Peanut Bud Necrosis Disease: An Overview

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Abstract

Peanut bud necrosis disease (PBND) was first recorded in India in 1949. The economic importance of the disease was realized during the late 1960s when incidences up to 100% were recorded in many groundnut-growing regions in India. The disease has been described under different names. It was shown to be economically important in parts of Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, and Uttar Pradesh. Although it was earlier reported to be caused by tomato spotted wilt virus (TSWV), recently, the causal virus of PBND in India was shown to be a serologically distinct tospovirus, now referred to as peanut bud necrosis virus (PBNV), transmitted by Thrips palmi. *Surveys in many groundnut-growing countries indicate that PBNV is restricted to South and Southeast Asia. Several cultural practices are available to control the disease. Excellent progress has been made in the identification of sources of field resistance.*

The PBNV genome contains three RNA species and the sRNA has recently been sequenced and the two genes it codes for have been identified. Progress achieved will lead to the production of high quality diagnostic aids and for the development of transgenic resistance. Future research will focus on epidemiology, development of early-maturing resistant cultivars, sequencing of the entire viral genome, the production of high quality diagnostic aids, and assessment of biodiversity among PBNV isolates.

Introduction

The occurrence of a disease with symptoms similar to those of peanut bud necrosis disease (PBND) was mentioned in the Annual Report of the Indian Agricultural Research Institute in 1949. This appears to be the first record of occurrence of PBND in India. The name "Bud Necrosis" was given in 1968 and the disease was considered to be distinct at that time because none of the other groundnut viruses reported until 1968 were known to produce the bud necrosis symptom (Reddy et al. 1968). To our knowledge, PBND has been described in India since 1962 under at least seven different names: groundnut mosaic, groundnut rosette, bunchy top, chlorosis, ring mottle, bud blight, and ring mosaic (Reddy 1988). ICRISAT has conducted regular surveys in the major groundnut-growing areas of India from 1976 to 1982, and occasional surveys till 1992. The disease was

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apparently economically important in parts of Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, and Uttar Pradesh. Peanut bud necrosis disease is also currently recognized as economically important in parts of China, Nepal, Sri Lanka, and Thailand. Its distribution appears to be restricted to Asia. Losses due to PBND have been estimated at over 89 million US $ per annum (ICRISAT 1992).

Symptoms

Symptoms produced by peanut bud necrosis virus (PBNV) in groundnut are difficult to distinguish, if at all, from those caused by tomato spotted wilt virus (TSWV). Initial symptoms appear on young quadrifoliolates as mild chlorotic mottle or spots, which develop into necrotic and chlorotic rings and streaks. Necrosis of the terminal bud, a characteristic symptom, occurs on crops grown in the rainy and postrainy seasons, when ambient temperatures are relatively high. Secondary symptoms are stunting, axillary shoot proliferation, and malformation of leaflets. If plants are infected early, they are stunted and bushy. If plants older than 1 month are infected, the symptoms may be restricted to a few branches or to the apical parts of the plants.

Due to the severity of the symptoms, the virus causes severe losses to the groundnut crop, especially when plants are infected before they are a month old. Seeds from such plants are small, shriveled, mottled, and discolored. Late-infected plants may produce seed of normal size. However, the testae on such seed are often mottled and cracked.

Causal Virus

Until 1990, PBND in India was reported to be caused by TSWV (Reddy et al. 1991). High-quality antisera became available for the detection of tospoviruses, to which the group TSWV belongs, only during the late 1980s. Data from serological comparisons and subsequently from sequencing of nucleic acids revealed the existence of several distinct tospoviruses (German et al. 1992, de Avila et al. 1993).

In 1992, the virus causing PBND was identified as a distinct tospovirus and named PBNV. With ELISA as well as Western blots, PBNV was shown to be serologically distinct from TSWV and Impatiens necrotic spot virus (INSV) (Reddy et al. 1992).

PBNV contains three RNA species of about 9.0 kb (1RNA), 5.0 kb (mRNA), and 3.0 kb (sRNA) (D.V.R. Reddy and S. Gowda, unpublished). Recently sRNA has been sequenced and the details will be provided elsewhere in these proceedings (Satyanarayana et al. 1995).

Transmission

Sap transmission. Peanut bud necrosis virus can be transmitted by mechanical sap inoculations if care is taken to extract the virus only from young infected leaflets with primary symptoms. Extracts should be prepared in neutral phosphate buffer containing an antioxidant such as merceptoethanol, and must be kept cold throughout the inoculation process.

Thrips transmission. Amin et al. (1981) reported that the virus causing PBND in India is transmitted by *Frankliniella schultzei* and *Scirtothrips dorsalis.* Subsequent investigations, which involved accurate identification of thrips, showed that in fact *Thrips palmi* transmits PBNV, and not *F. schultzei* or S. *dorsalis,* which are also present on the plants. Further experiments showed that T. *palmi* could acquire PBNV as larvae and transmit it as adults. Maximum transmission (100%) was obtained when there were 10 adults per plant. The majority of individual adult thrips transmitted the virus for more than half of their life period, indicating the degree of erratic transmission. Cowpea was found to be the best host for rearing and multiplying *T. palmi* under laboratory conditions (Vijaya Lakshmi 1994, Wightman et al. 1995, these proceedings).

Diagnosis

Several methods can be used for the diagnosis of PBNV. The following are recommended, especially for developing countries.

- Sap inoculations on to cowpea (cv C-152) and *Petunia hybrida.* Cowpea produces concentric chlorotic and necrotic lesions; *Petunia* produces necrotic lesions (Reddy et al. 1991).
- ELISA using polyclonal antibodies. They clearly distinguish PBNV from TSWV and INSV (Reddy et al. 1992).
- Presence of typical tospovirus particles in leaf extracts. Even in leaf dip preparations, if young tissues showing initial symptoms are used, PBNV particles can be observed. They are 80-100 nm in diameter, and are surrounded by a double membrane of protein and lipid.

Management of PBND

Several cultural practices such as adjustments to sowing dates, sowing at the recommended rate, adopting measures to maintain plant population, intercropping with fast-growing cereal crops such as maize and pearl millet can reduce the incidence of PBND. These practices have been shown to reduce infestation by *T. palmi.*

Roguing of infected plants, especially during early stages of plant growth, should be avoided because this practice creates gaps in the field and can increase PBND incidence.

Excellent progress has been made in the identification of sources of field resistance to PBND. Since this aspect will be covered in two presentations (see Buiel et al. 1995, Dwivedi et al. 1995, these proceedings), we do not wish to deal with it here. Although many high-yielding PBNV-resistant varieties have been developed, they are medium-maturing types. Some of the field-resistant genotypes such as ICGV 86388, show resistance to PBNV and less colonization by vector thrips compared with susceptible genotypes (Buiel et al. 1995, Dwivedi et al. 1995, these proceedings). Cultivars such as ICGS 11, Kadiri 3, and ICGS 44 are field resistant to PBND.

Future Research

Peanut bud necrosis virus and *T. palmi* have extremely wide host ranges. Therefore, the virus is a potential threat to cropping systems which include legumes, vegetables, and ornamentals. Some of these crops are grown under irrigation and protected with insecticides. These conditions are likely to result in a gradual buildup of PBNV inoculum, leading to disease epidemics. Therefore, it is essential to closely monitor the incidence of PBNV in various cropping systems, which include highly susceptible hosts of the virus and the vector.

Since the field-resistant groundnut varieties are of medium duration, attempts should be made to breed early-maturing cultivars for environments where they are needed, To achieve this rapidly, transgenic groundnuts expressing PBNV genes could be developed. sRNA of PBNV has been fully sequenced and the coat protein gene located (Satyanarayana et al. 1995, these proceedings) for utilization in the transformation and regeneration of groundnut.

Thrips are known to have several parasites and predators. They have not been tested for their effectiveness to reduce populations of *T. palmi.* It would also be useful to study the effect of a range of synthetic and natural insecticides on the parasites and predators of *T. palmi,* once they are identified.

Peanut bud necrosis virus is currently known to cause economic losses to many commercial crops other than groundnut. These include chilli, potato, tomato, tobacco, and early-maturing legumes such as mung bean and urd bean. Data generated for the management of PBNV on groundnut are likely to be applicable to these crops. Due to the specialized skills required for the detection of PBNV, the economic importance of PBNV in many other high-value crops has not been realized.

Variation among isolates of PBNV has not yet been fully investigated. This should be carried out because of its implication for the durability of host-plant resistance. Limited tests conducted in the case of groundnut (Buiel et al. 1995, these proceedings) indicate that the resistance is effective in India in different ecoregions. These tests should be extended to PBNV hot spots in other South Asian countries.

Thrips palmi has recently been detected in large populations in many southeastern states of USA. In these states, currently, TSWV is considered to be economically important. Tb date, we have no evidence of the occurrence of PBNV in the USA or in other countries where *T. palmi* may be occurring in large populations. The potential threat from PBNV therefore exists in such places, indicating the need to conduct surveys for the occurrence of PBNV.

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