter (**RYM Figure 3**) and constructed with 180 capsid proteins arranged in T=3 icosahedral lattice (Qu et al 2000). RYMV is easily transmitted by mechanical means by rubbing the surface of rice leaves with infected rice extracts added with carborundum or celite as an abrasive (Bakker 1974). It is similar in morphology, physical properties, and vector relations with Cocksfoot mottle and Phleum mottle viruses of the genus *Sobemovirus* (Catherall 1970, Serjeant 1967).

RYMV is physically stable (Bakker 1975). In infected leaf extracts, RYMV infectivity was inactivated at 65°C (55-70°C) in 10 minutes. Extracts diluted in phosphate buffer retained the infectivity for 99 days at 20°C or for 260 days at 4°C. Infected rice extracts retained the infectivity for at least 1 year at 4°C in dried conditions with CaCl₂, and several months in freezer. Dilution end point for infectivity of infected rice extract was 10⁶ to 10⁹ (Fauquet and Thouvenel 1977). RYMV is readily transmitted by mechanical means by rubbing with abrasives. It is transmitted from rice to rice with the aid of harvesting stickers or other tools through contacts via injury. RYMV is released from infected rice plants into the flooding water during puddling and leveling, and infects transplanted rice seedlings through injuries.

RYMV concentration in infected rice tissues is high, and purification of the virus from infected rice plants is rather easy (Bakker 1975). For purification of RYMV, seedlings of cultivar Sindano at the 5- to 6-leaf stage were inoculated with RYMV and harvested 10-12 days after the inoculation. Two to 3 weeks after the harvest, newly developed leaves were harvested again. The leaves were used soon after the harvests or kept at -25°C. Leaves were homogenized in 0.1 M phosphate buffer, pH 5.0 containing 0.2% 2-mercaptoethanol. The homogenate was filtered through a cloth and centrifuged at a low speed. The supernatant was emulsified with 0.5% (in volume) chloroform for 5 minutes and centrifuged at a low speed. The aqueous layer was collected, mixed with 20% (NH₄)₂ SO₄ and centrifuged at a low speed. The pellet was suspended in the phosphate buffer and dialyzed against the same buffer. The dialyzed fraction was centrifuged at a high speed. The pellet was suspended in 0.01 M phosphate buffer (pH 7.0) and dialyzed again with the phosphate buffer. The yield obtained was about 1 mg/g leaves (Bakker 1974) or 400 to 600 mg/g leaves (Fauquet and Thouvenel 1977).

The specific extinction coefficient of purified fraction at 260 nm was 6.5 (1 mg/ml, 1 cm light path) (Bakker 1974, 1975). UV light adsorption spectrum showed the maximum peak at 260 nm while the minimum peak was at 243 nm, resulting in 1.29 ratio, with A_{260nm} / A_{243nm} of 1.46 (Fauquet and Thouvenel 1977). The isoelectric point was pH 6.0. The sedimentation coefficient at 20°C was 109 S (Bakker 1974) or 116.5 (Fauquet and Thouvenel 1977). The density of RYMV was 1.359. Purified virus particles does not withstand freezing.

RYMV contains a single-stranded RNA of molecular mass of about 1.4×10^6 , corresponding to about 23% of particle weight. The molar ratio of nucleotides is as follows: G, 29 %; A, 21 %; C, 25 %; and U, 25 %. The nucleotide sequences of RYMV isolates from Cote d'Ivoire and Nigeria were determined. The genome contains four open reading frames (ORF). ORF1 encodes P1 protein of 17.8 kDa, ORF2a, which is a large polypeptide containing the domains of VPg, protease, helicase, and RNA polymerase. ORF2b overlaps with ORF2a and encodes a single protein. ORF4 encodes a capsid protein of 26 kDa (Yassi et al 1994). The genome organization of RYMV is similar to that of *Cocksfoot mottle virus*. P1 protein is dispensable for virus replication and important to virus spread in infected plant tissues (Bonneau et al 1998). A small circular RNA (sc-RNA) of about 210 or 220 nucleotides is associated with some RYMV isolates (Collins et al 1998, Sehgal et al 1993, Pinel et al 2003). RYMV sc-RNA is similar to viroid-like satellite RNAs found in association with other plant viruses (Bussiere et al 1996). Purified sc-RNA is infectious when inoculated with RYMV genomic RNA, but is not infectious when alone. Plants inoculated with both RYMV genomic RNA and the sc-RNA showed similar but somewhat more severe stunting than those inoculated with genomic RNA of RYMV alone.

RYMV appears variable. Several strains have been identified based on their serological properties and sequence similarities (Fauquet and Thouvenel 1977; Konate et al 1997; Mansour and Baillis 1994; N'Guessan et al 2000, 2001; Pinel et al 2000; Traore et al 2001). Strains S1, S2, and S3 were identified in West Africa and strain 4 in East Africa (N'Guessan et al 2000, 2001; Pinel et al 2000). Those strains are serologically differentiable and can also be determined based on sequence similarities of the coat protein gene or on discriminating monoclonal antibodies (N'Guessan et al 2000, 2001). In West Africa, S1 is predominant in the Savanna regions while S2 is predominant in forested zones where rainfed rice is planted (the rice belt) and in the central region where lowland irrigated rice and yam are planted. Using eight monoclonal antibodies against a RYMV isolate, S3, S4, and S5 were differentiated in triple antibody sandwich enzyme-linked immunosorbent assay (TAS-ELISA) (N'Guessan et al 2000). Two isolates from Nigeria and one from Sierra Leone were assigned to S3. Meanwhile, five from Madagascar, two from Tanzania, and one from Kenya were assigned to S4 and two from Tanzania were assigned to S5. Phylogenetic studies on the coat protein gene of 40 isolates indicated diversity of RYMV in East Africa than in West and Central Africa, suggesting earlier diversification of RYMV in East Africa (Abubakar et al 2003). In Central Africa, the collected RYMV isolates were assigned to S1 serotype (Traore et al 2001). The nucleotide sequences of the coat protein gene of seven isolates from West Africa indicated that they belong to a sister group of S1 (Abubakar et al 2003). Phylogenetic analysis on nucleotide sequences of 14 isolates, 2 of which sequences have been reported, indicated marked decrease in nucleotide diversity from east to west across the continent (Fargette et al 2004).

Isolates of S1 and S2 interacted in rice tissues of doubly infected plants (N'Guessan et al 2001). The S1 content in doubly infected plants was about 1/10 of that in singly infected plants, while S2 contents in doubly and singly infected plants were comparable. When 15 RYMV isolates belonging to S1 or S2 were planted, yield losses were in the range of 44-49% for S1 isolates but 31-35% for S2 isolates (N'Guessan et al 2001).

2.4. Host range

RYMV infects *Oryza sativa* and *O. glaberrima* and has a wide host range in grass weeds. Many grasses have been tested for host range by mechanical inoculation (Bakker 1970, 1974; Fauquet and Thouvenel 1977). Plants that showed systemic infection and developed symptoms included *Dinebra retroflexa*, *Diplachne caudate*, *Eragrostis aethiopica*, *E. ciliaris*, *E. namaquensis*, *O. alta*, *O. australiensis*, *O. barthii*, *O. brachyantha*, *O. glaberrima*, *O. latifolia*, *O. nivarra*, *O. punctata*, *O. ridleyi*, *O. rufipogon*, *O. spontanea*, and *Phleum arenarum*. Plants that showed systemic but symptomless infection included *Eleusine coracana* and *Eragrostis tenella*. Plants that showed localized infection and gave positive recovery from the inoculated leaves only included *Bromus hordeaceus*, *Dactyloctenium aegyptium*, *Eleusine coracana*, *Eragrostis aspera*, *Eragrostis chapelieri*, *E. cillanensis*, *E. macilentia*, *E. pilosa*, *E. tef*, *O. alta*, *O. eichingeri*, *O. glandiglumis*, *O. latifolia*, *O. minuta*, *O. officinalis*, and *Setaria viridis*.

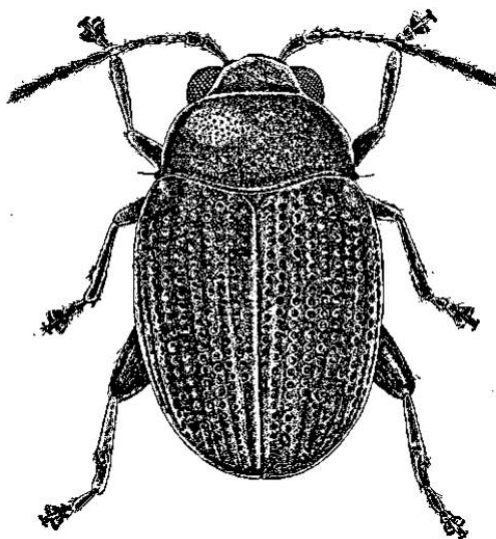
Natural infection of weeds was not found in Kenya and Nigeria (Bakker 1974, Rossel, 1986). Grasses common in the field in Kenya were collected and transplanted in the field with rice seedlings inoculated at 30 days after transplanting as a spreader row (Okioma et al 1983). Plants of *Dinebra retroflexa* and *Oryza* sp. showed symptoms and gave positive reaction in the tube precipitine test using antiserum to RYMV. *Eleusine indica* and *Eragrostis tenuifolia* showed no symptoms but gave positive serology.

2.5. Transmission by vectors

RYMV is mechanically transmissible and transmitted in a semipersistent manner by chrysomelid beetles belonging to the subfamilies *Criocerinae*, including *Chaetocnema abyssinica*, *C. kenyensis*, *C. pallidipes*, *C. pulla* (RYM Figure 5), *Cryptocephalinae* sp., *Dactylispa bayoni*, *Dactylispa* sp, *Di cladispa paucispina*, *D. viridicyanea*, *Monolepta flaveola*, *Oulema dunbrodiensis*, *Sesselia pusilla*, and *Trichispa sericea* (Bakker 1970, 1971, 1974; Banwo et al 2001; Reckhaus and Andriamasinthseho 1997). All the beetles are common leaf feeders on rice and grasses in Africa. They eat the edge or surface of leaves along with the veins or gnaw at the stem. Of these vector species, *Chaetocnema* spp., *D. viridicyanea*, and *T. sericea* cause feeding damage on rice. A long-horned grasshopper *Conocephalus merumontanus* also transmitted RYMV (Bakker 1974).

Chaetocnema spp. and *S. pusilla* occurred abundantly in RYMV affected areas in Kenya (Bakker 1974). *C. pulla* collected from rice fields were infective of RYMV. *C. pulla* and other *Chaetocnema* spp. are considered important vectors of RYMV in Kenya. *S. pusilla* feeds on blooming flowers of *Cyperus* spp. and other grasses and considered less important. In Tanzania, *Chaetocnema* sp., *C. pulla*, *Dactylispa* sp. are important vectors, while in Madagascar, *Hispa gestroi* (*Di cladispa gestroi*) and *T. sericea* are important.

Minimum acquisition access feeding time for RYMV is 15 min. for *C. pulla* and *T. sericea* (Bakker 1974). Minimum inoculation access feeding time is 15 min. in *C. pulla* and *T.*



RYM Fig. 5. Beetle *Chaetocnema pulla* (2 mm).
Source: Bakker (1974).

sericea and 30 min for *S. pusilla*. Beetles that acquired RYMV retain virus infectivity for from 5 to 8 days in *S. pusilla*, 5 days in *C. pulla*, and only 1 day in *T. sericea*. Transmission efficiency of RYMV for *C. pulla* was 45% on seedlings of cultivar Sidano, 19% on Basmati 217, and 39% on IR22. For *S. pusilla*, transmission efficiency was 28% on Sidano, 15% on Basmati 217 and 21% on IR22, while that for *T. sericea* was 26% on Sidano, 8% on Basmati 217 and 18% on IR22 (Bakker 1974).

Transmission through seeds or soil was not observed (Bakker 1970, 1974; Fauquet and Thouvenel 1977; Konate et al 2001). Rice plants infected with RYMV generally produce no grains, thus, it is difficult to test possible seed transmission. When seeds of RYMV-infected BG90-2 plants were tested for the presence of virus in ELISA and by infectivity assay, RYMV antigen was present in the glumella, endosperm and embryo in 65-100% of tested seeds (Konate et al 2001). No seedborne infection was observed in seedlings that germinated from infested seeds. Infectivity in immature grains was reduced to nearly zero with the advent of maturation. Virus infectivity in seeds is likely inactivated in matured seeds after desiccation. In 1991, when experimental fields were newly developed in an isolated place 50 km far from the nearest rice fields in Côte d'Ivoire, two plants were found infected with RYMV (Tsuboi 1999). In considering all potential sources for infection in the fields, the possible source was narrowed down to seeds used in the fields. The circumstances indicated possible infection of germinated seeds with RYMV released from contaminated unfilled grains from RYMV-infected plants during the process of rinsing and seeding rice seeds (Tsuboi 1999).

2.6. Diagnosis and serology

Diagnosis based on the symptoms is widely practiced in the field although it is not always conclusive. The symptoms on mildly infected plants can be confused with mineral deficiencies or other physiological disorders. As RYMV is transmissible by mechanical means, diagnosis can be easily done by inoculating rice seedlings by rubbing with extracts of diseased plants at about 100 times dilution added with carborundum or celite as an abrasive (Bakker 1974, Tsuboi 1999). Serological diagnosis is reliable as virus content in infected plants is generally high and specific antisera do not cross-react with any other known virus that infects rice (Mansour and Bailis 1994, Konate et al 1997).

RYMV is a good immunogen and antisera with titers of 1/1024 or 1/512 were obtained by immunizing rabbits with purified virus fractions (Bakker 1974, Fauquet and Thouvenel 1977, IITA 1980). In the agar gel diffusion test, spurs were formed between antisera to an isolate in Kenya (Bakker 1974) and to an isolate in Côte d'Ivoire (Fauquet and Thouvenel 1977), indicating presence of serotypes. Monoclonal antibodies to RYMV isolates were obtained (Konate et al 1997, N'Guessan et al 2000). Using a set of monoclonal antibodies in TAS-ELISA, serogroups S1, S2, S3, S4, and S5 were differentiated (N'Guessan et al 2000, 2001).

RYMV was detected in infected rice extracts using antisera in the agar gel diffusion test (Bakker 1970, 1974; Fauquet and Thouvenel 1977; John et al 1984; Mansour and Baillis 1994; Reckhaus and Randrianangaly 1990), tube precipitine test (Okioma et al 1983), immunosorbent electron microscopy (Mansour and Baillis 1994), and ELISA (Ghesquière et al 1997, John and Thottapilly 1987, Mansour and Baillis 1994, Ndjioudjop et al 1999).

Using reverse transcriptase-polymerase chain reaction (RT-PCR), S1 and S2 isolates were differentiated (N'Guessan et al 2000).

2.7. Disease cycle and epidemiology

Infection of seedlings in the nursery beds appears to be limited (Bakker 1974, John et al 1988). Major infection likely occurs after transplanting, as fields transplanted with seedlings from the same nurseries often show severe infection in one field but not in some other fields. In RYM endemic areas, RYM symptoms appear from 2 to 3 weeks after transplanting. Infected plants occur randomly over whole fields, along the levees, or near water inlets or outlets (Bakker 1974, Jones et al 1988, Tsuboi 1999, Tsuboi et al 1996, WARDA 2000). Infected plants may form patches. Dispersal of RYM in the field is generally slow, indicative of the limited movement of its beetle vectors. The dispersal likely occurs largely by mechanical means and rather infrequently by vector beetles. Infected plants increase the number and may spread eventually to the entire fields.

Infected rice plants including volunteer plants and rice stubble are the primary source of RYM. RYM is dispersed from the source plants to newly planted fields by beetle vectors or by mechanical means through contact with the winds, flooding water, agricultural tools, human, or animals (Sarraf and Peters 2003). During the rice harvest, RYM is easily dispersed by sickles contaminated with the virus. After the rice harvest, infected ratoons develop new leaves with the symptoms. Infection of seedlings with RYM also occurs during transplanting. During land preparation and paddling, RYMV is released into flooding water from infected rice stubble and volunteer rice plants, which are damaged and buried into wet soil. Infection of uprooted seedlings with RYMV in flooding water occurs through injuries on roots and also on leaves, if seedlings are transplanted soon after land preparations (Calvert et al 2003, Tsuboi 1999, Tsuboi et al 1996, WARDA 2000). When uprooted seedlings or their leaves were soaked in water containing RYMV-infected rice extract, they had infection (Abo et al 2000, Bakker 1974, Calvert et al 2003, Tsuboi 1999, Tsuboi et al 1996, WARDA 2000). The infectivity of flooding water was reduced day by day to a very low level in two weeks after land preparations (Tsuboi 1999). Guttation fluid from infected rice plants is also infective (Bakker 1974).

RYMV incidence is generally higher in irrigated areas where double cropping rice is practiced. The incidence is higher during the dry season than during the rainy season in Côte d'Ivoire (Tsuboi 1999) but higher during the rainy season in Nigeria (John et al 1988). Generally, the incidence is higher in flooded fields than in upland fields, in transplanted fields than in direct seeded fields, and in fields plowed using a hoe than in fields plowed using a cultivator (Abo et al 2000, Tsuboi 1998, Tsuboi et al 1996).

Africa covers diverse environments (GRiSP 2013). The major RYM sources in newly planted fields appear different among areas or countries. In Kenya and Nigeria, natural infection by RYMV of indigenous wild rice and weed species has not been found (Bakker 1974, Rossel 1986). However in Kenya, *Oryza* sp. and three weeds had infection with RYM when planted in rows with RYM-infected rice plants as a spreader row (Okion et al 1983). Natural infection of *O. longistaminata*, which is common in small ponds and swamps, was found in Niger and Mali (John et al 1984). The role of chrysomelid vectors in RYM epidemics is not certain. In Tanzania, the population of major vectors *Chaetocnema* sp. and *C. pulla* were monitored in broadcasted rainfed rice fields (Banwo and Makundi 2001). The population of *Chaetocnema* sp. was significantly higher in fields in RYM-prone areas than in RYM-free areas located within a 1 km radius. The population of *C. pulla* was significantly higher at 63 days after planting in RYM-prone fields than in RYM-free fields, indicating the importance of some beetles in RYM epidemics. The role of vectors was apparently limited in some countries.

In Cote d'Ivoire, the population of vector beetles is generally low and dispersal of RYM from initially infected plants to surrounding plant is limited (Tsuboi 1999). In Niger, RYM is generally sporadic or none, and occurs severely in some fields where infected

plants occur in patches, along with levees, roadsides, or at the corner of a road, indicating dispersal by factors other than beetles (Sarra 2003). *C. pulla* and other *Chaetocnema* spp. are considered important as vectors of RYM in Kenya (Bakker 1974). *Chaetocnema* sp., *C. pulla*, and *Dactylispa* sp. are important in Tanzania while *Hispa gestroi* (*Dicladispa gestroi*) and *T. sericea* are important in Madagascar.

2.8. Losses

RYM is one of the most important diseases in rice in Africa. Yield losses due to RYM range from 25 to 100% in affected fields (Abo et al 1998). Yield reduction is more severe in plants infected at earlier growth stages (Bakker 1974). In cultivar Sindano planted separately in pots, grain yield reduction was 58% in plants inoculated at 34 days after seeding but about 100% in plants inoculated at from 49 to 110 days. In resistant cultivar Basmati 217, the reduction was 100% in plants inoculated at 25 days, 94% in plants inoculated at 39 days, 52.5% at 53 days, 30% at 67 days, 5.2% at 81 days, but none in plants inoculated at 96 days.

In a field study, yield reduction in susceptible cultivar Bouake' was nearly 100% in plants inoculated at from 7 to 35 days after transplanting, 51% in plants inoculated at 42 days, 39% in plants inoculated at 49 days, 32% in plants inoculated at 56 days, and 11% in plants inoculated at 63 days (Tsuboi 1999). Yield reduction varied depending on RNMV isolates. Variability was less in resistant cultivars Tog572 and Gigante and in moderately resistant cultivars Fkr27 and Tox3211 but greater in susceptible cultivar Wita78 (N'Guessan et al 2001). Yield loss in plants infected with 15 isolates varied from 1 to 49% in cultivar Ita312 and 10 to 78% in cultivar Ngoyumaboi. Two of 15 isolates caused especially severe yield losses at 70 and 65% on average.

2.9. Cultivar resistance

Rice cultivars have been tested for their resistance to RYMV largely by mechanical inoculation after transplanting (Attere and Fatokun 1983, Bakker 1974, Fomba 1988, John and Thottapilly 1987, John et al 1984, Okyoma and Sarkarung 1983, Raymundo and Buddenhagen 1976, Raymundo and Konteh 1980, Taylor 1989). At the International Institute of Tropical Agriculture (IITA), Nigeria, screening of rice cultivars and lines for RYM resistance started soon after the disease occurred in the area by mechanical inoculation (John et al 1985, 1988).

Cultivar Moroberekan, LAC 23, OS 6, CT 19, and several lines showed resistance to RYM. Many *O. glaberrima* lines did not show clear symptoms but gave positive ELISA. Among the lines, TOG 5674 and TOG 5681 had especially low amount of RYMV. LAC 23 had least amount of RYMV. In 1976, 16 of 163 rice lines tested showed resistance, including TOS 2583, TOS 4053, TOS 4092, IRAT 13, Iguape Casteto, Moroberekan, Fossagbe, Juma 1, Gbengben, CI 9680, Merikan Largu and several lines (Raymundo and Buddenhagen, 1976). In 1980, 25 of several hundreds rice cultivars were tested were resistant (Raymundo and Konteh 1980). In 1980, 455 lines of *Oryza glaberrima* were tested for resistance to RYMV in the field (Attere and Fatokun 1983). Thirty-five lines showed no symptoms while a few lines showed initial symptoms that subsequently developed green leaves and showed recovery from infection. In greenhouse tests on 35 selected lines, 13 lines that included 3 from Mali, 8 from Nigeria and 2 from Upper Volta, did not show symptoms. In 1987, a scoring system based on symptom severity and serological indexing was developed for RYMV (John and Thottapilly 1987). Upland Japonica rice cultivars generally had moderate resistance to RYMV (Thottapilly and Rossel 1993).

In Côte d'Ivoire, of 15 IRAT cultivars tested, all were susceptible (Fauquet and Thouvenel 1977). In another test, all indica cultivars tested, except WITA 9, were susceptible (Tsuboi 1999). Cultivars that showed resistance included tropical japonica cultivars Moro-

berekan, Lac 23, and OS 6; japonica cultivars Sasanishiki, Koshihikari, and Hitomebore; and four lines from crosses between *O. sativa* and *O. glaberrima*. The tropical japonica cultivars showed symptom-less infection.

In Kenya, 11 of 30 cultivars tested developed only minute streaks on leaves (Okio-ma and Sarkarung 1983). All upland rice cultivars tested, including three upland cultivars, Ngovie, Moroberekan and 056, showed resistance to RYMV while all lowland cultivars from outside Africa were susceptible. In Sierra Leone, 20 of 600 rice cultivars and lines tested for resistance to RYMV showed resistance (Fomba 1988). Most cultivars and lines from outside Africa were susceptible. In another test, ROK16 was found resistant among eight upland cultivars evaluated in the field (Taylor 1989). In Madagascar, among 503 cultivars and lines tested in the fields and by mechanical inoculation in the greenhouse only Indica cultivar Bekarosaka showed no symptoms and gave negative reactions in ELISA and RT-PCR, indicating high level of resistance comparable to that in the cultivar Gigante (Rakotomalala et al 2008).

Upland japonica cultivar Azucena and a few lines of *O. glaberrima* showed moderate resistance to RYMV and gave a low virus titer in infected seedlings at the early stage of infection indicating a delay in virus infection in newly developing leaves (Ioannidou et al 2000). *O. glaberrima* lines Tog5672 and Tog568 and Gigante were resistant to RYM (Ndjiondjop et al 1999, Thottappilly and Rossel 1993). The resistance in both Tog5681 and Gigante was controlled by a single recessive gene *rymv1* located on chromosome 4 (Albar et al 2003; Ndjiondjop et al 1999, 2001). The propagation of RYMV in seedlings of resistant cultivars Tog5681 and Gigante, moderately resistant cultivars Azucena and Ac. 2428, and susceptible cultivars Tog5673 and IR64 was compared using protoplasts, and in inoculated or systemically infected leaves in ELISA and Northern blot analysis (Ndjiondjop et al 2001). Results indicated that cell- to-cell movement of the virus in Tog5681 and Gigante did not occur, and the movement was delayed in Azucena and Ac. 2428. The resistance in Bekarosaka from northwest Madagascar was evaluated using 4 RYMV pathotypes to compare with resistant cultivar Gigante. Genetic analysis revealed that the resistance was allelic to the resistance gene in Gigante (Rakotomalala et al 2008). Bekarosaka and Gigante likely share the *Rym 1-2* allele for resistance to RYMV. *Rym1-2* was efficient against major strains of RYMV, but had severe infection with RYMV isolates from northwest Madagascar.

RYMV resistance in Azucena and IRAT177 is polygenic (Ghesquière et al 1997). Analysis of quantitative trait locus (QTL) on the resistance on populations from Azucena x IR64 and IRAT177 x Apura revealed close linkage on chromosome 12, indicating that a single region on chromosome 12 confers the RYMV resistance in Azucena and IRAT177. The QTL corresponds to a small chromosomal segment known to contain a cluster of major blast resistance genes (Ghesquière et al 1996, 1997). The QTLs were further analyzed on double haploid populations of IR64 x Azucena (Albar et al 1998, Pressoir et al 1998, Ahmadi et al 2001). Fifteen QTLs were detected on seven chromosomes. A QTL located on chromosome 12 and another QTL on chromosome 7 appeared to be the major genetic factor controlling virus content in infected plants.

Cultivar reactions to RYMV were different among locations tested. Wita7 and Wita8 were susceptible in Côte d'Ivoire but were tolerant in Burkina Faso and Mali (Coulibaly 1999, N'Guessan et al 2001). Ita212 and Ngoyumaboi were scored resistant (Abo et al 1998, Coulibaly 1999) or susceptible depending on locations tested. When RYMV isolates were transferred serially up to eight passages by mechanical inoculation on cultivar Gigante or Azucena, symptom severity and virus content in infected tissues with the advance of passages increased (Fargette et al 2002). The RYMV variant C14* transferred via six passages on Gigante seedlings had a single nucleotide substitution in the putative VPg domain of C14 RNA (Fargette et al 2002, 2004). Based on the single-nucleotide polymorphism, the

mutant was detected in plants showing milder symptoms during the passages (He'brard et al 2006). The mutant developed symptoms in Gigante.

When 58 RYMV isolates collected in the fields in Côte d'Ivoire were inoculated to eight cultivars including Gigante and partially resistant Moroberekan, Lac23, Faro11, and ITA235, three isolates induced symptoms on Gigante while 30 to 50% of the isolates induced symptoms on partially resistant cultivars (Sorho et al 2005). Occurrence of resistance-breaking isolates in fields where resistant cultivars have never been planted earlier indicated that the resistance would not be durable. When the resistance breaking mutant was serially transferred via four passages on IR64, it did not change its symptom severity.

A genetically modified line encoding the RNA polymerase gene from RYMV showed partial resistances to RYMV and produced less RYMV in infected plants (Pinto et al 1999, Sorho et al 2005). The then West Africa Rice Development Association (WARDA), now AfricaRice, obtained interspecific hybrid lines in crosses between *O. glaberrima* and rice cultivars by the embryo rescue. The hybrid lines were called new rice for Africa (NERICA) and boast of higher yields (by 50% without fertilizer and by more than 200% with fertilizer), earlier maturity (by 30 to 50 days), resistance to local stresses, and higher protein content (by 2%).

2.10. Control

Chemical control of the vector beetles is not economically and environmentally feasible and thus not adopted for RYM (Bakker 1974). The presence of a large number of vector species makes chemical control of the disease troublesome and less feasible (Calvert et al 2003). Rouging of infected rice plants and eradication of infected rice stubble and volunteer rice plants have been considered effective in reducing RYM incidence (Bakker 1974). Dispersal of RYM by mechanical infection occurs readily at transplanting during weeding and at the harvest (Bakker 1974). There are several chrysomelid beetles that transmit RYMV in rice. Rats, domestic cows, donkeys, and humans are also potential transmitters (Sarraf and Peters 2003). Cultivar resistance has been the most practical means to control RYM. However, many resistant cultivars may not be stable and may be less resistant or even susceptible in other locations.

It is likely that seedling infection occurs primarily after transplanting. When RYMV-infected fields are plowed and puddled after rice harvest rice for transplanting, infected rice stubble is damaged and releases RYMV into flooding water making the water virus infective (Tsuboi 1999). Uprooted seedlings had infection after transplanting in such fields. When seedlings were transplanted soon after land preparation, the risk for seedling infection was high. However, when seedlings were transplanted 2 weeks after the land preparation, the risk was greatly reduced. When rice fields affected with RYM at about 20% were plowed using a cultivator or a hoe, and transplanted with RYM-free seedlings soon after the land preparation, RYM incidence was about 6% (Tsuboi 1999). When transplanted 7 days after the preparation, the incidence was reduced to half. When transplanted 2 weeks after, the incidence was reduced to 1/3 or 1/6. Thus, to lower seedling infection with RYMV through flooding water, transplanting of seedlings more than 2 weeks after land preparation was proposed.

Integrated management practices recommended for RYMV in Côte d'Ivoire include:

- rouging (selected removing of infected plants) at the early growth stage;
- in RYMV affected fields, harvesting rice plants right above the ground level and keeping soil dry after the harvest to reduce development of sprout from rice stubble;
- in the fields where RYM symptoms developed on rice stubbles, plowing the fields and killing RYM-infected stubble before flooding the fields with water;

- plowing and puddling land for transplanting and waiting for 2 weeks or more for transplanting;
- selecting rice seeds of higher gravity to eliminate unfilled grains, some of which are infected with RYMV.

If such practices are difficult to practice, flooding the fields and directly broadcasting germinated seeds are recommended. In RYMV-prone areas, these management practices have been disseminated to farmers and extension people with major success (Tsuboi 1999).

References

- Abo, M. E. 1998. Studies on the mode of transmission of rice yellow mottle virus, PhD thesis. Zaria, Nigeria: Ahmadu Bello University.
- Abo, M., Alegbejp, M., Sy, A., Misari, S. 2000. An overview of the mode of transmission, host plants and method of detection of rice yellow mottle virus. *J. Sustainable Agric.* 17:19-36.
- Abo, M. E., Sy, A. A., Alegbejo, M. D. 1998. Rice yellow mottle virus (RYMV) in Africa: evolution, distribution, economic significance on sustainable rice production and management strategies. *Journal of Sustainable Agriculture*, 11(2/3):85-111, 146.
- Abubakar, Z., Ali, F., Pinel, A., Traore', O., N'Guessan, P., Notteghem, J., Kimmins, F., Konate', G., Fargette, D. 2003. Phylogeography of Rice yellow mottle virus in Africa. *J. Gen. Virol.* 84:733-743.
- Ahmadi, N., Albar, L., Pressoir, G., Pinel, A., Fargette, D., Ghesquiere', 2001. A genetic basis and mapping of the resistance to Rice yellow mottle virus. III. Analysis of QTL efficiency in introgressed progenies confirmed the hypothesis of complementary epistasis between two resistance QTLs. *Theor. Appl. Genet.* 103:108-1092.
- Albar, L., Lorieux, M., Ahmadi, N., Rimbault, I., Pinel, A., Sy, A. A., Fargette, D., Ghesquiere', A. 1998. Genetic basis and mapping of the resistance to rice yellow mottle virus. I. QTLs identification and relationship between resistance and plant morphology. *Theor. Appl. Genet.* 97:1145-1154.
- Albar, L., Ndjiondjop, M. -N., Esshak, Z., Berger, A., Pinel, A., Jones, N., Fargette, D., Ghesquiere', A. 2003. Fine genetic mapping of a gene required for Rice yellow mottle virus cell-to-cell movement. *Theor. Appl. Genet.* 107:371-378.
- Attere, A. F., Fatokun, C. A. 1983. Reaction of *Oryza glaberrima* accessions to rice yellow mottle virus. *Plant Dis.* 67:420-421.
- Awoderu, V. A. 1987. *FAO Plant Prot. Bull.* 35:32-33.
- Awoderu, V.A., 1991. Rice yellow mottle virus in West Africa. *Trop. Pest Management* 37:356-362.
- Bakker, W. 1970. Rice yellow mottle, a mechanically transmissible virus disease of rice in Kenya. *Neth. J. Plant Path.* 76:53-63.
- Bakker, W. 1971. Three new beetle vectors of rice yellow mottle virus in Kenya. *Neth. J. Pl. Path.* 77:201-206.
- Bakker, W. 1974. Characterization and ecological aspects of rice yellow mottle virus in Kenya. *Agricultural Research Reports* 829, Centre for Agricultural Publishing and Development, Wageningen, Netherlands, 152 p.
- Bakker, W. 1975. Rice yellow mottle virus. *C.M.I./A.A.B. Description of Plant Viruses* No. 149.
- Banwo, O. O., Makundi, R. H., Abdallah, R. S., Mbapila, J. C. 2001. Bionomics of vectors and dynamics of rice yellow mottle virus in Tanzania. *IRRN* 26(2):41-42.
- Bonneau, C., Brugidou, C., Chen, L., Beachy, R. N., Fauquet, C. 1998. Expression of the rice yellow mottle virus P1 protein in vitro and in vivo and its movement involvement in virus spread. *Virology* 244:79-86.

- Bussiere, F., Lafontaine, D., Perreault, J-P. 1996. Complication and analysis of viroid-like RNA sequences. *Nucleic Acids Res.* 24:1793-1798.
- Calvert, A. Koganezawa, H., Fargette, D., Konate, G. 2003. Pp. 269-293 in: G. Loebenstein, G. Thottappilly, eds., *Virus and Virus-like Diseases of Major Crops in Developing Countries*. G. Kluwer Academic Publishers, Netherlands.
- Catherall, P. L. 1970. Phleum mottle virus. *Plant Pathol.* 19:101-103.
- Collins, R. F., Gellatly, D. L., Sehgal, O. P., Abouhaider, M. G. 1998. Self-cleaving circular RNA associated with rice yellow mottle virus is the smallest-viroid-like RNA. *Virology.* 241:268-175.
- Coulibaly, M. M. 1999. Etude de la resistance du riz (*Oryza* sp.) au virus de la panachure Jaune du riz (RYMV). Ph.D. Thesis, Ouagadougou University, Burkina Faso.
- Fargette, D., Pinel, A., Traore, O., Ghesquiere, A., Konate, G. 2002. Emergence of resistance-breaking isolates of *Rice yellow mottle virus* during serial inoculations. *Eur. J. Plant Pathol.* 108:585-591.
- Fargette, D., Pinel, A., Abubakar, Z., Traore, O., Brugidou, C., Fatogoma, S., He'brard, E., Choisy, M., Se're, Y., Fauquet, C., Konate, G. 2004. Inferring the evolutionary history of Rice yellow mottle virus from genomic, phylogenetic, and phylogeographic studies. *J. Virol.* 78:3252-3261.
- Fauquet, C., Thouvenel, J. C. 1977. Isolation of rice yellow mottle virus in Ivory Coast. *Plant Dis. Repr.* 61:443-446.
- Fomba, S. N. 1988. Screening for seedling resistance to rice yellow mottle virus in some rice cultivars in Sierra Leone. *Plant Dis.* 72:641-642.
- Fomba, S. N. 1990. Rice yellow mottle virus on swamp rice in Guinea. *IRRN* 15:21.
- GRiSP (Global Rice Science Partnership). 2013. **Rice almanac, 4th edition**. J. Maclean, B. Hardy, G.P. Hettel, eds. Los Baños, (Philippines): International Rice Research Institute. 283 p.
- He'brard, E., Pinel-Galzi, A., Bersoult, A., Sire, C., Fargette, D. 2006. Emergence of a resistance-breaking isolate of Rice yellow mottle virus during serial inoculations is due to a single substitution in the genome-linked viral protein VPg. *J. Gen. Virol.* 87:1369-1373.
- IITA. 1980. International Institute of Tropical Agriculture Annual Report for 1979, Ibadan, Nigeria, 137 p.
- Ioannidou, D., Lett, J. M., Pinel, A., Assigbe'tse, K., Brugidou, C., Ghesquiere, A., Nicole, M., Fargette, D. 2000. Responses of *Oryza sativa* Japonica sub-species to infection with *Rice yellow mottle virus*. *Physiol. Mol. Plant Pathol.* 57:177-188.
- John, V. T., Thottappilly, G., Awoderu, V. A. 1984. Occurrence of rice yellow mottle virus in some Sahelian countries in West Africa. *FAO Plant Prot. Bull.* 32:86-87.
- John, V. T., Thottappilly, G., Ng, Q., Alluri, K., Gibbons, J. W. 1985. Varietal reaction to rice yellow mottle disease. *FAO Plant Prot. Bull.* 33:109-111.
- John, V. T., Thottappilly, G. 1987. A scoring system for rice yellow mottle virus disease (YMV). *IRRN* 12(3):26.
- John, V. T., Thottappilly, G., Masajo, T. M., Reckhaus, P. M. 1988. Rice yellow mottle virus disease in Africa. Fifth International Congress of Plant Pathology, Kyoto, Japan, August 20-27.
- Konate, G., Sarra, S., Traore, O. 2001. Rice yellow mottle virus is seed-borne but not seed transmitted in rice seeds. *Eur. J. Plant Pathol.* 107:361-364.
- Konate, G., Traore, O., Coulibaly, M. 1997. Characterization of rice yellow mottle virus isolates in Sudano-Sahelian areas. *Arch. Virol.* 142:1117-1124.
- Mansour, A. N., Baillis, K. W. 1994. Serological relationships among rice yellow mottle virus isolates. *Ann. Appl. Biol.* 125:133-140.
- Ndjiondjop, M. N., Albar, L., Fargette, D., Fauquet, C., Ghesquiere, A. 1999. The genetic basis of high resistance to rice yellow mottle virus (RYMV) in cultivars of two cultivated rice species. *Plant Dis.* 83:931-935.

- Ndiondjop, M. N., Brugidou, S. Z., Fargette, D., Ghesquiere, A., Fauquet, C. 2001. High resistance to rice yellow mottle virus in two cultivated rice cultivars is correlated with failure of cell to cell movement. *Physiol. Mol. Plant Pathol.* 59:309-316.
- N'Guessan, P., Pinel, A., Sy, A., Ghesquiere, A., Fargette, D. 2000. Evidence of the presence of two serotypes of rice yellow mottle sobemovirus in Cote d'Ivoire. *Eur. J. Plant Pathol* 167-178.
- N'Guessan, P., Pinel, A., Sy, A., Ghesquiere, A., Fargette, D. 2001. Distribution, pathogenicity and interactions of two strains of *Rice yellow mottle virus* in forested and savanna zones of West-Africa. *Plant Dis.* 85:59-64.
- Okioma, S. N. M., Muchoki, R. N., Gathuru, E. M. 1983. Alternative hosts of rice yellow mottle virus in the Lake Victoria Basin of Kenya. *Rop. Pest Manage.* 29:295-296.
- Okioma, S. N. M., Sarkarung, S. 1983. Screening rice varieties for resistance to rice yellow mottle virus disease. *Trop. Pest Management* 29:145-147.
- Opalka, N., Brugidou, C., Bonneau, C., Nicole, M., Beachy, R. N., Yeager, M., Fauquet, C. 1998. Movement of rice yellow mottle virus between xylem cells through pit membranes. *Proc. Nat. Acad. Sci. USA* 95:3323-3328.
- Purseglove J.W., 1972, *Oryza* L. In Pp. 161-198: J.W. Purseglove, ed. *Monocotyledons* 1. Longman, London.
- Pinel, A., Nguessen, R., Pousalem, M., Fargette, D. 2000. Molecular variability of geographically distinct isolates of Rice yellow mottle virus in Africa. *Arch. Virol.* 145:1521-1638.
- Pinel, A., Traore, G., Abubakar, Z., Konate, G., Fargette, D. 2003. Molecular epidemiology of the RNA satellite of the Rice yellow mottle virus. *Arch. Virol.* 148:1721-1733.
- Pinto, Y. M., Kok, R. A., Baulcoumbe, D. C. 1999. Resistance to rice yellow mottle virus (RYMV) in cultivated African rice varieties containing RYMV transgenes. *Nature Biotech.* 17:702-707.
- Pressoir, G., Albar, L., Ahmadi, N., Rimbault, I., Lorieux, M., Fargette, D., Ghesquiere, 1998. A. Genetic basis and mapping of the resistance to rice yellow mottle virus. II. Evidence of a complementary epistasis between two QTLs. *Theor. Appl. Genet.* 97:1155-1161.
- Qu, C., Lars, L., Opalka, N., Brugidou, C., Yeager, M., Beachy, R., Fauquet, C., Johnson, J., Lin, T. 2000. 3D domain swapping modulates the stability of members of an icosahedral virus group. *Structure* 8:1095-1103.
- Rakotomalala, M., Pinel-Galzi, A., Albar, L., Ghesquiere, A., Rabenantoandro, Y., Ramavovololona, P., Fargette, D. 2008. Resistance to Rice yellow mottle virus in rice germplasm in Madagascar. *Eur. J. Plant Pathol.* 122:277-286..
- Raymundo, S. A., Buddenhagen, I. W. 1976. A rice virus disease in West Africa. *Int. Rice Comm. Newsl.* 25:58.
- Raymundo, S. A., Konteh, I. K. 1980. Distribution, importance, screening methods and varietal reaction to rice pale yellow mottle disease. *Int. Rice Comm. Newsl.* 29(1):51-53.
- Reckhaus, P. M., Randrianangaly, S. 1990. Rice yellow mottle virus (RYMV) on rice in Madagascar. *IRRN* 15(1):30.
- Reckhaus, P. M., Andriamasinthseho, H. E. 1997. Rice yellow mottle virus in Madagascar and its epidemiology in northwest of the island. *Z. pflanzenkrankh. Pflanzenschutz.* 104:289-195.
- Rossel, H. W., Ayotade, K. A., Thottappilly, G., Adeoti, A. A., Alluri, K., Alam, M. S., Kaung, Z. 1982a. A new record of rice yellow mottle virus disease in Badeggi, Nigeria. *Int. Rice Comm. Newsl.* 31:23-24.
- Rossel, H. W., Thottappilly, G., Buddenhagen, I. W. 1982b. Occurrence of rice yellow mottle virus in two important rice-growing areas of Nigeria. *FAO, Plant Prot. Bull.* 30:137-139
- Rossel, H. W. 1986. Rice yellow mottle and African soybean dwarf, newly discovered virus diseases of economic importance in West Africa. *Trop. Agric. Res. Ser.* 19:146-153, *Trop. Agr. Res. Cent.* Tsukuba, Japan.

- Sarra, S., Peters, D. 2003. *Rice yellow mottle virus* is transmitted by cows, donkeys, and grass rats in irrigated rice crops. *Plant Dis.* 87:804-808.
- Sehgal, O. P., AbouHaidar, M. G., Gellatly, D. L., Ivanov, I., Thottappilly, G. 1993. An associated small RNA in rice yellow mottle sobemovirus homologous to the satellite RNA of Lucerne transient streak sobemovirus. *Phytopathology* 83:1309.
- Serjeant, E. P. 1967. Some properties of cocksfoot mottle virus. *Ann. Appl. Biol.* 59:31-38.
- Sorho, F., Pinel, A., Taore', O., Bersoult, A., Ghesquie're, A., He'brard, E., Konate', G. Se're, Y., Fargette, D. 2005. Durability of natural and transgenic resistances in rice to *Rice yellow mottle virus*. *Eur. J. Plant Pathol.* 112:349-359.
- Taylor, D. R. 1989. Resistance of upland rice varieties to pale yellow mottle virus (PYMV) disease in Sierra Leone, IRRN 14(1):11.
- Taylor, D. R., Fofie, A. S., Suma, M. 1990. Natural infection of rice yellow mottle virus disease (RYMV) on rice in Sierra Leone, IRRN 15:19.
- Taore, O., Pinel, A., Fargette, D., Fance, I., Konate, G. 2001. First report and characterization of rice yellow mottle virus in Central Africa. *Plant Dis.* 85:920.
- Thottappilly, G., Rossel, H. W. 1993. Evaluation of resistance to rice yellow mottle virus in *Oryza* species. *Indian J. Virol.* 9:65-73.
- Tsuboi, T. 1999. Cultural control of rice yellow mottle virus in Cote d'Ivoire (In Japanese). *Outlines of Japan's Overseas Cooperation in Agriculture, Forestry and Fisheries* 19(5):1-14.
- Tsuboi, T., Goto, A., Boua, B., Kato, H. 1996. Epidemiology of rice yellow mottle virus disease in Cote d'Ivoire (In Japanese). *Ann. Phytopathol. Soc. Jpn.* 62:323-324.
- WARDA, 2000. Rice yellow mottle virus. Pp. 27-37 in: West Africa Rice Development Assoc. Annu. Report 2000.
- World Rice Statistics. 1985. International Rice Research Institute. Philippines.
- Yassi, M. N. A., Ritzenthaler, C., Brugidou, C., Fauquet, C., Beachy, R. N. 1994. Nucleotide sequence and genome characterization of rice yellow mottle virus RNA. *J. Gen. Virol.* 75:249-257.