Guava (Psidium guajava Linn.) is an important fruit crop of subtropical countries. In India, it is grown almost in all the states. Bihar has the largest area (24.7 thousand ha.) under guava cultivation followed by Uttar Pradesh (18.5 thousand ha.) and Maharashtra (14.8 thousand ha.). The per ha productivity is the highest in Madhya Pradesh (20t/ha) compared to very low in Uttar Pradesh (7.3t/ha). It is a hardy crop and is cultivated successfully even in neglected soils and is attacked by a large number of pathogens, mainly fungi. About 177 pathogens (167 fungi, 3 bacteria, 3 algae, 3 nematodes and one epiphyte) are reported on various parts of the crop, including fruits, causing various diseases. Wilt is the most destructive. In India losses due to this disease are substantial. The disease is soil-borne and is difficult to control.

Wilt of guava was first reported in 1935 from Allahabad. To understand the disease some work was done at Allahabad Agricultural Institute, Naini under the leadership of Dr. J. C. Edward and Bidhan Chandra Agriculture University, Kalyani under the leadership of Dr. S. B. Chattopadhyay. In the recent past some efforts were made at Central Institute for Subtropical Horticulture, Lucknow and Horticulture Agroforestry Research Programme, Ranchi for the better understanding of the disease. The work on this problem was reviewed by Misra and Prakash, 1990; Misra, 1995, 2001, 2005; Misra and Pandey, 1996; Negi et al., 2001. The present communication reviews the present status of the guava wilt disease.

After the first report of guava wilt in 1935 from Babakkarpur, Allahabad, Das Gupta and Rai (1947) recorded the disease in the severe form in the orchards of Lucknow. Dey (1948) reported it from Allahabad, Kanpur and Lucknow. During 1949-50, guava trees suffered serious losses in 11 districts of UP (Anonymous, 1949, 1950). Prasad et al. (1952) estimated that guava wilt spread rapidly to cover about 20,000 sq. m area in UP. Mathur (1956) reported upto 30 percent trees affected with wilt in Allahabad, Farrukhabad, Unnao (15-30%), Kanpur and Jaunpur (5-15%) and less than 5 per cent in Gorakhpur, Ballia, Hardoi, Barabanki and Varanasi. Edward and Srivastava (1957) reported wilt as the most serious disease threatening guava cultivation in UP. Later, it was also reported from western parts of UP (Singh and Lal, 1953), Varanasi (Pandey and Dwivedi, 1985), Kaimganj (Farrukhabad), Bithoor (Kanpur), Ganga Ghat (Unnao), Abbubakarpur (Allahabad), Lucknow, Bichpuri (Agra), Sasni (Aligarh) (Misra and Prakash, 1986; Misra, 1987). In West Bengal the disease spread in the Gangetic alluvial region of Baruiupur area in the district of 24 Parganas and in the laterite zone of Jhargram and Midnapur (Chattopadhyay and Sen Gupta, 1955). Chattopadhyay and Bhattacharjya, (1968a,b) reported the disease from Kashakul, Bankura. The disease was also reported from Haryana (Suhag, 1976; Mehta, 1987), Punjab (Chandra Mohan et al., 1986), Rajasthan (Kayal, 1972 Bhargava et al. 2003), Delhi state (Anonymous, 1953), Jharkhand (Srivastava et al., 2001), Andhra Pradesh (Jhooity et al., 1984), M.P. in the Hatod area near Indore - (Personal observation), Orissa (Das et al.,1993) and Thanjavur district of Tamil Nadu (Personal observation) (Map-1).

Wilted guava plants have also been reported from Florida, U.S.A. (Webber, 1928), Taiwan (Hsieh et al., 1976; Leu and Kao, 1979), Cuba (Rodriguez and Landa, 1977), South Africa (Grec, 1985, Vos et al., 2000), Brazil (Tokeshi et al., 1980; Rodriguez et al., 1987 Junqueira et al., 2001), Pakistan (Ansar et al., 1994), Bangladesh (Hamiduzzaman
et al., 1997), and Canberra, Australia (Lim and Manicom, 2003) (Map 2).

**Losses**

Singh and Lal (1953) estimated 5-15 percent loss amounting to almost 1 million rupees due to guava wilt every year in 12 districts of U.P. In West Bengal, the disease reduced the yield by 80 percent i.e., from 113.5q/ha in healthy plantations to about 18.16-22.7q/ha in affected orchards (Chattopadhyay and Sen Gupta, 1955). Chattopadhyay and Bhattacharjya (1968a,b) attempted in vain to regenerate the affected trees. The new seedlings, grafted or planted in the affected areas showed stunted growth, flowered rarely and succumbed to wilt within a very short time. Seven thousand acres of land in Andhra Pradesh under guava cultivation, reduced to half the land value by the presence of the disease (Jhooty et al., 1984). About 150 and 300 acres of guava wilt affected orchards in Punjab and Haryana respectively were uprooted during 1978-81 (Jhooty et al., 1984). In general, losses due to wilt in guava around Lucknow area vary from 5-60 per cent (Misra and Shukla, 2002).

**Symptomatology**

The affected plants show yellow colouration with slight leaf curling at the terminal branches, becoming reddish at the later stage and subsequently premature shedding of leaves take place. Twigs become bare and fail to bring forth
new leaves or flowers and eventually dry up. Fruits of all the affected branches remain underdeveloped, become hard, black and stony. The entire plant becomes defoliated and eventually dies. It requires almost sixteen days for complete wilting. Some affected trees linger on even up to 252 days and then die (Misra and Pandey, 2000b). Misra and Pandey (2000b) also studied variations in the symptoms during different time of the year. They noticed yellowing of the leaves with inter-veinal chlorosis during the month of August, which drop even with the slight shaking of the plants. During September, general drooping of the leaves takes place. During October, complete wilting of plants are seen with almost dried leaves and small dried black fruits hanging on the branch. A few plants also show partial wilting, which is a very common symptom of wilt in guava. Some plants may also show variable degree of wilting, leaf yellowing, drooping of leaves, drying of terminal branches or partial wilting during different months but later escape/resist wilting. These plants start recovering from December onward. It was recorded that out of total wilting plants, around 17 per cent plants, which initially show some symptoms of wilting, ultimately escape/resist wilting (Misra and Pandey, 2000b).

The finer roots show black streaks, which become prominent on removing the bark (Das Gupta and Rai, 1947). The roots also show rotting at the basal region and the bark is easily detachable from the cortex. The cortical regions of the stem and root show distinct discoloration and damage. Light brown discoloration is noticed in vascular tissues (Chattopadhyay and Bhattacharya, 1968a,b). Wilted plants later show bark splitting. The pathogen attacks young as well as old fruit bearing trees but older trees are more prone to the disease (Misra and Shukla, 2002). New seedlings and grafts also show disease symptoms (Singh and Lal, 1953; Edward, 1960a). Chakraborty and Singh (1989) identified mainly two types of symptoms i.e. slow wilt, where plant takes several months or even a year or two to wilt after the appearance of initial symptoms and sudden wilt, where plant takes 15 days to one month to wilt after the appearance of initial symptoms.

Causal organism

The exact causal agent of the disease is still not fully understood. Various pathogens were involved with the affected plants viz. *Fusarium oxysporum* f. sp. *psidii*, *F. solani*, *Macrophomina phaseolina*, *Rhizoctonia bataticola*, *Cephalosporium* sp., *Gliocladium roseum* and *Verticillium albo-atrum* etc. have been reported by different workers. Vestal (1941) reported *Cephalosporium* sp. to be the causal organism of wilt of guava. Dey (1948) also invariably isolated *Cephalosporium* from roots of wilted plants. Das Gupta and Rai (1947) for the first time reported the association of *Fusarium* sp. Later, Prasad *et al.* (1952) attributed wilt due to *Fusarium oxysporum* (Fr.) Schl. and proposed the name *Fusarium oxysporum* (Fr.) Schl. f. sp. *psidii* Prasad, Mehta and Lal. It was supported by Edward and Srivastava (1957) and Pandey and Dwivedi
Edward (1960b,c) explained that *F. oxysporum* f. sp. *psidii* penetrate either directly through the root piliferous layer of the guava seedlings or through openings caused by secondary roots. Hyphae were found in the xylem vessels of the roots of the inoculated plants. He also observed that *F. oxysporum* f. sp. *psidii* existed in a variety of forms, which differ in cultural and morphological characters.

Tandon and Agarwal (1954) on the other hand reported wilt and dieback of guava to be caused by *Gloeosporium psidii* from Allahabad. The fungus penetrated the petiole and attacked the young leaves, which became distorted with dead areas at the margins or tips and in severe cases, died. Pink spore mass of fungus was found on twigs. These findings were closer to die back and not wilt. In West Bengal, both *Macrophomina phaseolina* and *F. solani* were reported to be the incitant of wilt either individually or in combination. In either case, the fungus first colonizes the surface of roots and then enters in to its epidermal cells. Thereafter, intercellular mycelium establishes first in epidermal cells and then spreads into cortical cells, which get damaged considerably and filled up with the mycelium. *Fusarium solani* enters the xylem vessels, grows inside and blocks them. *Macrophomina phaseolina* first invades the phloem and destroys it. The xylem vessels are also attacked in a few cases (Chattopadhyay and Bhattacharjya, 1968a,b; Chattopadhyay and Sengupta, 1955). Histopathological observations made by various workers in naturally wilted and artificially inoculated plants revealed the presence of *F. solani*, *F. oxysporum* and *M. phaseolina* in vascular tissues (Chattopadhyay and Bhattacharjya, 1968a,b; Edward, 1960c; Chandra Mohan, 1985; Pandey and Dwivedi, 1985; Sohi, 1983a,b). *Gliocladium vermoesenii* Corda, a known saprophytic fungus, is also found associated with diseased plants (Chandra Mohan, 1985). From Varanasi, *F. oxysporum* f. sp. *psidii*, *F. solani*, *F. coerelium*, *F. moniliforme* and *Rhizoctonia solani* were also found on rhizoplane and rhizosphere of guava (Dwivedi, 1991a; Dwivedi and Dwivedi, 1999). In *in vitro* studies Misra and Pandey (1992) found *Cylindrocarpon lucidum*, *Gliocladium virens* and *Bartlinia robillardoides* (which were isolated from wilted plants) to cause drooping and subsequent wilting of guava seedlings grown in Hoagland's solution in artificial testing. The recent studies at CISH, Lucknow (Misra and Pandey, 1997; Misra and Pandey, 2000a) revealed *Gliocladium roseum* to be the pathogen of guava wilt, which reproduced symptom of wilt on artificial inoculation. The authors and his associates succeeded in reproducing wilt by artificial inoculation in grown up 6-7 years old guava plants by *Gliocladium roseum* by stem hole inoculation technique. They earlier also reported (Misra and Pandey, 1999c) *Gliocladium virens*, *G. penicilloides*, *Fusarium oxysporum*, *F. solani* and *Acremonium* sp. to be associated with guava wilt symptoms indicating it's complex nature. They standardized the stem hole inoculation technique reproducing wilt symptoms quickly. Compared to soil inoculation and root inoculation techniques their stem inoculation method was superior producing quick wilting symptoms within a month. The former methods produced wilting between 3 to 6 months. Pandit and Samajpati, (2002) reported wilt to be caused by *Botyodiplodia theobromae* in Midnapur (W.B.), while Gupta et al. (2003) reported association of *Verticillium albo-atrum* with guava wilt symptoms indicating it's complex nature. They standardized the stem hole inoculation technique reproducing wilt symptoms quickly. Compared to soil inoculation and root inoculation techniques their stem inoculation method was superior producing quick wilting symptoms within a month. The former methods produced wilting between 3 to 6 months.

The reports from other parts of the world are different. Webber (1928) reported *Clitocybe tabescens* killing guava trees in Florida (USA) in 1928. In Cuba three nematodes viz., *Meloidogyne* sp., *Helicotylenchus* sp. and *Pratylenchus* sp. have been found associated (Rodriguez and Landa, 1977) with guava wilt. In Taiwan, the disease is reported to be caused by *Myxosporium psidii* Corda (Hsieh et al., 1976; Leu et al., 1979). Tokeshi et al. (1980) isolated *Pseudomonas* sp. from wilt-affected plants. Disease similar to wilt caused by *Erwinia psidii* was also observed at Sao Paulo (Brazil) in 1982 (Rodrigues et al., 1987) and Planaltina (Jungueira et al., 2001). In South Africa, *Septofusidium* sp. was found associated with the rapid death of guava plants (Grech, 1985). From Pakistan (Punjab) disease is reported in the name of decline and *Fusarium oxysporum* and *Colletotrichum gloeosporiodes* (*Glomerella cingulata*) are considered.
associated with the disease and are supposed to act synergistically when present together (Ansar et al., 1994). Hamiduzzaman et al. (1997) reported from Bangladesh that wilt incidence was maximum when seedlings were inoculated by *F. oxysporum* f. sp. *psidii* along with the nematodes *H. dihystera* and *H. indicus*. From South Africa, Vos et al. (2000) reported guava wilt disease (GWD) caused by *Penicillium vermoeenseni*. Lim and Manicom, (2003) from Australia reported wilt of guava by *F. oxysporum* f. sp. *psidii*.

**Epidemiology**

Mehta (1951) reported severe incidence of wilt in alkaline soils at pH ranging from 7.5 to 9.0, while Sen and Verma (1954) reported high disease incidence in lateritic soils at pH 6.5. A soil saturation of 60-80% has been reported optimum for disease development in west Bengal (Chattopadhyay and Bhattacharjya, 1968b). A pH 6.0 has been reported optimum for the development of the disease. Both pH 4.0 and 8.0 reduces the disease. Low incidence of the disease has been reported at 630 ppm N and is more both at higher as well as at lower levels of nitrogen. Moderate to high concentrations of phosphates (207-345 ppm) are effective in reducing the disease (Chattopadhyay and Bhattacharjya, 1968b). Mehta (1987) reported more disease in clay loam and sandy loam compared to heavy soil types.

Guava seedlings are more susceptible to *Macrophomina phasolina* as well as *F. solani* than the older plants of 3 years age. On the other hand Chattopadhyay and Bhattacharjya (1967) reported that *F. solani* could infect guava plants from one month-old plants to more than 4 years old trees. According to Misra and Shukla, (2002) guava plants above five-year-old were more susceptible to the disease.

Infected guava plants start showing sign of wilting with the onset of rainy season in August with maximum number dying in Sepember and October (Das Gupta and Rai, 1947; Edward, 1960a; Suhag, 1976).

Dwivedi et al. (1990) at Varanasi also found more pathogenic fungi during rainy season. The fungi survived better in association with root bits in adverse climatic conditions in the summer months, while in rainy and winter months they survive on roots. Extensive studies on the progress of natural wilting of guava plants during different months have been made by Misra and Pandey (1999a,d, 2000b) at Lucknow. They found maximum wilting during October. Some plants, which show slight yellowing started recovering from December onwards. On analyzing the weather data, they found higher rainfall during July-September with maximum temperature ranging from 31.3 to 33.5°C, minimum temperature ranging from 23 to 25°C and humidity of 76 percent. They also found that minimum two months are required for the complete wilting of plants (Fig.1).

**Varietal reaction**

Mathur and Jain (1960) reported cultivars White guava No. 6229, Supreme, Clone 32-12, Webber and Popeno from Florida (USA), Hart and Rolf from Florida, acclimatized at Allahabad, Riverside and Rolf from California (USA), Safeda from Sri Lanka, Banarasi (Andhra strain), Dholka, Sindh and Nasik (Bombay strain) were tolerant to wilt. Edward (1961) reported cultivar Chittidar, Hafsi, Safeda Riverside, Rolf and Stone acid to be susceptible and guava species *Psidium cattleianum* var. *lucidum* and other genera *Syzygium cuminii* (Jaman) resistant to wilt. Edward and Gaurishanker, (1964) again reported that *Syzygium cuminii*, *Lagerstromeia indica*, *Psidium cattleianum* (*Psidium molle*), *P. quianense*, Chinese guava (*P. friedrichsthalianum*) and Philippine guava to be resistant to wilt. Stock-scion
compatibility investigations with cv. Safeda as scion and the above resistant material as rootstock proved Lagerstroemia indica incompatible, Syzygium cumini (Jamun) as partially compatible and other compatible. They suggested Chinese guava as rootstock for combating wilt disease. From Basti (U.P.) Singh et al. (1977) reported that among 10 red-fleshed cultivars, only one of Allahabad was found infected by Fusarium solani. Among the 15 white-fleshed cultivars, Lucknow 49 was free from the disease and in Allahabad Safeda incidence was only 4 percent, whereas Karela and Behat Coconut suffer heavily (33%). None of the 7 species, Psidium aracae, P. cattleianum, P. cattleianum var. lucidium, P. coreicum, P. cujavillus, P. quineese and P. fridichsthalianum developed wilt infection. West Bengal, Variety 1 (from Baruipur) was found fairly resistant against F. solani and M. phaseoli. Variety 4 (from Tollygunge) was found moderately resistant to M. phaseolina and variety 5 (from Bankura) was reported to have moderate resistance against both the organisms, either separately or in association (Sen Gupta et al., 1989). Misra (1998-99) studied the relative field tolerance of 20 guava cultivars and categorized them into different groups on the basis of their natural susceptibility. The cultivars Allahabad Safeda, Florida Seedling, Guinees, Hafsi, Karela, Mirzapuri Seedling, Nasik, Pear Shaped, Sindh, Superior and White Fleshe proved highly susceptible; Behat Coconut and Pourtgal as susceptible; Apple Colour, Chittidar, Seedless and Spear Acid, Superior Sour Lucidium, Red Flesh and Smooth Green as tolerant. Misra et al. (2003c) identified F1 population of Psidium molle X Psidium guajava free from wilt when grown in wilt sick plot and inoculated repeatedly with Gliocladium roseum, Fusarium solani and Fusarium oxysporum. A local variety Pei-Pa in Taiwan was reported resistant and Psidium friedrichsthitianum has been reported as possible root stock (Leu and Kao, 1979). The strawberry guava (Psidium cattleianum) has been reported relatively hardy species from Reunion (Normand, 1994). In South Africa, Fan Retief, a most extensively cultivated guava variety has been reported highly susceptible to wilt (Du Preez, 1995). Vos et al. (2000) screened thirty thousand guava seedlings in vitro against Penicillium vermoensennii (guava wilt pathogen) and identified 3 selections which were 100% tolerant to guava wilt disease.

**Disease management**

Time to time recommendations for the control of guava wilt have been suggested by different workers. These are summarized below.

**Disease management through chemicals**

During 1949, control of wilt was achieved with Chaubatia paste (Anonymous, 1949) but this control measure is not considered valid, as guava wilt is a soil borne disease. Jain (1956) found chemotherapeutic action of 0.1 percent water-soluble 8-Quinolino sulphate against the wilt pathogen (Fusarium oxysporum f. sp. psidii). It's injection in apparently healthy guava plants in a diseased area provided protection against wilt at least for one year and when injected into slightly wilted plants, it was beneficial for their partial recovery. Suhag (1976) reported control of wilt by severe pruning and then drenching with 0.2 per cent either Benlate or Bavistin 4 times in a year and spraying twice with Metasystox and Zinc sulphate. But due to soil borne nature of the disease, pruning does not seem to control wilt. Misra and Pandey (1999b) reported that though different fungicides viz. Bavistin, Topsin M, Indofil M-45, Thiram, Biltex check the various wilt pathogens in laboratory effectively, these pathogens increase it's aggressiveness with profuse spore mass production in the soil, once the effect of these fungicides diminishes. Bhargava et al. (2003) also found control with thiophanate methyle in lab. In Taiwan, Carbendazim, Captapol and Thiabendazole proved effective against wilt pathogen under laboratory experiments but failed in vivo (Leu et al., 1979). In South Africa tebuconazole, propiconazole, prochoraz, triforine and carbendazim + flusilazole were effective under in vitro evaluation (Joubert and Frean, 1993). Antibiotic action (Dwivedi 1990) and heavy metals Hg, Cd and Cu (Dwivedi 1991b) were found effective for control of wilt. Nematodes are reported to aggravate the wilt incidence in guava. Disinfection of soil with DBCP at 52.8ml/10m² or Metham sodium at 252.5ml/10m² was achieved to control nematodes (Rodriguez and Landa, 1977).

Besides fungicides some soil amendment chemicals/cakes/fertilizers were also evaluated for control of wilt. Mathur et al. (1964) found wilt control by soil treatment with 1.82 kg. lime or gypsum/tree, although the control mechanism was
not well understood. At CISH, Lucknow wilt was controlled by application of 6 kg. neem cake + 2 kg. gypsum per plant (Misra and Pandey, 1994-95). Oil cakes like neem cake, mahua cake, kusum cake supplemented with urea @ 10 kg and 1 kg respectively also check the disease (Das Gupta and Ghoshal, 1977). Suhag and Khera (1986) advocated that spread of wilt could be checked by judicious amendments of N and Zn.

**Disease management through cultural practices**

Mathur (1956) advocated that wilt could be controlled by proper sanitation in the orchard. Wilted trees should be uprooted, burnt and trench should be dug around the tree trunk. Edward (1960a) suggested that while transplanting, roots of plants should not be severely damaged. Maintenance of proper tree vigour by timely and adequately manuring, inter-culture and irrigation enable them to withstand infection. The pits may be treated with formalin and kept covered for about 3 days and then transplanting should be done after two weeks. Symptoms of the disease do not appear under green manuring and the disease development is less when organic sources of nitrogen are used (Chattopadhyay and Bhattacharjya, 1968b). Soil solarization with 30µm transparent polyethylene sheet during May-June (Dwivedi, 1993b) have been suggested for the control of wilt pathogens. Prasad et al. (2003), Khan and Misra (2003) and Misra et al. (2004b) reported intercropping with turmeric or marigold to check the wilting of guava. These cultural practices are useful and should be adopted to escape wilt.

**Disease management through varietal resistance**

None of the guava varieties in India is reported free from wilt incidence and hence these cannot be recommended directly for cultivation in wilt infested areas. No information is available in literature regarding breeding varieties for wilt resistance except the information on relative resistance to the natural incidence, which is provided under heading varietal reaction. To combat the disease, option of resistant rootstock seems to be of great use and some work was done on this aspect and is summarized here. Since interspecies and intergeneric graft compatibility is possible, Edward (1961) suggested guava species *Psidium cattleianum* var. *lucidium* and *Syzygium cumini* (Jamun), which seldom get attacked with wilt, may be an effective way for the control of wilt disease. Edward and Gaurishanker (1964) in their further studies found *Psidium cattleianum* (*Psidium molle*), *P. quianense*, Chinese guava (*P. friedrichstalianum*) and Philippine guava compatible and suggested them for the use of rootstock. A local variety Pei-pa In Taiwan was reported resistant and *Psidium friedrichstalianum* has been recommended as possible rootstock (Leu and Kao, 1979). Misra et al. (2003c) identified F1 population of *Psidium molle* X *Psidium guajava* free from wilt, when grown in wilt sick plot and artificially inoculated repeatedly with *Gliocladium roseum*, *Fusarium solani* and *Fusarium oxysporum*. As graft compatibility is very successful, this resistant rootstock is very useful for the control of wilt.

**Disease management through bio-control agents**

Due to soil borne nature of wilt pathogen, it is not practical to completely control with any chemical. The effects of chemicals are also hazardous for the soil and environment, moreover when the effect of chemicals diminishes, the pathogen become more virulent and aggressive (Misra and Pandey 1999b). Hence, considering the above facts, it was considered more desirable to use the bio-agents for the management of the wilt disease.

Dwivedi (1992) advocated *Trichoderma* spp. and *Streptomyces chibaensis* for the control of wilt. Seed oil of *Foeniculum vulgare* were also reported to control wilt (Dwivedi, 1993a). In Pakistan, combined use of Tospin M sprays and the antagonists *Trichoderma harzianum* and *Arachniotus* sp. added in soil amended with wheat straw controlled decline of guava (Ansar et al., 1994). Logani et al. (2002) reported application of wiltnema (seven plant extract - *Allium cepa*, *A. sativum*, *Ocimum sanctum*, *Azadirachata indica*, *Datura stramonium*, *Cannabis sativa* and *Nicotiana tabacum*) for prevention of wilt and better growth of guava plants. Srivastava et al. (2001) found that use of VAM symbiont at the rate of 5kg/tree is beneficial for the control of wilt.

At CISH, Lucknow, three bio-agents were found effective for the control of the wilt disease viz.
Aspergillus niger strain AN 17, Trichderma harzianum, and Penicillium citrinum (Misra et al., 2000, 2004; Prakash et al., 2002). When these fungi were tested for the control of wilt pathogen in laboratory conditions, they were found quite effective (Misra et al., 2004). When relative growth of the three bioagents was studied, it was found that Aspergillus niger was fastest growing and most effective (Misra and Prasad, 2003). These can be grown easily on any substrate like maize/bajra seeds etc. and can also be multiplied on cheap substrates like Sacchrum sp. (grass) and dry and green leaves of Psidium guajava (Shukla et al., 2003). It was also found that at village level these bioagent can be multiplied in earthen pots (Misra and Prasad, 2004). Among these, Aspergillus niger was found very fast growing, easy to propagate and most effective in controlling the wilt disease in field. Besides this quality, it is also growth enhancer and the plants treated with Aspergillus niger developed faster with more height, more thickness and more numbers of leaves (Misra et al., 2000). Dwivedi and Shukla (2002) reported that out of three biogents Trichoderma harzianum, T. viride and Gliocladium virens, T. viride is best for the control of wilt. Singh et al. (2003) reported bioagent Aspergillus niger most effective in controlling the wilt disease followed by Trichoderma viride. Technique of multiplication of bio-agents and application in the field have been standardized (Misra et al., 2003b,d ; Misra and Singh, 2005) and is summarized below.

Integrated eco-friendly approach

Considering the complexity of the problem, integrated eco-friendly approach for the control of guava wilt was suggested by Misra et al. (2003c, 2004b) and Misra (2005) which comprised of bioagent Aspergillus niger strain AN17, resistant root stock (P. molle x P. guajava), intercropping of guava with turmeric or marigold as well proper cultural practices. All these approaches need to be integrated to minimize losses due to the disease.

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