DISCUSSION

Root-knot nematodes are sedentary obligate endoparasites and they induce certain structural physiological and biochemical changes in the host plant. Second stage juveniles after penetration move intercellularly and intracellularly in the root tissue (Christie, 1936) and form gallaries and burrows of broken and separated cells (Roman, 1961). Eventually, they become sedentary with their heads inserted in vascular tissue and body in the cortical region of the root. Xylem vessels become abnormal and highly variable in shape (Pasha et al., 1987). Giant cells, the permanent feeding site induced by the nematode develop around their heads. Giant cells which act as transfer cells provide nutrition to the sedentary females. The cells around the female become hyperplastic dividing repeatedly by mitosis and this contributes cells for the development of galls. These structural and accompanied physiological and biochemical alterations cause malfunctioning of the root system. Absorption of water and minerals and their conduction are impaired. Overall impact of the infection leads to poor growth and reduced yield of the host. Infected plants also show various deficiency symptoms. Galled root becomes short, thick and deformed. Root growth suppression disturbs the root/shoot ratio resulting in appearance of water stress symptoms in plant tops, especially during periods of moisture stress and high temperature (Wilcox and Loria, 1986). In the present study, the chick-pea cultivars inoculated with different inoculum levels (Pi) of Meloidogyne incognita (race 1) showed poor growth and characteristic deficiency symptoms. All the cultivars were susceptible to this race of M. incognita but the extent of growth suppression varied between the cultivars. Similarly, the growth suppressions were related to the inoculum level (Pi) of the nematode. Similar relationship was visible for the deficiency symptoms. The lowest inoculum level used (10 J2) generally did not cause measurable effect on the growth parameters of all the cultivars. Most significant suppressive effect was obtained by highest inoculum level (10,000 J2). The inoculum level of 1000 J2 also caused suppression of most of the parameters in the cultivars. Root galling (number of galls per root system), which is related to
plant damage, and egg mass production (number of eggs per root system) which is related to the population increase, were also inoculum density (Pi) dependent. Root galling and egg mass production were invariably greatest on all the cultivars when the Pi was 10,000 J2. This experiment demonstrated that these selected cultivars now grown on commercial scale by the farmers in India, are susceptible to *M. incognita* (race 1) one of the common root-knot nematode (Khan and Khan 1990, 1991) in India. The growth suppression which would reflect in yield and egg mass production which may have significant impact in terms of damage on the ensuing crops were dependent on the initial inoculum density (Pi) of the nematode.

*Meloidogyne incognita* (race 1) invariably inhibited root nodulation on all the chick-pea cultivars and this inhibition increased with the increase in the initial inoculum level. Mutual suppression of root galling caused by root-knot nematode and root nodulation by root nodule bacteria have been reported in literature (Huang and Barker, 1983; Verdejo et al., 1988; Taha, 1993). In this study, however, effect of root nodule bacteria on root galling was not ascertained. But inhibitory effect of the nematode on root nodulation was apparent, irrespective of the cultivar, and inhibition was inoculum density dependent. Various explanations including competition for space and nutrition between the two organisms have been offered for this kind of mutual inhibitory effects (Masefield, 1958). According to Epps and Chambers (1962) competition between nematode juveniles and root nodule bacteria may be responsible for this inhibition. In the present case, it is quite apprehensible that the suppression of root nodulation on cultivars may have been caused by nutritional interference, particularly carbohydrates or physiological changes brought about by the nematode infection and/or competition for infection site (Taha, 1993). Functional nodules were found to be decreased significantly as a result of *M. incognita* infection. This may be due to invasion of nodules by the nematode which causes histological changes in nodular tissue of infected plants (Taha and Raski, 1969; Robinson, 1961; Barker and Hussey, 1976). Root nodule bacteria, *Rhizobium* spp. are symbiotic nitrogen fixing microorganisms, developing association with roots of leguminous
crops. *Rhizobium* ensures better plant growth and increase leaf chlorophyll content. Reduction of functional nodules is liable to affect nitrogen fixing efficiency of the plants, resulting in reduced benefit derived by the plants.

Pathogenicity of *Fusarium oxysporum* f.sp. *ciceri* has been well established by earlier workers (Westerlund *et al.*, 1974; Mani and Sethi, 1985; Bhatti *et al.*, 1987 and Bhatti and Kraft, 1992). Response of the cultivars included in the study, however, is not determined. The cultivars were variable in their response to *Fusarium oxysporum* f.sp. *ciceri* as all were not equally susceptible. Pusa-212 was found to be resistant. *F. oxysporum* f.sp. *ciceri* caused wilting and yellowing of the foliage. Wilt symptoms appeared first in older leaves which moved upward in later stages. Plants of all the cultivars except Pusa-212 showed suppressed growth and roots exhibited necrotic patches and vascular discolouration extending to the top of the shoot. Similar symptoms were observed by Westerlund *et al.* (1974) and MacHardy and Beckman (1981) on *Fusarium* wilt infected plants. The disease intensity was related to the inoculum level of the fungus, as it increased with an increase in the inoculum level. The inoculum level, therefore, is a significant determinant for the crop damage caused by the fungus. After penetration, wilt inducing *Fusarium* spp. enter the xylem and spread in whole vascular system (Orion and Hoestra, 1974), and in later stage grow into the adjacent xylem parenchyma cells and the cortical tissues. Mechanical plugging, toxins, hydrolytic enzymes and growth regulators are claimed as causes of wilting in infected plants. However, according to Beckman (1984) wilting develops successfully if these fungi penetrate the epidermal cell and successfully invade the cortical tissue and reach vascular system and colonize the xylem vessels.

Among the cultivars only Pusa-212 showed resistance against the fungus. The resistance against the wilt fungus may be due to vascular occlusion which involves formation of gel plugs, tyloses, deposition of additional wall layer and the infusion of these structures with phenols and other metabolites (Beckman, 1984). Gel formation can result in vessel blockage and tylosis completely seals off the infected vessel (Mai and Abawi, 1987).
Root-nodulation was suppressed by the infection of *Fusarium oxysporum* f.sp. *ciceri*. The number of functional nodules declined and of non-functional increased as observed in case of the plants infected with root-knot nematodes. Although this effect of *Fusarium* infection on root nodules has been observed in number of studies (Twng-Wah and Howard, 1969; Sawada, 1982, 1983) but the mechanism involved is not properly determined. It looks plausible that *Fusarium* infected roots due to physiological and structural modifications are rendered unsuitable for the development of root nodules. This suppression may also be due to competition between the two microorganisms at initial stage of the infection. Though, not considered in this study, wilt causing fusaria are known to cause less infection on nodulated roots than non-nodulated roots (Zombolim and Schenk, 1984).

Sulphur dioxide as an air pollutant causes severe damage to crop plants (Varshney and Garg, 1979, Bender *et al.*, 1986; Flagler and Younger 1986, Kumar and Yadav, 1988 and Khan and Khan 1993). Chick-pea cultivars used in the present study suffered growth losses, though differently, and exhibited chlorosis, intercostal necrosis and browning of the leaflet margins. Alterations in physiological and biochemical processes of the exposed plants are implicated as causes for growth suppressions of the plants and development of the symptoms on foliage. Sulphite ions which are formed by reaction of SO$_2$ with water in host cells are phytotoxic and cause injury to leaf tissue (Thomas *et al.*, 1944). Sulphite ions also induce bleaching and phaeo-phytinization or photo-oxidation of leaf pigments (Varshney and Garg, 1979) leading to chlorosis and necrosis of leaf tissue. Synthesis of leaf pigments and photosynthesis are impaired or destruction of the pigments may occur. Singh (1989) observed reduction in leaf pigment content of chick-pea as a result of SO$_2$ exposure. The chlorosis, necrosis and browning of leaflets of the chick-pea cultivars observed in the present study may have resulted through these adverse effects of SO$_2$ in the plant leaves. In addition to destruction of chloroplast and photo-oxidation of chlorophyll molecules (Nieboer *et al.*, 1976) resulting in reduced net photosynthesis, SO$_2$ adversely
affects stomatal conductance (Sheng and Boris, 1988). Toxicity of SO$_3$ is related primarily to its reducing properties rather than its acidity because it is 30 folds more toxic than SO$_4$ (Thomas et al., 1943). Excess of sulphate may interfere with ion absorption, leading to disruption in nutrient balance. Such altered physiological, biochemical and structural conditions may have caused poor growth of the chick-pea plants exposed to SO$_2$. Sulphur dioxide exposed plants of chick-pea of all the cultivars suffered growth losses. In general, concentration of SO$_2$ was determinant for the extent of suppression in a given parameter. Variations in the extent of suppressions of each considered parameters were observed which indicates that the growth response of the chick-pea cultivars would be different under air pollution stress especially caused by SO$_2$. These variations in the response of the cultivars would be helpful for selection of a cultivar for agricultural purpose under a given air pollution condition. The intensity of the symptoms caused by SO$_2$ was dependent on its concentration. The intensity of the symptoms was greater at 0.2 ppm than 0.1 ppm on all the cultivars. So was the growth of the plants. Therefore, it becomes obvious that the harmful impact of SO$_2$ on crop plants like chick-pea is concentration dependent and expected damage can be correlated by determining the concentration in a polluted area. Leguminous crops like soybean (Davis, 1972; Miller et al., 1974; Sprugel et al., 1980; Kress et al., 1986; Sheng and Boris, 1988 and Singh, 1993), Windusa bean, *Phaseolus vulgaris* (Goodzik and Linkens, 1974), lentil, *Lens culinaris* and chick-pea (Singh, 1989) has been demonstrated to suffer growth and yield losses by earlier workers. It seems therefore, desirable to determine response of the cultivars of different crops including legumes against various known concentrations of common air pollutants for the benefit of farmers.

Chick-pea strain of *Rhizobium* is slow growing, non-acid producing and very sensitive even to a slight acidity (Vincent, 1977). Chick-pea plants exposed to sulphur dioxide artificially had smaller number of root nodules and this inhibition was greater in higher concentration of SO$_2$. This may be due to aciditification of soil by SO$_2$. Sulphur dioxide is converted to H$_2$SO$_4$ when it
comes in contact with soil-water which lowers the soil pH, hence providing unfavourable conditions for root nodule bacteria. Inhibition of root nodulation by SO$_2$ has been reported by Shriner (1974) on kidney bean and soybean, Singh (1989) on lentil and chick-pea, Kumar and Prakash (1990) on pea and Singh (1993) on soybean.

Interaction between root-knot nematodes (*Meloidogyne* spp.) and wilt-inducing fungi (*Fusarium* spp.) is well established on a number of crops. Their interaction is generally synergistic in causation of damage to the crop plants. Wilting of the plants is enhanced in the presence of the nematodes. Interaction of root-knot nematodes with wilt-causing fusaria has been studied on a number of leguminous crops also viz., alfalfa (McGuire *et al.*, 1958; Griffin, 1986), cowpea (Thomason *et al.*, 1959), pea (Davis and Jenkins, 1963), beans (Riberio and Ferraz, 1984; Singh *et al.*, 1981) and chick-pea (Goel and Gupta, 1984; Mani and Sethi, 1987; Kumar *et al.*, 1988). In the present study, *M. incognita* and *F. oxysporum* f.sp. *ciceri* interacted synergistically in suppressing the plant growth parameters of the cultivars of chick-pea. Severity of wilt symptoms particularly in Pusa-244 was enhanced when both the pathogens were present together. Its degree was influenced with the sequence of inoculation. The intensity on Pusa-244 was greater in sequential inoculation than concomitant inoculation. The date of appearance of wilting was also advanced. Pusa-212 which was resistant to *F. oxysporum* f.sp. *ciceri* became susceptible in the presence of the nematode.

Root-knot nematodes alter the root exudates of the infected plants which are generally accumulated near the root tip and attract the wilt fungus. Root exudates of nematode-infected plants contain higher concentrations of Ca, Mg, Na, K, Fe and Cu and during first fourteen day of infection, carbohydrates are the major organic constituents of root exudates but nitrogenous compounds predominate afterwards (Van Gundy *et al.*, 1977). The interaction of root-knot nematode and *Fusarium* wilt fungus are complex and involve modification of host physiology (Powell, 1971). The basis for interaction between root-knot nematodes and wilt causing fungi is generally suggested to be nutritional. Accord-
According to Powell (1971) and Webster (1985) root-knot nematode infection predisposes the host plant to wilt fungus. The giant cells which are produced after 3-4 weeks of nematode inoculation are very active metabolically and contain maximum concentration of DNA and photosynthates. The greatest host-predisposing capability of *Meloidogyne* spp. has been observed at this stage which results in synergistic interaction with *Fusarium* wilt fungus (Porter and Powell, 1967; Webster, 1985). Wilting severity and suppression in plant growth was greatest in chick-pea plants which received the nematode 3-weeks prior to the fungus (sequential inoculation). During this time period, *M. incognita* may have induced development of giant cells which became metabolically active and nutritionally rich when the fungus was added. Wilt fungi produce toxins which induce appearance of wilting symptoms (Bell ad Mace, 1981). Giant cells being rich in nutrients, serve a good site for the colonization of the fungus which ultimately leads to formation of more toxins and hence wilt severity is increased. Owens and Specht (1966) believed that during development of giant cells, infected root of host plant exhibit a decrease in cellulose and lignin while amino acids, hemicellulose, lipids, minerals, nucleotides, organic acids, proteins, DNA and RNA are increased considerably. These biochemical changes enrich the medium which is the cause for rapid growth and colonization of wilt fungus. However, the exact mechanism of interaction between root-knot nematodes and *Fusarium* wilt fungi are not well understood (Mai and Abawi, 1987). Wounding of roots by root-knot nematodes, once thought to be responsible for synergistic interaction between root-infecting fungi and nematodes, is no more considered of paramount importance in their interaction (Powell, 1977). Westerlund *et al.* (1974) while studying pathogenicity of *F. oxysporum* f.sp. *ciceri*, found that this species may require wounding for efficient infection. It is likely that wounding caused by *M. incognita* on chick-pea cultivars might have contributed to some extent for synergistic interaction between the two pathogens.

Root nodulation was suppressed by interaction of *M. incognita* and *F. oxysporum* f.sp. *ciceri* on both the chick-pea cultivars. This suppression was greater when nematode inoculation preceded in sequential inoculation. As ex-
plained earlier, this may be due to competition for space between the organisms or nutrient deficient status of the host plant because of dual infection by *M. incognita* and *F. oxysporum* f.sp. *ciceri* (Masefield, 1958; Epps and Chambers, 1962). Similar observations were made by Kumar *et al.* (1985), Mani and Sethi (1987) and Khan and Salam (1990). Greater reduction in functional nodules may also be due to invasion of the nodules by the nematode causing histological changes in nodular tissue or greater stress on the nutrient status of the host due to dual infection.

Breaking of resistance of Pusa-212 against *F. oxysporum* f.sp. *ciceri* in the presence of *M. incognita* is significant agriculturally. As mentioned earlier, a number of defence mechanisms are suggested to be operative in resistant cultivars (Beckman, 1984; Mai and Abawi, 1987). *M. incognita* seems to have disturbed the defence mechanism(s) in Pusa-212, hence wilt symptoms appeared. According to Francl and Wheeler (1993), breaking of resistance by nematodes implies that the nematode affects the physiology of the plant in some way, rendering it incapable of expressing the resistant reaction. In other words, the gene action is rendered ineffective due to disruption at the cellular or tissue level but the gene is not made inoperative. Breaking of resistance by root-knot nematodes has been reported in several crops viz., tomato (Harrison and Young, 1940; Pitcher, 1974; Carter, 1978), pea (Davis and Jenkins, 1963), chrysanthemum (Johnson and Littrell, 1969) and watermelon (Donald and Johnson, 1973). The favourable changes in nutrient status of the host plant by the nematode, may have predisposed it to the fungus.

Interaction of sulphur dioxide and *Meloidogyne incognita* (race 1) was not synergistic in causing suppressive effect on plant growth except the root length. The interactive effect was generally additive as greater suppression in plant growth of the chick-pea cultivars was observed under this treatment in comparison to the suppressions caused by either of the pathogens individually. Root-knot disease was suppressed in plants exposed to sulphur dioxide. Gall formation and egg mass production declined in the plants of both the cultivars.
Sulphur dioxide may have affected root-knot nematodes directly through soil acidification or indirectly through host modification (Jackson, 1967; Khan and Khan, 1993). *M. incognita* being a sedentary obligate parasite, depends on the host plant for its energy and nutrition demand which become increased during oviposition (Melakeberhan and Webster, 1993). Suppression of root galling and egg mass production may have arised from improper development of the nematode because of inadequate nutrient supply and less infection sites due to poor shoot and root growth of SO2-exposed plants of chick-pea. Suppression of gall formation and egg mass production in the simultaneous inoculation exposure of the nematode and SO2 gives an evidence of direct effect of pollutant on the nematode juveniles, possibly making them incapable of root penetration. Pre-inoculation exposure of plants to SO2 has been found to suppress root-knot disease (Singh, 1989; Pasha, 1990) which was claimed to be due to alteration in physiology of SO2-exposed plants as suggested by Shimazaki *et al.* (1980). In the present study, SO2 *in vivo* suppressed juvenile hatching of *M. incognita* when the egg masses were exposed to SO2 in artificial conditions. It may be inferred that SO2 after entering the soil may have direct suppressive effect on the nematode during its pre-penetration stage. Reduced juvenile penetration and altered host physiology would reflect in root galling and egg mass production. Some studies, however, showed that non-injurious concentration of air pollutants including SO2 either enhance root-knot disease (Khan and Khan, 1991; Khan and Khan, 1993) or fail to affect adversely nematode population (Weber *et al.*, 1979). In general, lower concentration of SO2, however, does not affect reproduction of plant parasite nematodes (Weber *et al.*, 1979; Brewer, 1979). Lower concentration of SO2 also does not cause appreciable alteration in the host physiology. Higher concentration of SO2, on the other hand, alters host physiology appreciably and suppresses root-knot disease significantly (Weber, *et al.*, 1979; Shimazaki *et al.*, 1980; Khan and Khan, 1993). In the present study, the poor nutritional status of the plants of chick-pea cultivars exposed to SO2 as evidenced from their reduced root and shoot growth may have caused stress on availability and adequate supply of nutrients to the developing females leading to suppression of the disease. In
addition to other adverse effects of SO$_2$ on the nematode directly or indirectly, it also looks plausible that poor root growth of the host may have not be able to provide adequate accommodation to the females for their proper development and egg laying. Therefore, both physiological and physical causes were impediment for proper development of the nematode. Nematode-inoculated chick-pea cultivars exposed to SO$_2$ were not synergistically suppressed. Inhibitory effects of SO$_2$ on the development of galls and reproduction of the nematode possibly caused favourable effect on plant growth of the chick-pea cultivars.

Sulphur dioxide caused suppression of root nodulation. Numbers of functional nodules declined and those of non-functional nodules showed an increase. This suppressive effect of SO$_2$ may have caused adverse influence on nitrogen fixing efficiency of the roots. In the nematode-inoculated plants further adverse effects on root nodulation resulted as observed for plants exposed to SO$_2$ alone. Greater adverse effects of SO$_2$ on nematode inoculated plants was apparently caused by joint effects of SO$_2$ and *M. incognita*. SO$_2$ has been found to be responsible for inhibition of root nodulation (Shriner, 1974; Singh, 1989; Kumar and Prakash, 1990). Decline in the numbers of total and functional nodules would be reflected in the nitrogen fixing capability. This is an important and significant impact of SO$_2$ which becomes more pronounced on root-knot nematode infected plants. Similar effect is likely to occur on other legumes. This aspect needs further investigation in order to realize its full implications in agriculture.

In general, fungus-inoculated chick-pea cultivars exposed to SO$_2$ had suppressed growth in comparison to the plants exposed to either of the pathogens individually. Pusa-212 which showed resistance against *F. oxysporum* f.sp. *ciceri* developed wilt symptoms on exposures to SO$_2$. The fungus and SO$_2$ acted additively and resistance of Pusa-212 against the fungus was broken. SO$_2$ exposures rendered the cultivar susceptible to the wilt fungus. The poor nutritional status of the plants of Pusa-212 as a result of SO$_2$ stress, possibly rendered the genes responsible for resistance ineffective. Exposure of *F. oxysporum* f.sp.
ciceri to SO$_2$ in vivo was found to be inhibitory for its growth. SO$_2$, however, at 0.5 ppm did not suppress it. Soil is a major sink for the removal of many gaseous pollutants including SO$_2$ (Bremner and Banwart, 1976). *Fusarium oxysporum* act as a minor sink for the removal of atmospheric SO$_2$ (Craker and Manning, 1974). It is likely that SO$_2$ in the soil being low in concentration, favourably influenced *F. oxysporum* f.sp. *ciceri*. The fungus, in turn, caused greater wilting. This effect of SO$_2$ is great agricultural significance. Similar effects of SO$_2$ may occur on resistant cultivars of other crops, which would create difficulty in their cultivation in a given air pollution condition. Screening of crop cultivars for their response to SO$_2$ and other air pollutants and performance of resistant cultivars to fungal pathogens particularly, soil-borne fungi, under air pollution stress seems highly desirable.

Impact of SO$_2$ on soil-borne fungal pathogens has not gained adequate study. SO$_2$ decreases the severity and incidence of fungal disease in plants and obligate fungal parasites are more sensitive than other fungal pathogens. Rust fungi (Johanson, 1954; Weinstein, 1975; Giacomo *et al.*, 1990), powdery mildews (Kock, 1935; Pasha, 1990; Khan *et al.*, 1991), rose black spot (Saunders, 1966) and maize leaf spot (Laurence *et al.*, 1979) are inhibited by SO$_2$. Sulphur dioxide predisposes plants to fungus infection by decreasing their vigour and growth. When fungus establishes itself within the host tissue it is not likely to affected by SO$_2$ (Heagle, 1973). Once within the roots, the wilt-inducing fusaria may grow fast spreading into the vascular elements, hence blocking the vascular system and producing phytotoxins which ultimately cause wilting and growth suppression (Mai and Abawi, 1987).

Fungus inoculated chick-pea cultivars under SO$_2$ stress exhibited suppression in root nodule formation which may be due to acidification of soil. Chick-pea strain of *Rhizobium* is sensitive to even slight acidity. Reduction in root-nodulation may have arised also due to competition of wilt-fungus with root nodule bacteria for nutrient supply or bacterium might have been affected by phytotoxin produced by the fungus. Any stress on the host or *Rhizobium* affects
nodulation (Huang, 1987). Wilt symptoms appeared earlier and more severe when fungus inoculated plants were exposed to sulphur dioxide. Biochemical changes in host plant as a result of their exposure to SO₂ may have made conditions favourable for the colonization and pathogenic effect of the wilt fungus.

In the present study, maximum suppression in plant growth of chick-pea cultivars was observed when the fungus + nematode inoculated plants were exposed to SO₂. Chick-pea plants exhibited more suppression as compared to plants exposed to each of the pathogens individually or in combination i.e., nematode + fungus, nematode + SO₂ and fungus + SO₂. This suppression may be attributed to biochemical, physiological and structural changes induced by the pathogens as described earlier. Suppression in nematode reproduction was maximum in the presence of the three pathogens. This may be due to direct or indirect effect of SO₂ on the nematode by soil acidification or inadequate supply of nutrient to nematode due to adverse impact of SO₂ on plant metabolism or competition for nutrient between the fungus and the nematode. Wilting appeared earlier and was more severe in plants exposed to all the three pathogens. Alteration caused by SO₂ may have caused better root penetration by the nematode juveniles and giant cells induced by the nematode provided adequate nutrients for the multiplication and spread of the fungus in the vascular system. The fungus by growing rapidly in adjacent xylem parenchyma cells and the cortical tissue and by rapid mechanical plugging and production of wilt toxins, may have caused greater and earlier wilting of plants. Root nodulation which involves many components in a delicately balanced state was adversely affected under this treatment, because any stress on legume host or rhizobia disrupts the nodulation process (Huang, 1987).

The findings of the present investigation demonstrate that, all the six commercial cultivars of chick-pea used in the study, which are being grown in India, are more or less susceptible/sensitive to root-knot nematode, *Meloidogyne incognita* (race 1), wilt fungus, *Fusarium oxysporum* f.sp. *ciceri* and sulphur
dioxide. Pusa-212, however, is an exception. It is resistant to *F. oxysporum* f.sp. *ciceri*, relatively less sensitive to SO2 but susceptible to *M. incognita* (race 1). Cultivation of these cultivars in the fields infested with the root-knot nematode or the wilt fungus or in areas polluted with SO2 is not desirable, and efforts, therefore, should be made to develop resistant/tolerant cultivars of chick-pea in order to minimize the damages caused by these biotic pathogens and SO2. The results further suggest that root-knot nematode, *M. incognita* (race 1) and wilt fungus, *F. oxysporum* f.sp. *ciceri* interact synergistically causing greater wilting of the cultivars susceptible to the fungus. Resistance of chick-pea cultivars against the wilt fungus is likely to be broken due to their interaction, as found in case of Pusa-212. SO2 stress on the crop plants, like chick-pea may also enhance their susceptibility to the wilt fungus and cultivars showing resistance against the wilt fungus may be predisposed by SO2. *M. incognita* infected plants are likely to suffer greater damage caused by SO2, accompanied with pronounced SO2-induced symptoms. SO2 stress can also influence the interactive effect of root infecting pathogens like *M. incognita* and *F. oxysporum* f.sp. *ciceri* causing greater plant damage and wilting. Intensity of wilting may be increased and appearance timing advanced. These findings are of great agricultural importance and need to be fully investigated in order to realize total implications of air pollution on crop plants. The problem, needs serious consideration by the management pathologist/nematologist and plant breeders. Root nodulation which is a beneficial system for the crops is adversely affected by the all the components of the pathosystem, the nematode, the fungus and SO2 and through their interactions. Suppressive effect of SO2 on root nodulation and enhanced suppression in the presence of the nematode and fungus and another significant findings which need attention.