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REVIEW ARTICLE

# Discussion on the consequences of chickpea wilt and management through induced resistance

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## Abstract

Chickpea (*Cicer arietinum* L.) is a crucial source of dietary protein and accounts for 18% of global legume production. However, the crop faces a variety of biotic and abiotic constraints, with fusarium wilt being the most common soil-borne disease. This disease poses a significant threat to chickpeas, leading to yield losses of up to 80% worldwide. Fusarium wilt pathogens exhibit host specificity and characteristic symptoms in mature plants include brown to black discoloration of the xylem vessels, wilting, and leaf burning caused by phytotoxins produced by the pathogen. To combat this fungal disease, several cultural, biological, and chemical methods have been extensively employed. While chemical control methods have proven to be highly effective and widely adopted by growers, they come with several adverse consequences for humans, the environment, soil, and water. Moreover, improper and excessive use of fungicides can lead to the development of resistance in plant pathogens. Thus, there is a pressing need for an environmentally friendly approach that promotes plant resistance. One such approach is induced resistance, which involves enabling plants to build their own resistance mechanisms. Induced resistance can take different forms, such as systemic acquired resistance based on the salicylic acid pathway, and induced systemic resistance based on the jasmonic acid pathway.

## Keywords

Fusarium wilt; chickpea; systemic induced resistance; systematic acquired resistance; bio-control

## Introduction

Chickpea (*Cicer arietinum* L.) is a self-pollinated, annual diploid plant that is also known as Bengal gram or garbanzo bean. In Asia, Africa, Central America, and South America, chickpea is a significant grain legume crop (1). Leguminaceae is the family of legumes that includes chickpeas. Chickpeas are cool-season legumes that can be found in tropical, subtropical, and temperate climates (2). Chickpea is known as the "poor man's meat" because it offers a high-protein and low-cost alternative to animal protein. After dry beans (*Phaseolus vulgaris* L.) and dry peas (*Pisum sativum* L.), chickpea is the world's third most significant pulse crop. Chickpea is a legume that originated in the Middle East and is now grown in 45 nations. India is the world's largest producer of chickpeas. India produces 67.32% of chickpeas, Pakistan 6.19%, and Australia 5.72% (3). Chickpea production reached 73.3 million tonnes in 2011-2013. In India, 9.2 million tonnes were

produced, with an average yield of 920 kg/ha. Madhya Pradesh ranked first in production (40.60%). Maharashtra ranked second in terms of area (16.57%) and third in terms of production (13.07%). Rajasthan ranked second in production (14.09%). Andhra Pradesh recorded the highest yield (1522 kg/ha) (4).

Unsaturated lipids containing acids like linoleic acid and oleic acid, as well as protein (18–22%), carbohydrates (6–62%), fat (4.5%), calcium (280 mg/100 g), iron (112.3 mg/100 g), and phosphorus (301 mg/100 g) are present in chickpea. Because of its nutritional worth, the market is crowded (5). Among pulses, chickpea proteins have a higher glutelin content (6). The most common fungal diseases that affect chickpeas are fusarium wilt (*Fusarium oxysporum* f. sp. *ciceri*), aschochyta blight (*Aschochyta rabiei*), dry root rot (*Rhizoctonia bataticola*), and wet root rot (*R. rolfsii*), as well as viral diseases including the beet western yellow virus, bean leaf roll virus, soybean dwarf virus, pea seed-borne virus, and the chlorotic stunt virus (Table 1) (7).

**Table 1.** Yield loss in chickpea crop

| Disease | Yield loss | Year | References |
|---|---|---|---|
| Fusarium wilt | 80-100% | 2016 | (11) |
| Aschochyta blight | 25-50% | 2020 | (3) |
| Botrytis grey mould | 50-60% | 2006 | (12) |
| Dwarf chlorotic virus | nearly 100 % | 2009 | (17) |
| Dwarf chlorotic virus | 75-90% | 2009 | (17) |
| Luteovirus | 50-60% | 2008 | (13) |
| Faba bean necrotic yellow virus | 40-50% | 2008 | (13) |

*Fusarium* spp. was identified as the cause of chickpea wilt by Prasad and Padwick. Padwick later gave the fungus its name. Synder and Hansen renamed *F. orthoceras* var. *ciceri* as *F. oxysporum* f. sp. *ciceri* which is now widely accepted (8). Wilt is one of the most common diseases that affect chickpea plants. It is a seed and soil-borne disease (3). Wilt lowers chickpea output by reducing seed yield and weight. In India, annual output losses from the disease were estimated at 10-15%, but severe outbreaks account for 70% of overall crop yield losses (9). *F. oxysporum* is a widespread soil fungus found all over the world in cultivated soil. Some occur only as saprophytes in the rhizosphere of plants. Based on their host plant species and plant cultivars, there are more than 53 forms, and 29 varieties, respectively (10).

The use of resistant cultivars in the disease management strategy of fusarium wilt is both practicable and effective globally (11). It is difficult to understand why it has taken so long for the scientific community and agrichemical industry to recognize the hazards to human health and the environment of the increased dependence on pesticides, acknowledge the potential of induced systemic resistance in plants, and appreciate its significance to fundamental science and as a technology for plant disease control (12). The wilt is treated with four fungal bioagents (*Trichoderma harzianum, T. viride, T. hamatum* and *Gliocladium virens*) and two bacterial bioagents (*Pseudomonas fluorescens* and *Bacillus subtilis*) (Table 2). The ability of these fungicides, including carbendazim 50wp, dividend star, aliette 80wp, copper oxychloride, defeater 20wp, ridomil gold, and thiowet jet 80wp, to prevent the growth of *F. oxysporum* colonies was investigated using the poisoned food approach.

## Geographical Distribution of Chickpeas

Chickpeas are the third-largest pulse produced globally, with a yearly production of 11.67 million tonnes. This ranking places chickpea behind beans (25.66 million tonnes) and peas (11.69 million tonnes) with a mean annual production of 11.67 million tonnes from 2004 to 2017 (Table 3). These three types of pulses beans, peas, and chickpeas account for more than 70% of all pulse production worldwide, with chickpeas contributing over 17%. Chickpea is third among the most popular pulses consumed (13).

**Table 3.** Mean annual global production of pulse crops 2004–2017 (54)

| Pulse | Production (tons) |
|---|---|
| Beans | 25,657,833 |
| Peas | 11,691,517.3 |
| Chickpeas | 11,672,579 |
| Cowpeas | 6,498,236.8 |
| Faba beans | 4,468,240.1 |
| Lentils | 4,990,522.6 |
| Pigeon peas | 4,449,435.9 |
| Other pulses | 6,254,656.9 |
| Total pulses | 75,683,021.6 |

**Table 2.** Control of chickpea wilt by biological control and within induced resistance

| Antagonists | Nature of disease control | Year | Reference |
|---|---|---|---|
| Salicylic acid + *Pseudomonas fluorescens* | Bacterium induced resistance and reduced wilt by 26-50%. Salicylic acid reduced wilt by 52-64 %. Reduction in disease with combined application. | 2005 | (40) |
| *Bacillus subtilis* | Seed coating significantly reduced wilt by 30-40.8% | 2014 | (47) |
| *Trichoderma viride* + *T.harzianum* | Antagonists colonized chickpea roots and suppressed wilt. | 2013 | (48) |
| Non-pathogenic *Fusarium oxysporum* | Disease incidence reduced by 25-30% | 2004 | (28) |
| *P.fluorescens* | Seed treatment with culture suspension reduced pre and post emergence losses by 40%. | 2007 | (10) |

From 1961 to 2013, the harvested area used to produce chickpeas varied from 8.9 million ha in 1981 to 13.5 million ha in 2013 (Fig. 1). In terms of harvested area, earlier production trends from 1961 to 2001 were largely stable or slightly dropping. However, starting in the late 1900s, yield gains started to have an effect on overall production. Production began to rise steadily in the early 2000s and has been doing so ever since, especially after 2004. Over 50 nations produce chickpeas, with India being the largest producer and accounting for more than 70% of global production. India's dominance in chickpea production and the relative importance of the next-largest producers, Pakistan and Iran, contribute 10% and 5% of global production, respectively. Ethiopia, which has significantly boosted output in recent years and currently contributes over 2% of global production, is followed by other important producing nations like Turkey and Australia, which account for 4 and 3% of global production, respectively. Malawi, Mexico, Morocco, and Syria are also significant producers. Mean chickpea yields have a wide range in producing nations, varying from relatively low yields of 500–600 kg/ha in Syria, Pakistan, Malawi, Morocco, and Iran to comparatively high yields in Mexico and Ethiopia. The largest producer, India, consistently achieves mean yields of 900 kg/ha. The peak yields in Mexico are primarily due to the majority of the crop being grown during the chilly winter weather (14).

## Biology

Chickpea is an herbaceous annual plant that branches from the base. It is almost a small bush with diffused, spreading branches. The plant is mostly covered with glandular or non-glandular hairs, but some genotypes do not possess hair.

Based on seed size and colour, cultivated chickpeas are of two types (14).

### *Macrosperma (kabuli type)*

The seeds of this type are large (100-seed mass >25 g), round or ram head-shaped, and cream-colored. The plant is medium to tall in height, with large leaflets and white flowers, and does not contain anthocyanin.

### *Microsperma (desi type)*

The seeds of this type are small and angular in shape. The seed colour varies from cream, black, brown, yellow, to green. There are 2-3 ovules per pod, but on an average 1-2 seeds per pod are produced. The plants are short, have small leaflets and purplish flowers, and contain anthocyanin.

## Symptomatology of Fusarium Wilt

Chickpea wilt is caused by *Fusarium* species, according to Prasad and Padwick. Padwick later named the fungus in 1940 (15). Early wilt symptoms include flaccidity of individual leaves, dull green discolouration, desiccation, and plant collapse. These symptoms appear in the flowering stage following a 6-week seeding in Arizona during the months of October and November (9). Late wilt causes the dropping of petioles, rachis, leaflets, and foliage, which are noticeable at the podding stage in the months of March and April (9). Chickpea wilt is a vascular disease that causes browning or blacking of the xylem. All phases of the crop are affected. Two pathotypes have been identified, which cause unique yellowing and wilting syndromes with brown vascular discoloration in susceptible chickpeas. The yellowing syndrome is characterized by slow and progressive foliar yellowing and late plant death. The wilting syndrome is marked by rapid and severe chlorosis, flaccidity, and premature plant death (9). To date, eight races of *Fusarium oxysporum* f. sp. *ciceris* have been reported from India, Spain, and the United States (0, 1A, 1B/C, 2, 3, 4, 5, and 6) (16).

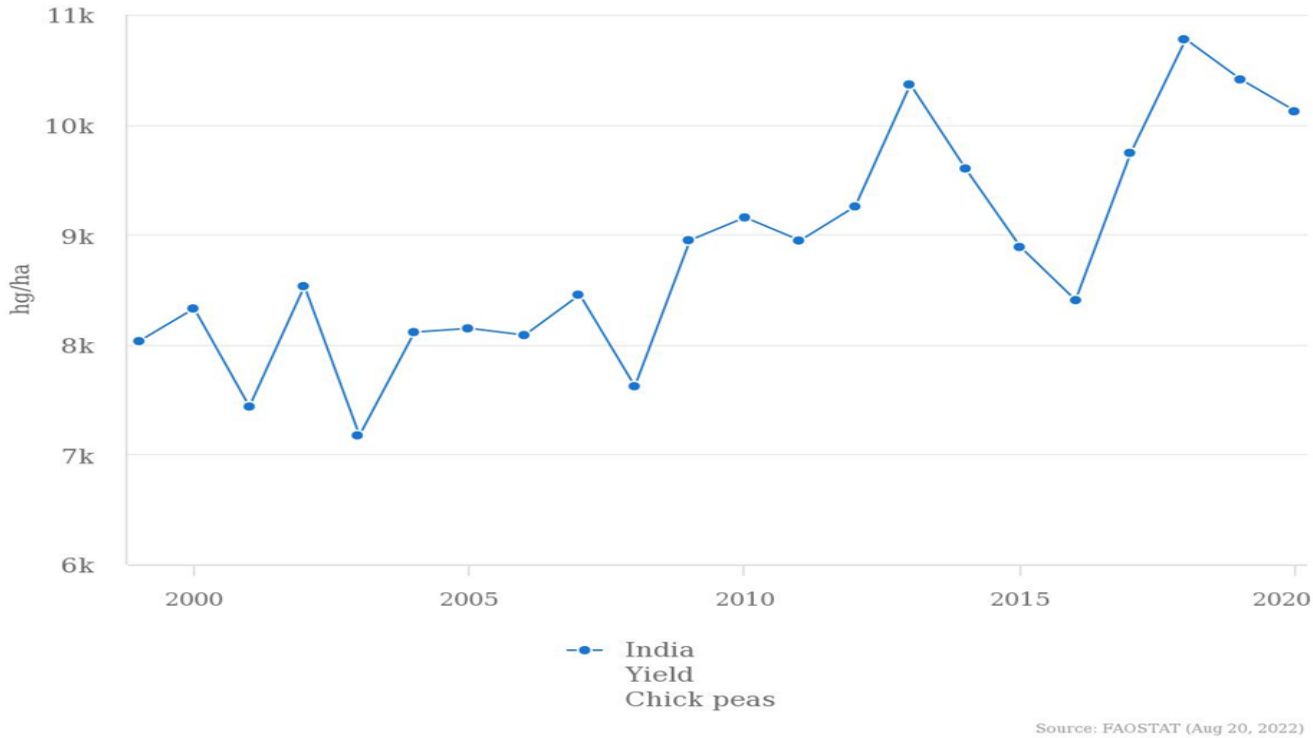


**Fig.1**. Yield of chickpea in India (54, 55)

## Survival and Primary Infection

The pathogen's primary inoculum is responsible for the formation of fusarium wilt in chickpeas, which is a monocyclic disease. Macroconidia, microconidia, and chlamydospores are the three types of asexual spores produced by the common soil inhabitant *Fusarium oxysporum* (17). The macroconidia have three or four septa, a tapering and curved apical cell, and a foot-shaped basal cell. They are generally straight to slightly curved, slender, and thin-walled (2). Infected seeds and plant debris can spread the disease. Chlamydospores are the principal source of wilt infection. The fungus may live for at least 6 years in soil and chickpea trash as a result of the presence of chlamydospores (9).

## Disease Cycle

The fungus can be spread by seed and lives on plant debris in the soil. Free chlamydospores were discovered in soil, seed hilums, cotyledons, and axis. Chlamydospores are the main source of infection. The pathogen can live for up to 6 years without a host. Microconidia and macroconidia exist in chlamydospores. The mycelium takes up residence in the host plant. The fungus remains dormant as chlamydospores in plant debris until stimulated to germinate, once carbohydrates are released from decaying plant tissue or from roots. After the chlamydospores germinate, conidia and new chlamydospores may be formed, as well as hyphae. Following germination, a thallus is produced, from which conidia form in 6–8 h, and chlamydospores in 2–3 days if conditions are favourable. Invasion of the roots is followed by the penetration of the epidermal cells of the host or the non-host (18) and the development of a systemic vascular disease in host plants. Mycelium spores penetrate the root through the cortex, epidermis, and xylem vessels. Penetration occurs when a wound is pierced (19).

Mycelium proliferates rapidly in xylem tissues, causing xylem vessels to become blocked. As a result, the plant wilts and dies. The roots do not appear to be decaying on the outside and appear to be in good health, but when split vertically from the collar region downward, the internal tissues, namely the pith and xylem, reveal a dark discoloration. Similarly, early wilting reduced the seed number/plant and caused more yield losses than late wilting. Disease is more severe in light sandy soil than heavy clay (20). At 20 °C, wilt incidence was higher than at 25°C. At 15°C, plant vascular discoloration and leaf chlorosis did not occur (21). Chauhan reported that the disease intensity increases with decreasing pH, with considerably low intensity at a pH of 9.2. A pH of 5.2 was found to be optimum (22) (Fig. 2).

## Agronomic Practices

Diseases are more prevalent in early-planted crops. Several studies have found that delaying the planting of crops results in improved yield and disease control, primarily due to the cold weather. Plants planted at a spacing of 7.5 cm show less wilt compared to plants spaced at 15-20 cm apart (23). When crops such as wheat, linseed, mustard, and barley are inter-cropped or mixed-cropped with chickpea, wilt is mostly reduced in linseed.

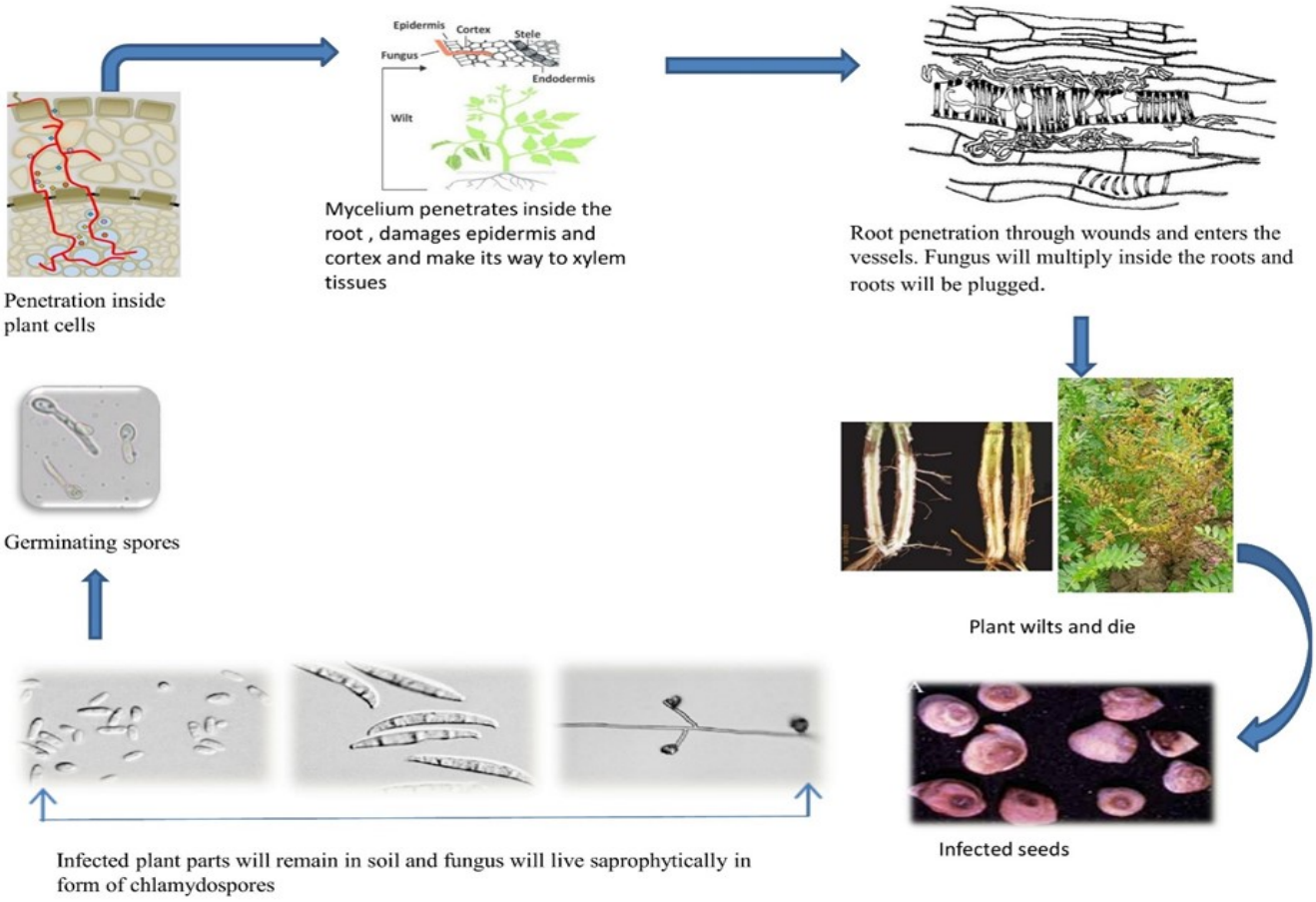


**Fig.2**. Disease cycle of *Fusarium oxysporum* f. sp. *ciceris* (9)

Control using plant extracts, such as plant-derived fungicides, is environmentally friendly and non-toxic. Farmers can easily prepare these extracts. In an *in vitro* investigation, four plant species were identified: *Azadirachta indica* A. Juss*, Datura metel* L. var *Ocimum sanctum* L.*,* and *Parthenium hysterphours* L. The methanolic extracts from these four plants were found to be efficient in controlling the mycelium proliferation of *Fusarium oxysporum ciceri* at a concentration of 40%. The germination of pathogen spores was completely prevented by a leaf extract of *A. indica* at a concentration of 100%. Researchers studied the effect of aqueous garlic leaf extract on *F. oxysporum* f. sp. *ciceri* and found that 7000 and 5000 ppm of the extract reduced wilt and fungal growth (24). To manage pests and diseases, synthetic fungicides should be employed. In many areas where chickpeas are grown, diseases caused by *F. oxysporum* and *Meloidogyne Javanica* (MJ) co-occur. When chickpea plants are infected by both of these pathogens simultaneously, the severity of fusarium wilt is increased (25).

## Chemical Control

Chemical control has been widely used in the past and present to control chickpea wilt disease. The sensitivity of twenty-seven isolates of *Fusarium oxysporum* f. sp. *ciceris* against 10 fungicides (Antracol, Captan, Benlate, TopsinM, Cobox, Dithane M-45, Acrobat, Ridomil, Vitavax and Daconil) (Table 4) was studied based on their sensitivity to fungicides at a concentration of 100 ppm using the poison food technique. After autoclaving, each fungicide was added to the Waksman agar medium. A 20 ml mixture of altered and unamended media was poured into petri plates. Using a sterile cork-borer, a 4 mm agar plug of the fungus was cut from the cultured plate and placed in the center of each petri plate as it solidified. After seven days of incubation at 26±20°C, the infected petri-plates were measured for radial colony growth (mm) of mycelium. Isolates with radial growth of the fungus greater than 35 mm, were classified as nonsensitive "N" while those with radial growth less than 35 mm were classified as sensitive "S" (26).

## Induced Resistance

A physiological "state of heightened defensive capacity", known as "induced resistance", occurs when a plant's intrinsic defenses are strengthened against subsequent biotic and abiotic factors. This improved state of resistance works well against a variety of parasites and pathogens (21). Systemic acquired resistance (SAR) and induced systemic resistance (ISR), which can be distinguished based on the type of elicitor and the regulatory mechanisms involved, are the two forms of induced resistance that are most thoroughly defined (27).

Plant growth-promoting rhizobacteria (PGPR), which colonise the root surface and the tightly adherent soil interface, are extensively researched PGPR in the rhizosphere. Competition for an ecological niche is the commonly acknowledged mechanism of bio-control mediated by PGPR. The development of systemic resistance (ISR) in host plants to a range of diseases is facilitated by PGPR as well as the production of inhibitory allelochemicals (28). SAR can be induced in plants by exposure to virulent microbes that are non-pathogenic or avirulent. The accumulation of proteins involved in pathogenicity (such as chitinase and glucanase) and salicylic acid occurs after a specific period of time, depending on the plant and elicitors. The most well-known approach for increasing ISR is the use of plant growth-promoting rhizobacteria, specifically *Pseudomonas* strains that do not have an obvious effect on the plant roots (29). Unlike SAR, ISR does not involve the accumulation of pathogenesis-related proteins or salicylic acid, but instead relies on pathways controlled by jasmonate and ethylene, which can be differentiated based on the nature of the elicitor and the regulatory pathways involved (Fig. 3) (30).

**Table 4**. List of fungicides used for the determination of variability in chickpea isolates of *Fusarium oxysporum* f. sp. *ciceri* (57)

| Sr. No. | Common Name | Trade Name | Chemical Name | Mode of Action | Formulation | Manufacturer |
|---|---|---|---|---|---|---|
| 1 | Copper-oxychloride | CupravitCobox (1965), cobox, Vitigran Blue (1988), Cuprasan (1992) | Copper Oxychloride | Contact | 50%WP | Agricide (Pvt) Ltd. |
| 2 | Metalaxyl | Ridomil Gold (1996) | Methyl-N- (2- methoxyacetyl)-N- (2,6) xyls | Contact | 60%WP | Novartis (Pvt) Ltd |
| 3 | Benomyl | Benlate, Sunlate, Benlate (1980) | Methyl-1- (butylcabamonyl)- 2- benzinidazol | Systemic | 50%WP | R.B. Avari Entreprises Ltd. |
| 4 | Captan | Orthocide, Captane, Marpan, Vondcaptan (1986) | N- Trichloromethyl thio)-3a, 4,7,7a tetrahydrophthalimide | Contact | 50%WP | ICI (Pvt) Ltd. |
| 5 | Propineb | AntracolMenizeb (1974) | Zinc Prophylenebisdithio carbamate | Contact | 80%WP | Bayer (Pvt) Ltd |
| 6 | Carboxin | Vitavax, DCMO (1975) | 5,6 dihydro-2- methyl,1,4 oxathin, 3 Carboxanilide | Systemic | 75%WP | Longxiang Chem. Co. Ltd |
| 7 | Acrobat | Arbotect, Comfuvaz, Mertect, Mycozol (1962) | 2-(4-Thiazolyl) benzimidazole | Contact | 40-60%WP | Merck & Co. |
| 8 | Dithane M-45 | Mancozeb | 16%Mn, 2%Zn, 62% Ethylenebisdithiocarbama | Contact | 80% WP | Rohm & Hass Ltd |
| 9 | Thiophanate methyl | Topsin-M (1979) | 1,2-di (3- ethoxycarboxyl) – 2- thioureido benzene | Systemic | 70%WP | Pennwalt corp. |
| 10 | Chlorothalonil | Daconil, Bravo, Termil, Nopocide (1982) | Tetrachloroisophthalonitrile | Contact | 75%WP | Uniroyal Crop Div. |

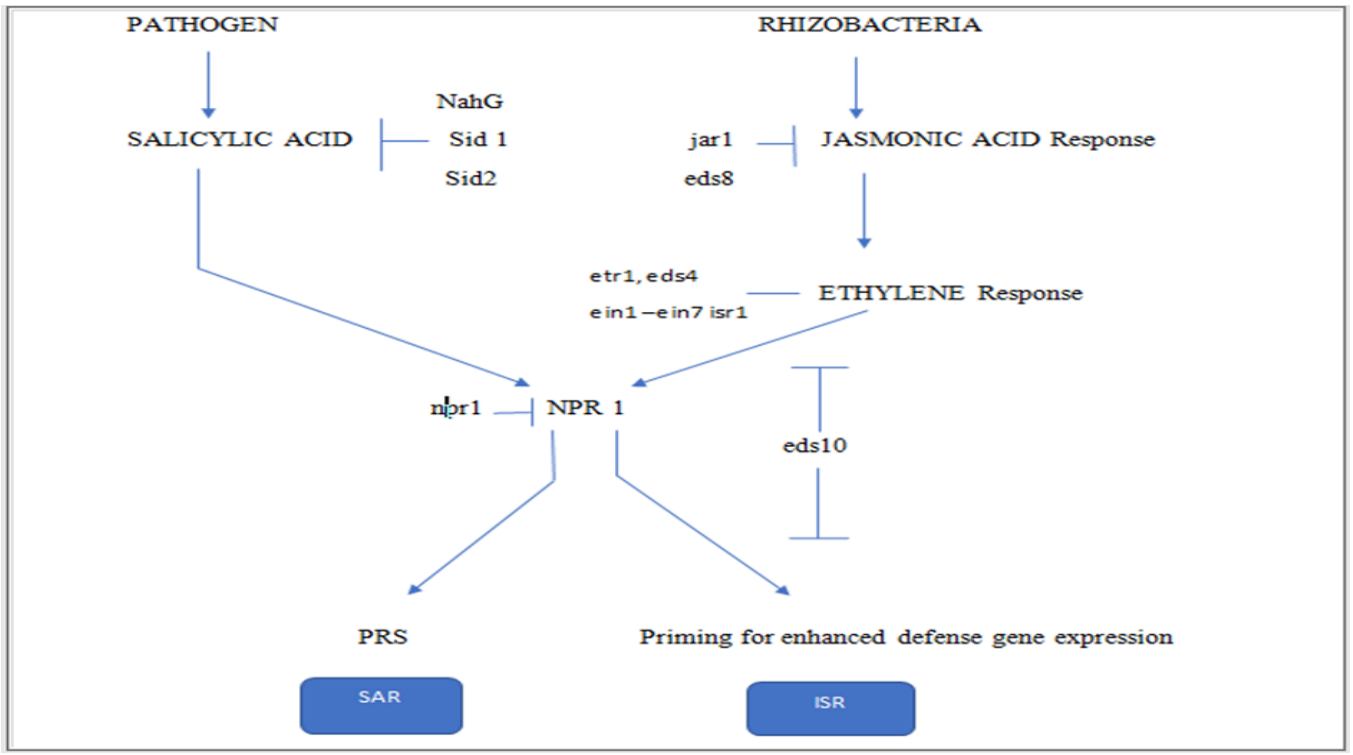


**Fig.3.** The pathogen-induced SAR and the rhizobacteria mediated ISR signal transduction pathways in *Arabidopsis* (21)

The rhizosphere microflora is crucial for plant growth and their adaptation to external challenges. Rhizobacteria that promote plant growth can also prevent disease by developing a systemic resistance in bacteria that combat soil-borne pathogens (31). Combining ISR and SAR can enhance protection against infections resistant to both pathways individually and extend protection to a wider range of pathogens compared to ISR or SAR alone (32). In *Arabidopsis*, three generally accepted pathways of induced resistance exist, two of which are associated with the direct production of pathogenesis-related (PR) proteins. One pathway typically triggers the production of PR proteins in response to attacks by pathogenic microorganisms, while the other is triggered by wound or necrosis-inducing plant pathogens. However, both pathways have alternative mechanisms for inducing resistance.

The wounding-induced pathway typically involves jasmonic acid (JA) as the signaling molecule, whereas the pathogen-induced pathway typically involves salicylic acid (SA), which is produced by the plant (33). When administered exogenously, these substances and their equivalents produce comparable effects, and there is undoubtedly significant cross-talk between the pathways (34). The JA-induced pathway is referred to as induced systemic resistance (ISR), which is also associated with various processes initiated by rhizobacteria. The pathways triggered by salicylate and jasmonate involve the production of a series of PR proteins, including anti-fungals (glucanases and chitinases), oxidative enzymes (such as peroxidases, thaumatins, polyphenol oxidases, and lipoxygenases) (35), and antibacterial low-molecular-weight compounds. Additionally, characteristics (phytoalexins) can assemble. Rhizobacteria-induced systemic resistance (RISR), also known as non-pathogenic root-associated bacteria, is a third type of induced resistance that contributes to the widespread evolution of plant disease resistance. When a plant is attacked by a pathogen, the plant's defenses are enhanced, and the severity of the disease is reduced. As the usual protein cascade induced by salicylate is absent in RISR, it potentiates plant defense responses.

## Role of Induced Systemic Resistance (ISR)

It is expected that plant roots in suppressive soils are connected to microbial populations that generally promote plant health. As a matter of fact, a number of bio-control PGPRs induce ISR in the host plant, allowing plants to survive pathogen attacks on leaves or roots without providing complete protection (36). ISR is elicited by several powerful biocontrol PGPR, regardless of antibiotic production (37). Transcriptome analysis of plants with roots colonized by different strains of *Pseudomonas* spp. (*P. fluorescens* WCS 417r, *P. thivervalensis*, and *P. fluorescens* CHA0) has revealed how these strains mediate ISR in *Arabidopsis thaliana*. Studies with mutant *A. thaliana* plants have shown that the salicylic acid (SA)-inducible route is involved in systemic acquired resistance, while the jasmonic acid (JA)/ethylene-inducible defense pathway is crucial for ISR (38). Hexenal, a volatile antifungal chemical, and the expression of enzymes involved in hexenal synthesis were increased in bean plants when ISR was induced by *P. putida* strain.

In order to address the issue of iron non-availability, particularly in calcareous soils, researchers have explored the role of siderophores, one of the factors of ISR, in influencing plant nutrition. This is achieved by incorporating strains of fluorescent pseudomonads that produce siderophores (FLPs) (39). A pot experiment using Fe-citrate, Fe-EDTA, and $Fe(OH)_3$ in varying concentrations was conducted to evaluate the effect of microbial siderophores on the iron nutrition of mung beans using the siderophores-producing bacterium *Pseudomonas*

strain GRP3. The chlorotic symptoms of the plants decreased, and their chlorophyll levels increased when the plants were infected with the bacteria. The peroxidase activity in the roots increased, while the catalase activity decreased. Moreover, both total and physiologically available iron increased significantly. Researchers have provided detailed information on the function of siderophores. This approach to siderophore production has the potential to increase iron availability to plants and reduce the need for fertilizers (40). Choudhary *et al.* (41) showed the effectiveness of a bacterial isolate to guard against pathogen infestation in both naturally occurring (*Pythium* and *Phytophthora* spp.) and artificially constructed (*Phytophthora* spp.) vegetable nurseries. After 21 days after seeding, tomato and chile plants were harvested, and their peroxidase and phenylalanine ammonia lyase (PAL) activity (ISR responsive proteins, not SAR-responsive proteins) were examined (42). Ganeshan *et al.* (43) discovered that the *Pseudomonas* sp. strains FQP-PB-3, FQP-PB-3, and GRP3 were the most effective in promoting shoot length and increasing PAL and peroxidase activity, which are well-known indicators of an active lignification process (44).

## Consequences of Management of Wilt

The current scenario is that a large number of chemicals are employed to treat ailments, but this has the unintended consequence of harming the ecosystem and endangering human health. Chemicals can also be used to develop disease resistance in plants. Induced systemic resistance is the name given to this form of resistance (ISR). Production of PAL, TPC, PO, and PPO occurs through many methods (45). Plants may fight disease caused by a range of pathogens through a number of processes that might be local or systemic, inducible or constitutive (46). Plant-derived bio-active substances can be used safely and successfully against disease. Due to their anti-bacterial activity against plant diseases, essential oils (EOs) and their derivative chemicals have attracted a lot of attention in recent years (47). The chemical makeup and anti-fungal activity of six plant EOs against *Fusarium oxysporum* f. sp. *ciceri* are investigated by the studies of (48), as well as the impact of essential oils on reducing the severity of fusarium wilt in chickpeas and their role in fostering systemic resistance by regulating phenolic and flavonoid compounds. ISR is used in a variety of microorganisms (*Trichoderma* spp., *Pseudomonas fluorescence*, *Bacillus* spp., and Rhizobacteria) as well as by products (seaweed, vermicompost, and vermiculite). Application of rhizobacteria-mediated induced systemic resistance of *Pseudomonas* spp. is capable of initiating plant-mediated resistance in above-ground plant sections (49). Plants that have been previously infected by a disease become more resistant to infection. This is referred to as acquired systemic resistance (49). Both local resistances mediated by key genes and systemic induced resistance generated after initial pathogen attacks are influenced by salicylic acid. SAR is aided by salicylic acid rather than ISR (50).

Plants treated with *P. fluorescence* showed a significant increase in shoot and root length. Wilt disease was reduced by 26-50%. Vitamin B2 (riboflavin) is a coenzyme that is created by plants and microorganisms and is used in a variety of physiological activities in plants, microbes, and animals. It also plays a role in both antioxidation and peroxidation (51). Non-Pathogenic strains isolated from suppressive soil strains had many modes of action against pathogenic strains and were used as biocontrol agents. Non-pathogenic strains fight for nutrients in the soil, limit chlamydospore germination, compete for infection sites on the root, and generate systemic resistance in plants that infiltrate host plant species before the pathogen (52). Biocontrol agents and chemical inducers worked best together to lessen the degree of damping off, root rot, or wilt and improve plant fresh weight (53). Saikia *et al.* (40) examined the effectiveness of *P. fluorescence* with or without modification in chickpea against fusarium wilt infection.

Jahan *et al.* (41) discovered that the *Bacillus subtilis* isolate k18 was an efficient wilt pathogen antagonist. Biochar is an excellent bio-fertilizer, bio-pesticide, and rhizobacteria carrying material. Chickpea output can be boosted by combining *Mesorhizobium ceceri* with a biochar amendment, which boosts growth and increases nodulation weight and number in the face of fungi like *F. oxysporum.* Biochar-treated plants produce more nodules and boost legume crop yields through heat tolerance, which is achieved by increasing the soil's water holding capacity and growing hostile microbial colonies (54). Biocontrol agents such as *Trichoderma harzianum, Aspergillus niger, B. subtilis, P. fluorescence, Rhizobium* spp.*,* and *Azospirillum* spp. can be used to control chickpea wilt. In dual culture, the bio control microorganisms such as *P. fluorescence* inhibit the pathogen (*F. oxysporum* f.sp. *ciceri*) growth by 70.94%, followed by *T. harzianum* (63.95%), *Rhizobium* spp. *(*60.79%), and *B. subtilis* (63.95%) (Table 4). Because wilt is a soil-borne pathogen, it is mostly controlled through chemical fumigation, such as methyl bromide, which has been outlawed due to health concerns, and then through the use of resistant types (42). However, in order to execute biological control commercially on a practical level, a better understanding of biocontrol agents' ecology and interactions with host plant pathogens, as well as the surrounding soil and rhizosphere, is required. Induced resistance has been proposed as a mechanism for non-pathogenic *F. oxysporum*-induced disease management.

Studies by Bekkar *et al.* (43) showed that tomato plants cultivated in suppressive soil had increased levels of hydrolytic enzymes associated with the PR protein. The use of rhizobacteria, combined with resistant cultivars and appropriate planting dates, may help manage fusarium wilt in chickpeas. *Paenibacillus* spp. and *Pseudomonas* spp. strains have demonstrated potential in reducing fusarium wilt infections in other crops, including chickpea, cotton, and radish (55). The induction of systemic resistance by plant growth-promoting rhizobacteria (PGPR) is dependent on the plant hormones jasmonic acid and ethylene. Various inorganic and organic compounds, as well as extracts from plants and microorganisms, have been reported to induce disease resistance in plants,

including INA (2, 6-dichloro-isonicotinic acid) and BTH (benzo 1, 2, 3) under the trademark BION.

Chemicals like salicylic acid, 2, 6-dichloro-isonicotinic acid (INA), and non-pathogenic bacteria can all cause systemic resistance. Chitosan, a polysaccharide, has been found to protect plants from diseases and can be used as soil additive, seed, and foliar spray (45). The accumulation of phenolic acid is linked to enzymes like polyphenol oxidase (PPO) and phenylalanine ammonium for pathogen attack (PAL) (Table 3). Chitosan significantly reduced the seed borne infection which ranged from 59 to 23 % (56). Gas chromatography identified six cinnamic acids, eight benzoic acids, and one cinnamic acid ester, as well as an increase in lignin concentration in chitosan-treated seeds. Biocontrol agents have been genetically modified through physical and chemical means to develop biocontrol agents with better toxicant tolerance, enhanced antagonistic potential, and improved survivability in the agro environment (47).

Seeds were treated with benzo (1, 2, 3)-thiadiazole-7- carbothioic acid S-methyl ester (Bion), salicylic acid, and di-potassium hydrogen phosphate to induce systemic resistance in chickpeas against wilt disease caused by *F. oxysporum* f. sp. *ciceri* ($K_2PHO_4$). Both seed dressing and soaking methods resulted in a reduction in infection. The highest reduction in wilt disease, 63%, was induced by bion dressing, followed by salicylic acid at 40% and $K_2PHO_4$ 30%. Bion and salicylic acid showed a 41 and 24% reduction in the disease, respectively, and $K_2PHO_4$ soaking indicated a reduction of 30% (48) (Table 5).

## Conclusion

Wilt disease is a serious problem in many crop plants as the pathogen has a high competitive saprophytic ability, allowing it to survive in the soil for extended periods. In recent years, biological control of fusarium wilt infections has been a major consideration in disease management. Induced resistance plays a significant role in suppressing wilt disease from a crop protection standpoint. To develop viable bio-control techniques for commercial situations, it is essential to have a better understanding of the mechanisms involved in the protection of plants by biocontrol agents. Improved forecasting of disease development and more effective utilization of biocontrol agents for managing fusarium wilt can be achieved by understanding how the inoculum density of *F. oxysporum* f. sp. *ciceri* affects disease development. Directly promoting plant growth, biological control, and developing systemic resistance in host plants are some of the advantageous impacts of PGPR. Certain strains of PGPR can induce ISR against multiple diseases affecting the same crop. The use of PGPR significantly reduces insect and nematode damage in addition to disease control. Therefore, in the present scenario, an alternative eco-friendly module for managing the disease and sustainable crop production is very much needed. Induced resistance is a healthy method of controlling the disease as it strengthens the host plant by increasing its resistance. Induced resistance through PGPRs, *Pseudomonas, Bacillus, Trichoderma* spp., and the use of salicylic acid as an inducer, are effective ways to prevent and suppress fusarium wilt chickpea.

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## Authors' contributions

AK conceived the idea of writing the review and designed the content. VK and SR collected the literature and prepared the article. All authors contributed to the content of the manuscript and approved the final version.

## Compliance with ethical standards

**Conflict of interest:** Authors do not have any conflict of interests to declare.

**Ethical issues:** None.

**Table 5.** Control of chickpea wilt by chemical control and within induced resistance

| Chemicals | Nature of disease control | Year | Reference |
|---|---|---|---|
| Bavastin 0.5g/kg of seed | Improved germination and disease control of wilt by 23.7% | 2011 | (46) |
| Benalate 0.15% (S.T) | Destroy seed borne inoculum completely | 2011 | (46) |
| Benomyl (soil drench) | Very effective in controlling wilt | 2011 | (46) |
| Bavistin + Thiram (2.5g/kg seed) | Decreased disease and increased yield under field condition | 2009 | (44) |
| Chitosan | Seed treatment at 0.3 and 1 % .Wilt symptoms reduced by 45-59% and prevented plant mortality. Enhanced polyphenol oxidase, pero-oxidase and phenylalamine ammonia lyase activities usually associated with defense. | 2007 | (21) |
| Salicylic acid | Seed soaking at 1.0 and 1.5 mM conc. | 2003 | (45) |
| Bion | Seed soaking at 0.3 and 0.4mM conc. Wilt was reduced in all treatments | 2005 | (34) |

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