Review Article

Induced resistance mechanism in plant and its importance in agriculture

ABSTRACT

The hazardous effect of chemical pesticides and their degradation products on the environment and human health led to the search for a successful and effective natural phenomenon of induced resistance in plant. The resistance in plants induces by pathogen was first recognized by Ray, 1901. Induced resistance was first described in *Arabidopsis* plants, inoculated with the root-colonizing on pathogenic bacteria *Pseudomonas fluorescens.* Induced resistance are of two types *i.e*., induced structural defense and induced biochemical defense. Structural defense comprises of cytoplasmic defence reaction, cell wall defense structure and histological defense structure (formation of cork layers, abscission layer and tylose) while biochemical defense includes Phytoalexins PR-proteins and secondary metabolites compounds. The mechanism of induced resistance is mainly based on systemic acquired resistance (SAR) and induced systemic resistance (ISR). In SAR, the defense mechanism is salicyclic mediated, mainly changes in gene expression whereas in ISR, jasmonic acid and ethylene are mediated, and also induced by non-pathognic rhizobacteria (*Pseudomonas fluorescens.*).

Exogenous application of 2, 6-dichloroisonicotonic acid and benzo- thiadiazole-7-carbothioic acid S-methyl ester (BTH), induces Resistance in plants, to certain pathogens. Although not fully understood, induced resistance in plants opens new horizons in plant protection, being a promising tool for ecologically-friendly disease control and sustainable agriculture. It remains a challenge for both fundamental and applied research.

KEY WORDS:

Induced resistance mechanism, Acquired resistance, Plant resistance, Agriculture, pathogenesis related proteins" (PRs), Benzothiadiazole, induced systemic resistance" (ISR)

INTRODUCTION

In nature, plants are continuously threatened by a wide range of harmful pathogens and pests, including fungi, oomycetes, bacteria, viruses, nematodes, and insect herbivores. According to their lifestyles, plant pathogens are generally divided into necrotrophs and biotrophs. Necrotrophs first destroy host cells, often through the production of phytotoxins, after which they feed on the contents while Biotrophs derive nutrients from living host tissues

commonly through specialized feeding structures (haustoria) that invaginate the host cell without disrupting it. But most of plant pathogens display both lifestyles depending on their life cycle are called hemi biotroph.

Presently disease control is largely based on the use of fungicides, bactericides and insecticides – chemical compounds toxic to plant invaders causative agents or vectors of plant diseases. However, the hazardous effect of these chemicals or their degradation products on the environment and human health strongly invite for new harmless means of disease control. Since the late 1950s increasing body of evidence on the natural phenomenon of induced resistance has been accumulated, culminating in its successful practical application in the last decade (Kuc, 2001). The resistance in plants induced by pathogens was first recognized by Ray (1901) and Beauverie (1901). Chester (1930) confirmed those studies, and, by summarizing field observations, supposed that this phenomenon may play an important role in the preservation of plants in nature. Convincing evidences however were obtained only in the 1960s, when reproducible models using tobacco plant were developed (Cruickshank and Mandryk, 1960;; Mandryk, 1963). Greenhouse and field experiments in the laboratory of Kuc and co-workers paved the way to the present comprehension of induced resistance as a tool in plant protection (Kuc, 2001), this being supported by numerous authors from around the world (Schönbeck *et al*., 1993; Kessman *et al*., 1994; Schneider *et al*., 1996; Van Loon *et al*., 1998; Benhamou and Picard, 1999; Tally *et al*., 1999; Cohen, 2001; Bokshi *et al*., 2003; Gozzo, 2003; Soylu *et al*., 2003). Exploiting uniquely the plant potential to combat pathogens, the induced resistance may diminish the use of toxic chemicals for disease control, and thus could be proposed as an alternative, non-conventional, non-biocidal and ecologically-friendly approach for plant protection and hence for sustainable agriculture.

Induced resistance

The induced resistance can be defined as an increased expression of natural defence

mechanisms of plants against different pathogens provoked by various type external factors. The term "**induced resistance**" (IR) is used synonymously to "**acquired resistance**" (AR). Depending on the mode of its expression, induced resistance can be systemic (SAR) or local (LAR). In the early 1960s Ross as a result of his carefully controlled laboratory experiments with tobacco-TMV system coined the terms LAR (Ross,1961a) and SAR (Ross, 1961b). Recently, the term "**induced systemic resistance**" (ISR) was introduced to designate the resistance induced in leaves of plants by inoculation of roots with non-pathogenic rhizobacteria. This novel type of induced resistance was first described in *Arabidopsis* plants,

inoculated with the root-colonizing on pathogenic bacteria *Pseudomonas fluorescens*; leaves of these plant revealed resistance against the bacterial leaf pathogen Pseudomonas syringae pv. Tomato (Pieterse *et al*., 1998). Rhizo-bacteria-mediated ISR has also been demonstrated against fungi, bacteria and viruses in Arabidopsis, bean, carnation, cucumber, radish, tobacco and tomato (Van Loon *et al*., 1998)

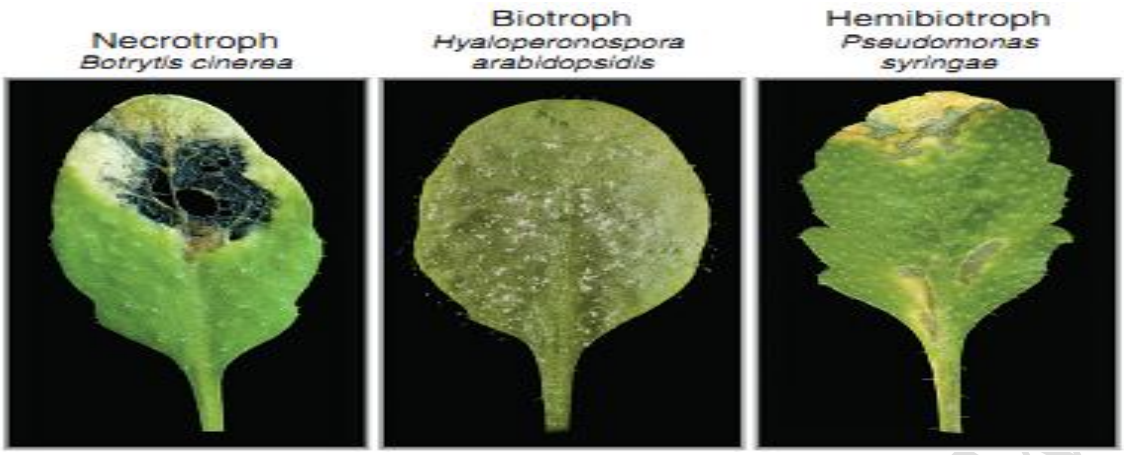


Fig.1 *Disease symptoms on Arabidopsis leaves caused by the necrotrophic fungus Botrytis cinerea, the botrophic oomycet Hyaloperonospora arabidopsidis, and the hemibiotrophic bacterium Pseudomonas syringae.*

Classification of Induce Resistance:

Mainly there are two types of induce resistance found in plants.

I. Induced Structural Defences II. Induce Biochemical Defences

I. Induced Structural Defences

As it is well known that a pathogen must penetrate before it can cause infection. If pathogen gains entry into the host, its movement and further spread is checked through parenchymatous cells. Such defence structures i.e. Waxes, cuticles, structure of epidermal cell wall, structure of natural opening etc., may be present before penetration or afterwards as a result of the infection of the host and the pathogen.

Most pathogens start to entry their hosts through wounds and natural opening and later produce various degrees of infection. Even after the pathogen has penetrated the preformed defence structures, however, plants usually respond by forming one or more types of structures that are more or less successful in defending the plant from further pathogen invasion. Some of the defence structures formed in the cytoplasm of the cells under attack is called **cytoplasmic defence reaction**; the walls of invaded cells are called **cell wall defence structures**; and still others involve tissues ahead of the pathogen (deeper into the plant) are called **histological defence structures**. Finally, the death of the invaded cell may protect the plant from further invasion is called the **necrotic** or **hypersensitive defence reaction.**

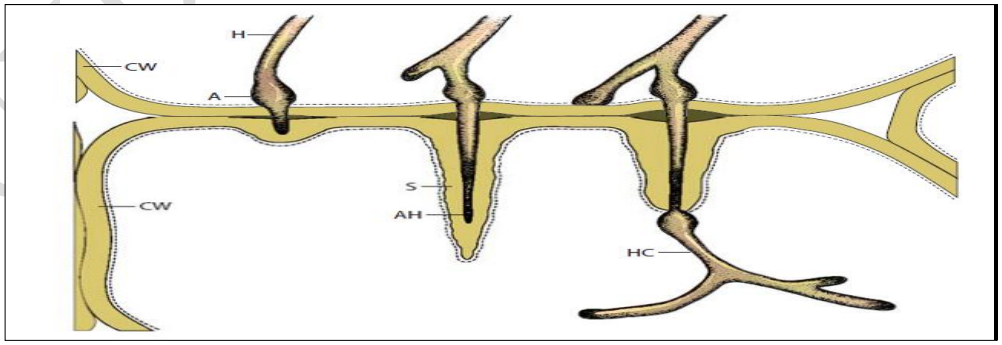


FIG. 2 *Formation of a sheath around a hypha (H) penetrating a cell wall (CW). A, appressorium; AH, advancing hypha still enclosed in sheath; HC, hypha in cytoplasm; S, sheath.*

A) Cytoplasmic Defence Reaction In a few cases of slowly growing, weakly pathogenic fungi, such as weakly pathogenic *Armillaria* strains and the mycorrhizal fungi, that induce chronic diseases or nearly symbiotic conditions, the plant cell cytoplasm surrounds the clump of hyphae and the plant cell nucleus is stretched to the point where it breaks into two. In some cells, the cytoplasmic reaction is overcome and the protoplast disappears while fungal growth increases. In some of the invaded cells, however, the cytoplasm and nucleus enlarge. The cytoplasm becomes granular and dense, and various particles or structures appear in it. Finally, the mycelium of the pathogen disintegrates and the invasion stops.

B) Cell Wall Defence Structures

Cell wall defence structures involve morphological changes in the cell wall or changes derived from the cell wall of the cell being invaded by the pathogen. The effectiveness of these structures as defence mechanisms seems to be rather limited, however. Three main types of such structures have been observed in plant diseases.

(1) The outer layer of the cell wall of parenchyma cells coming in contact with incompatible bacteria swells and produces amorphous, febrillar materials that surround and traps the bacteria and prevents them from multiplying.

(2) Cell walls thicken in response to several pathogens by producing what appears to be a cellulosic material. This material, however, is often infused with phenolic substances that are cross-linked and further increase its resistance to penetration.

(3) **Callose papillae** are deposited on the inner side of cell walls in response to invasion by fungal pathogens .Papillae seem to be produced by cells within minutes after wounding and within 2 to 3 hours after inoculation with microorganisms. Although the main function of papillae seems to be repair of cellular damage, sometimes, especially if papillae are present before inoculation, they also seem to prevent the pathogen from subsequently penetrating the cell. In some cases, hyphal tips of fungi penetrating a cell wall and growing into the cell lumen are enveloped by cellulosic (callose) materials that later become infused with phenolic substances and form a sheath or lignin tuber around the hypha.

C) Histological Defence Structures (Defence structures formed after infection)

1. Formation of Cork Layers

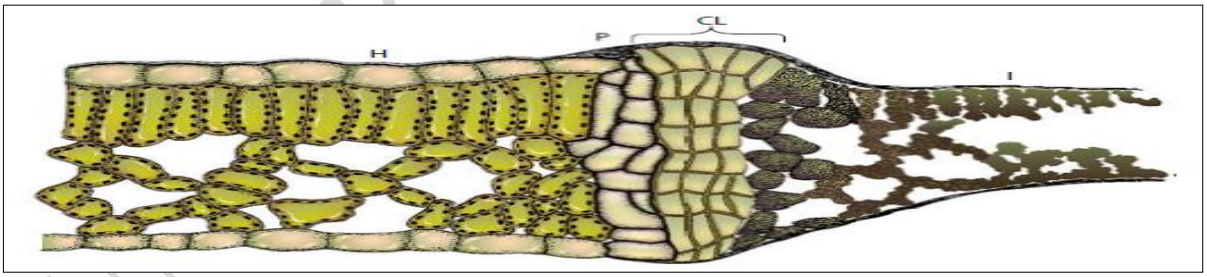


FIG. 3 *Formation of a cork layer (CL) between infected (I) and healthy (H) areas of leaf. P, phellogen. [After Cunningham (1928). Phytopathology* ***18****, 717–751.]*

Infection by fungi or bacteria, viruses and nematodes induces plants to form several layers of cork cells beyond the point of infection as a result of stimulation of the host cells by substances secreted by the pathogen. The cork layers inhibit further invasion by the pathogen beyond the initial lesion and also block the spread of any toxic substances. Furthermore, cork layers stop the flow of nutrients and water from the healthy to the infected area and deprive the pathogen of nourishment. The dead tissues, including the pathogen are thus delimited by the cork layers and may remain in place, forming necrotic lesions (spots) that are remarkably uniform in size and shape for a particular host–pathogen

combination. In tree cankers, such as those caused by the fungus *Seiridium cardinale* on cypress trees, resistant plant clones restrict growth of the fungus by forming ligno-suberized boundary zones, which included four to six layers of cells with suberized cell walls. In contrast, susceptible clones have only two to four layers of suberized cells and these are discontinuous, allowing repeated penetration by the fungus past the incomplete barrier.

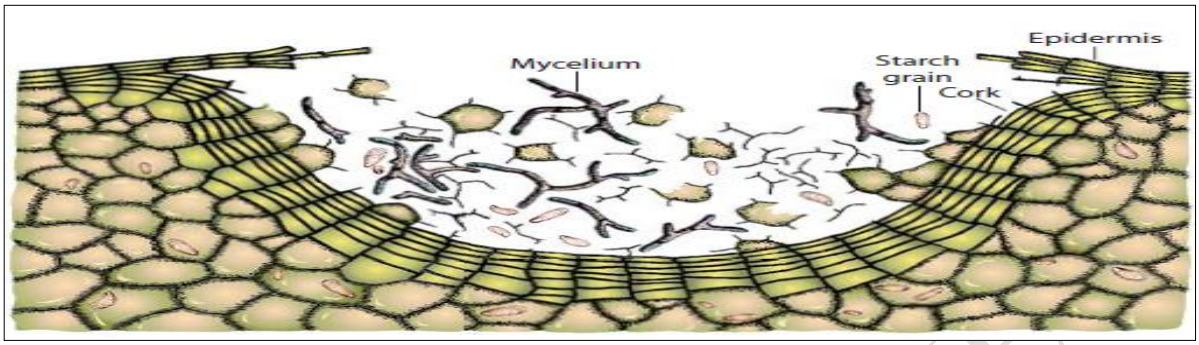


FIG. 4 *Formation of a cork layer on a potato tuber following infection with Rhizoctonia. [After Ramsey (1917). J. Agric. Res.* ***9****, 421–426.]*

Formation of Abscission Layers

Abscission layers are formed on young, active leaves of stone fruit trees after infection by any of several fungi, bacteria, or viruses. An abscission layer consists of a gap formed between two circular layers of leaf cells surrounding the locus of infection. Upon infection, the middle lamella between these two layers of cells is dissolved throughout the thickness of the leaf, completely cutting off the central area of the infection from the rest of the leaf (Fig. 6-7). Gradually, this are a shrivels, dies, and sloughs off, carrying with it the pathogen.

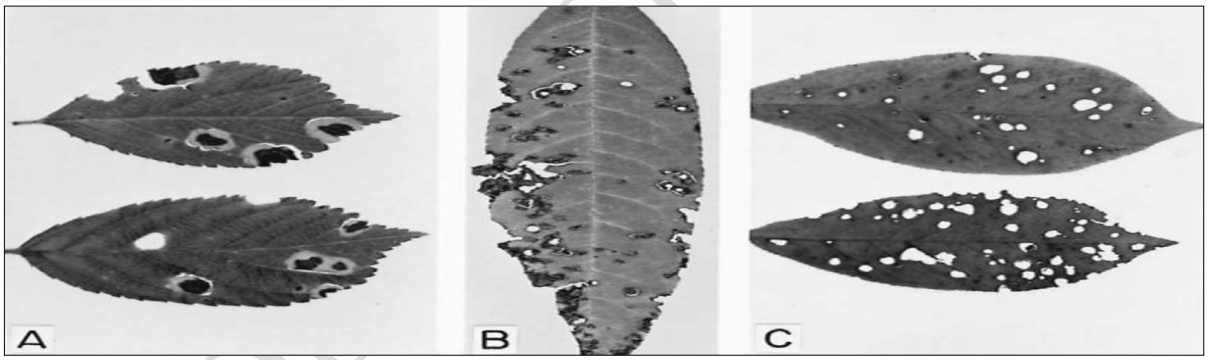


Fig. 5 *Schematic formation of an abscission layer around a diseased spot of a Prunus leaf. (A–C) Leaf spots and shot holes caused by Xanthomonas arboricola pv. pruni bacteria on (A) ornamental cherry leaves; characteristic broad, light green halos form around the infected area before all affected tissue falls off, (B) on peach, and (C) on plum. The shot hole effect is particularly obvious on the plum leaves.*

Formation of Tyloses

Tyloses form in xylem vessels of most plants under various conditions of stress and during invasion by most of the xylem-invading pathogens. Tyloses are over growths of the protoplast of adjacent living parenchymatous cells, which protrude into xylem vessels through pits .Tyloses have cellulosic walls and may, by their size and numbers, clog the vessel completely.

Deposition of Gums

Gum secretion is most common in stone fruit trees but occurs in most plants. The defensive role of gum stems from the fact that they are deposited quickly in the intercellular spaces and within the cells surrounding the locus of infection, thus forming an impenetrable barrier

that completely encloses the pathogen. The pathogen then becomes isolated, starves, and sooner or later dies.

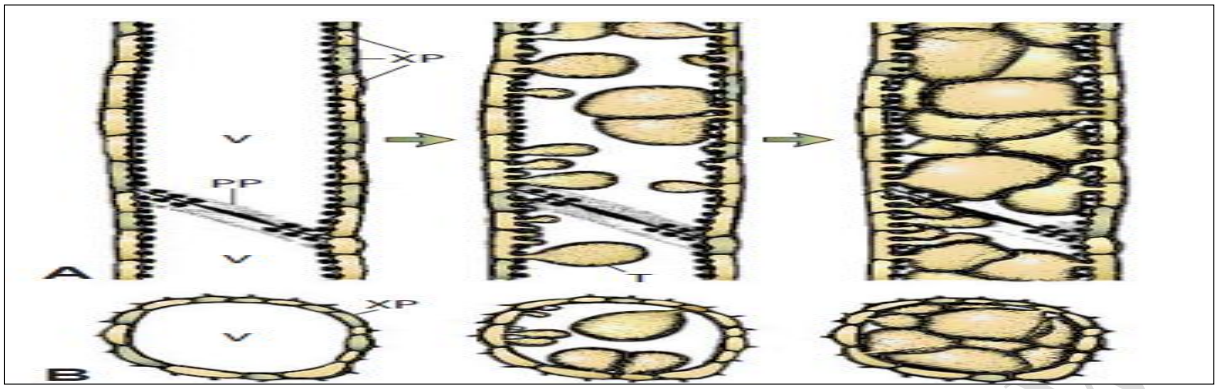


FIG. 6 *Development of tyloses in xylem vessels. Longitudinal (A) and cross section (B) views of healthy vessels (left) and of vessels with tyloses. Vessels at right are completely clogged with tyloses. PP, perforation plate; V, xylem vessel; XP, xylem parenchyma cell; T, tylosis.*

II. **Biochemical defence mechanism:** Inhibitors released by the plant in its environment defence through productions of Phytoalexins, PR Proteins and secondary metabolic compounds i.e., 2,6-dichloro-isonicotinic acid (INA), BTH**,** salicylic acid, Jasmonic acid etc.,

Phytoalexins are phenolic compounds which are not present in healthy plants but are produced upon stimulation of a plant by a pathogen or by a mechanical or chemical injury and derived from Greek word phyto meaning plant ; alexin means warding off compound. They are antimicrobial low molecular weight compounds and formed only when the host cells come into contact with the parasite. The resistance state is not inherited and confined to the tissue colonized by the fungus and its immediate neighbourhood.

PR Proteins: A group of plant-coded proteins induced by different stress stimuli, as "**pathogenesis related proteins" (PRs)** is assigned an important role in plant defence against pathogenic constraints and in general adaptation to stressful environment. Antonie *et al*. (1980) coined the term "pathogenesis-related proteins" (PRs), which have been defined as "proteins encoded by the host plant but induced only in pathological or related situations. Among the PRs, a protein has to be newly expressed upon infection but not necessarily in all pathological conditions refer to all types of infected states, not just to resistant, hypersensitive responses in which PRs are most common; they also include parasitic attack by nematodes, insects and herbivores. Induction only by abiotic stress conditions is not a sufficient criterion for inclusion as PRs. These considerations imply that the characteristics of the induction of PRs take priority over other identifying features, such as chemical properties or cellular localization (Van Loon *et al*., 1994; Van Loon, *et al*., 1999).

Originally, five main groups of PRs (PR-1 to PR-5) have been characterized by both molecular and molecular-genetic techniques in tobacco, numbered in order of decreasing electrophoretic mobility. Each group consists of several members with similar properties (Bol *et al*., 1990) (Table 1). Group PR-1 is the most abundant, reaching up to 1-2 % of total leaf proteins. PRs of group 5 share significant amino acid sequence homology with the sweet tasting protein in the fruits of the tropical plant *Thaumatococcus daniellii*, and have been named thaumatin-like (TL) proteins (Cornelissen *et al*., 1986).

Table 1. PR proteins induced in Samsun tobacco (NN genotype) by TMV infection (Bol *et al*. 1990).

Group	Acidic PR proteins		Basic PR proteins		Function
	Name	Mol wt (KD)	Name	Mol wt (KD)	
1	1a	15.8	16 KD	16.0	Unknown
	1b	15.6			
	1c	15.5			
2a	2	39.7	Gluc .b	33.0	β- 1.3-gluconase
	N	40.0			
	O	40.6			
	Q^+	36.0			
2b	O^+	25.0			β- 1.3-gluconase
3	P	27.5	Ch 32	32.0	Chitinase
	Q	28.5	Ch 34	34.0	
4	s1	14.5			Unknown
	r1	14.5			
	s2	13.0			
	r2	13.0			
5a	R	24.0	Osmotin	24.0	Unknown thaumatic type proteins
	S	24.0			
5b			45 KD	45.0	Unknown

An important common feature of most PRs is their antifungal effect, antibacterial, insecticidal, nematicidal, and – as recently shown – antiviral action. Toxicity of PRs can be generally accounted by their hydrolytic, proteinase- inhibitory and membrane-permeabilizing ability. Thus, hydrolytic enzymes (β-1,3-glucanases, chitinases and proteinases) can be a tool in weakening and decomposing of fungal cell walls, containing glucans, chitin and proteins, while PR-8 can disrupt gram-positive bacteria due to lysozyme activity (Van Loon and Van Strien, 1999; Van Loon, 2001; Selitrennik off, 2001).

Relevance of PRs to disease resistance.

The following lines of supporting evidence can be outlined as

a) Stronger accumulation of PRs in inoculated resistant as compared to susceptible plants. Besides previous data, substantiating this statement (Van Loon, 1985, and references therein), differential responses of resistant/susceptible plants were reported in tomato plants, inoculated with *Cladosporium fulvum* (Wubben *et al*., 1996); *Phytophthora infestans*-infected potato (Tónon *et al*., 2002); *Venturia inaequalis*-inoculated apple (Poupard *et al*., 2003); *Pseudomonas syringae*-infected grapevine (Robert *et al*., 2001); *Xanthomonas campestris* pv. *vesicatoria* and TMVPo- infected hot pepper (Park *et al*., 2004 a, b), etc.

b) Important constitutive expression of PRs in plants with high level of natural disease resistance. This correlation was observed in several pathosystems, such as apple – *Venturia inaequalis* (Gau *et al*., 2004), tomato – *Alternaria solani* (Lawrence *et al*., 2000), and potato – *Phytophthora infestans* (Vleeshouwers *et al*., 2000), the last authors proposing PR mRNAs as molecular marker in potato breeding programs.

c) Significant constitutive expression of PRs in transgenic plants over expressing PR genes accompanied by increased resistance to pathogens. Thus, increased tolerance to *Peronospora tabacina* and *Phytophthora parasitica* var. *nicotianae* was recorded in tobacco overexpressing PR1a gene (Alexander *et al*., 1993). Transgenic rice and orange plants overexpressing thaumatin-like PR-5 revealed increased tolerance to *Rhizoctonia solani* and

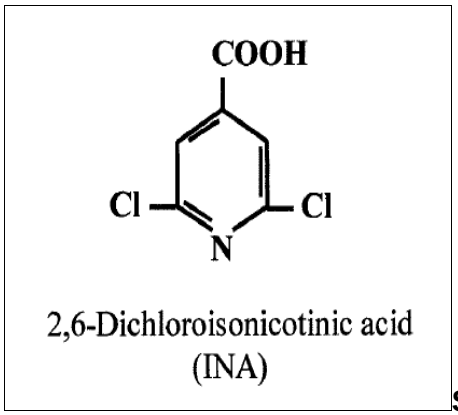
Phytophthora citrophthora, respectively (Datta *et al*., 1999; Fagoaga *et al*., 2001), while transgenic potato overexpressing PR-2 and PR-3 improved resistance to *Phytophthora infestans* (Bachmann *et al*., 1998). *Puccinia graminis* f. sp. *hordei* in the leaves (Schultheiss *et al*., 2003).

d) Accumulation of PRs in plants in which resistance is locally or systemically induced. Generalizing this broad research area it can be stated that PRs are recognized as markers of the systemic acquired resistance (SAR), and PRs genes are involved in the list of the so-called SAR-genes (Ward *et al*., 1991). Some SAR-inducing chemicals viz., benzothiadiazole (BTH), β-aminobutyric acid (BABA) or 2,6-dichloroisonicotinic acid (DCINA) are harmless commercially supplied compounds and have promising practical application as novel tools in plant protection (Van Loon, 1997; Ku_, 2001; Edreva, 2004 and references therein).

PRs members induced in resistant or SAR- expressing plants, as well as PRs from transgenic resistant plants exhibit high antimicrobial activity (Enkerli *et al*., 1993; Anfoka and Buchenauer, 1997; Rauscher *et al*., 1999; Tonón *et al*., 2002; Anand *et al*., 2004), this suggesting their direct role in disease resistance.

Table 2. Recognized and proposed families of pathogenesis-related proteins (Van Loon, Van Strien,1999)

Family	Type member	Properties
PR-1	Tobacco PR-1a	unknown
PR-2	Tobacco PR-2	β-1,3-glucanase
PR-3	Tobacco P, Q	chitinase type I, II, IV, V, VI, VII
PR-4	Tobacco "R"	chitinase type I, II
PR-5	Tobacco S	thaumatin-like
PR-6	Tomato Inhibitor I	proteinase-inhibitor
PR-7	Tomato P_{6g}	endoproteinase
PR-8	Cucumber chitinase	chitinase type III
PR-9	Tobacco "lignin-forming peroxidase"	peroxidase
PR-10	Parsley "PR1"	"ribonuclease-like"
PR-11	Tobacco class V chitinase	chitinase type I
PR-12	Radish Rs-AFP3	defensin
PR-13	*Arabidopsis* THI2.1	thionin
PR-14	Barley LTP4	lipid-transfer protein



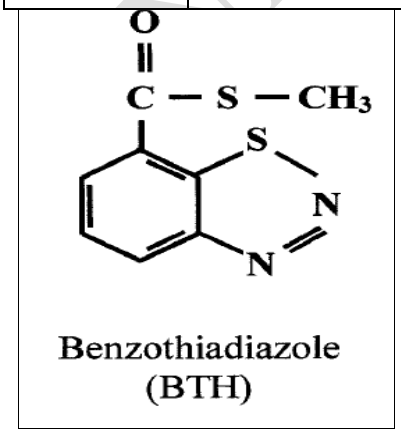
Secondary Metabolic Compounds

Pic 1.

2,6-dichloro-isonicotinic acid (INA): The first activator described was 2,6-dichloro-isonicotinic acid (INA). INA induced systemic resistance against a broad range of pathogens in several plant species and made available widely for research as well as effective in decreasing foliar diseases in green beans *Phaseolus vulgaris* (Dann and Deverall1995, 1996) in growth chamber and the field (Table 1). It decreased powdery mildew in cucumber (Hijwegenand Verhaar 1995) and barley (Kogel *et al*. 1994), and infections by *Cercospora beticola* in sugar beet(Nielsen *et al*. 1994) and also powdery mildew in roses (Hijwegen *et al*. 1996).

Table 3: **Control of plant diseases by INA**

Crops	**Disease/ Pathogen**	**Reference**
Barley	Powdery Mildew/ *Erysiphe graminis*	Kogel et al. (1994)
Cucumber	Powdery Mildew/*Spheerotheca fuliginea*	Hijwegen et al. (1995)
Cucumber	Anthracnose/ *Colletotrichum lagenarium*	Metraux et al. (1991)
Green Bean	Anthracnose/ *Uromyces appendiculatus*	Dann and Deverali (19954, 1996)
Pepper	Bacterial spot/ *Xanthomonas campestris*	Staub et al. (n.d)
Rice	Rice blast/ *Pyricularia oryzae*	Staub et al. (n.d)
Tobaco	Blue mould/ *Peronaspora tobacina*	Staub et al. (n.d)

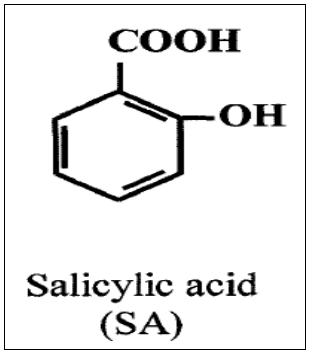


Pic 2.

Benzothiadiazole (BTH): The second activator was benzothiadiazole (BTH), which was particularly useful for patho-systems in wheat, rice, tobacco, and some vegetable crops (Table 3). BTH applied early in the growth of wheat recorded effective protection against powdery mildew for the season, and some protection against leaf rust and Septoria leaf spot (Gorlach *et al*. 1996). BTH decreased infection by fungi, bacteria, and viruses in tobacco (Friedrich *et al.* 1996) and in *Arabidopsis* (Lawton *et al.* 1996). BTH was registered for commercial use in Europe in 1996 against powdery mildew in wheat. BTH (50 ppm) significantly reduced rust severity in experiments in which it was sprayed onto fababean leaves 4 days before the challenge inoculation with *Uromyces viciae-fabae* spores.

Table 4 : **Control of plant diseases by BTH**

Crops	**Disease/ Pathogen**	**Reference**
Cucumber	Downey Mildew/*Pseudomonas cubensis*	Ciba data
Rice	Rice blast/ *Pyricularia oryzae*	Ciba data
Tobaco	Blue mould/ *Peronaspora tobacina*	Friedrich et al. 1996
Tomato	Bacterial spot/ *Xanthomonas spp*	Ciba data
Wheat	Powdery Mildew/ *Erysiphe graminis*	Goriach et al. 1996



Pic 3.

Salicylic Acid (Latin word *salix*, *willow tree*, from the bark of which the substance used to be obtained) is derived from the metabolism of salicin and a monohydroxybenzoic acid a type of phenolic acid and also a beta hydroxy acid. This colorless crystalline organic acid is widely used in organic synthesis and functions as a plant hormone. In addition to being a compound that is chemically similar but not identical to the active component of aspirin (*acetylsalicylic acid)* and best known for its use in anti-acne treatments. The salts and esters of salicylic acid are known as **salicylates**.

Salicylic acid also reverses the closure of stomata caused by abscisic acid (Rai *et al*., 1986). Exogenous application of salicylic acid improves the yield in crops (Singh & Kaur, 1980; Arfar *et al.*,2001). SA retards ethylene synthesis; stimulates photosynthetic machinery and increase the content of chlorophyll (Leslie & Romani, 1988). Recently, it has been recognized that salicylic acid is required in the signal transduction for inducing systemic acquired resistance against pathogenic infections (Metraux *et al*., 1990; Gaffney *et al*., 1993; Vernooij *et al*., 1994).

Salicylic Acid Biosynthesis-

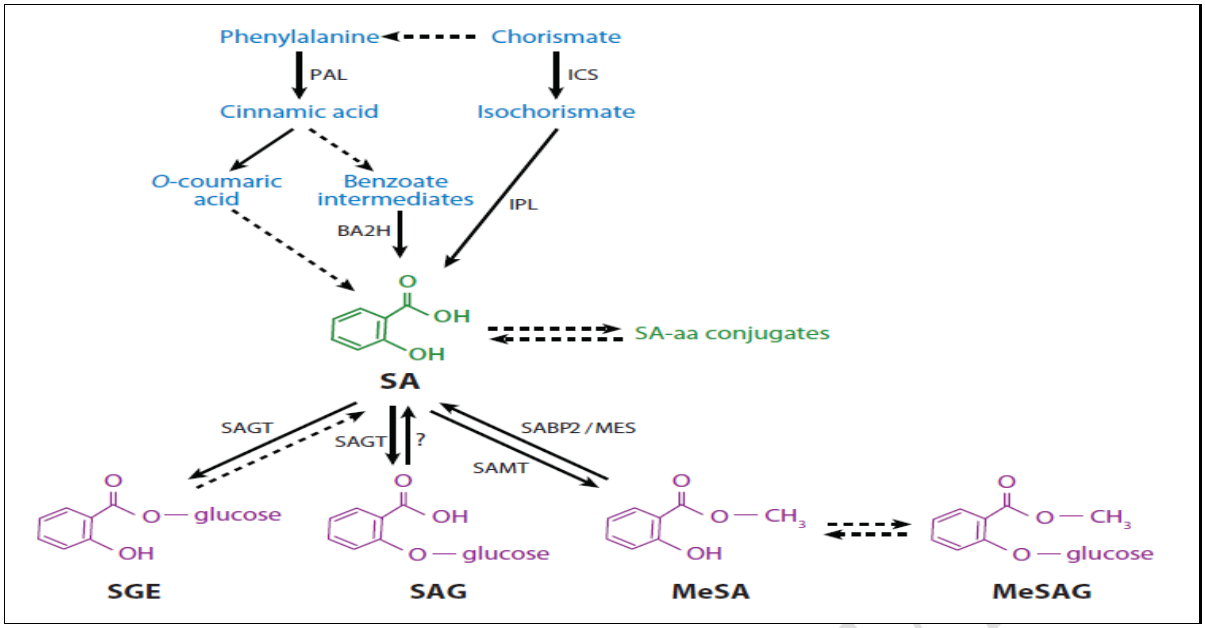


FIG.7 *Simplified schematic of pathways for SA biosynthesis and metabolism as adapted from Garcion & M´etraux(60). Abbreviations: PAL, phenylalanine ammonia lyase; ICS, isochorismate synthase; IPL, isochorismate pyruvate lyase; BA2H, benzoic acid-2-hydroxylase; SA, salicylic acid; SAGT, SA glucosyl transferase; aa, amino acid; SAMT, SA methyl transferase; SABP2, SA-binding protein 2; MES, methyl esterase; SGE, salicyloyl glucose ester; SAG, SA O-β-glucoside; MeSA, methyl salicylate; Me SAG, methyl salicylate O-β-glucoside.*

SA in plants can be generated via two distinct enzymatic pathways that require the primary metabolite chorismate (Garcion C *et al*., Wildermuth MC *et al*.,. 2006). Chorismate-derived l-phenylalanine can be converted into SA via either benzoate intermediates or coumaric acid via a series of enzymatic reactions initially catalyzed by Phenylalanine Ammonia Lyase (PAL). Chorismate can also be converted into SA via isochorismate in a two stepprocess involving ISOCHORISMATE SYNTHASE (ICS) and ISOCHORISMATEPYRUVATE LYASE (IPL) (Fig 7) (Strawn MA *et al*.,2007, Verberne M.C. *et al*., 2000; Wildermuth MC *et al*.,2001): the bulk of pathogen-induced SA is synthesized via this pathway in *Arabidopsis*, *Nicotiana benthamiana*, and tomato (Catinot J *et al*., 2008, Wildermuth MC *et al*.,2001, Uppalapati SR *et al*.,2007). *Arabidopsis* encodes two ICS enzymes; SA production, as well as pathogen resistance, is severely compromised in mutants lacking functional ICS1, which appears to be responsible for approximately 90% of SA production induced by pathogens or UV light (Garcion C *et al*., 2008).The appearance of residual SA in an *ics1/ics2*double mutant confirms that the *ICS* pathway is not the only source of SA in *Arabidopsis*.Most of the SA produced in plant is converted into SA *O-β*-glucoside (SAG) by a pathogen-inducible SA glucosyl transferase (SAGT) (Fig 7) (Dean JV *et al*., 2004 ,Dean JV *et al*., 2005 Dean JV *et al*., 2003 Lee H-I *et al*., 1998 Lee H-I1999 Song JT *et al*., 2006).

Arabidopsis encodes two SAGT enzymes; one preferentially converts SA into SAG, whereas the other forms the less abundant SA derivative, salicyloyl glucose ester (SGE) (Dean JV & Delaney SP., 2008). SA is likely synthesized in chloroplasts (Garcion C *et al.,* 2008 , Strawn MA*.,* 2007 Wildermuth MC *et al.,* ,2001) whereas tobacco SAGT appears to localize to the cytosol (Mohammed LA *et al*,2005). SAG is actively transported from the cytosol into the vacuole, where it may function as an inactive storage form that can be

converted back to SA (Dean JV *et al.*, 2004, Mohammed LA *et al*., 2005 Hennig J. *et al* 1993).

Jasmonic Acid is derived from the fatty acid linolenic acid and a member of the jasmonate class of plant hormones. It is biosynthesized from linolenic acid by the octadecanoid pathway. The main function of its various metabolites is regulating plant responses to abiotic and biotic stresses as well as plant growth and development. Regulated plant growth and development processes include growth inhibition, senescence, tendril coiling, flower development and leaf abscission. JA is also responsible for tuber formation in potatoes, yams, onions and converted to a variety of derivatives including esters such as methyl jasmonate; it may also be conjugated to amino acids. This chemical may have a role in pest control, according to an October 2008 BBC News report. In addition to some researchers have signed a licensing deal to market jasmonic acid as a spray to be applied to seeds prior to planting; such a spray has been found to stimulate the natural anti-pest defences of the plants.

Jasmonic Acid Biosynthesis-
Octadecanoid pathway

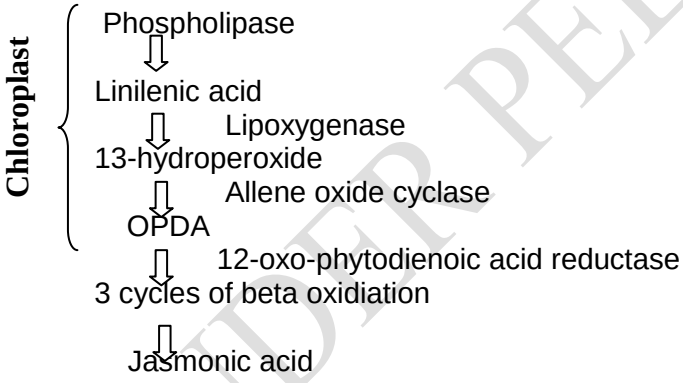
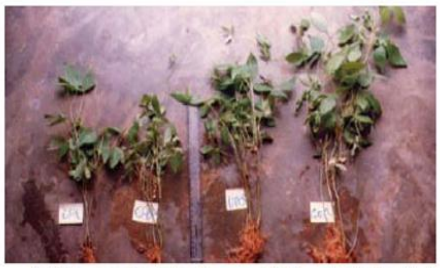


FIG.8 *Howe, G.A. 2001. PNAS 98:12315-19*

ISR relation to seed: Application of some PGPR strains to seeds or seedlings has also been found to lead to a state of induced systemic resistance (ISR) in the treated plant (Van Loon *et al*., 1998; Kloepper *et al*., 1999). ISR occurs when the plant's defence mechanisms are stimulated and primed to resist infection by pathogens (Van Loon, 1997).



A & B - control plants C,D – plants treated with panchagavya

Panchgavya Treated seedlings Control seedlings

FIG. 9 *Induced resistance through Panchagavya*

Panchagavya: An Organic formulation made up of five products of Cow Viz., Cow Dung, Urine, Milk, Curd and Ghee is known to induce systemic resistance in plants and also a wonderful bio-pesticide. Research carried out at the Department of Biotechnology, GITAM Institute of Technology proved that the formulation up-regulated certain genes of the pathogenesis pathway in plants bestowing disease resistance to the plants. Use of Panchagavya can be a sustainable alternative to chemical pesticides.

Systemic Resistance in Chickpea against Fusarium wilt:

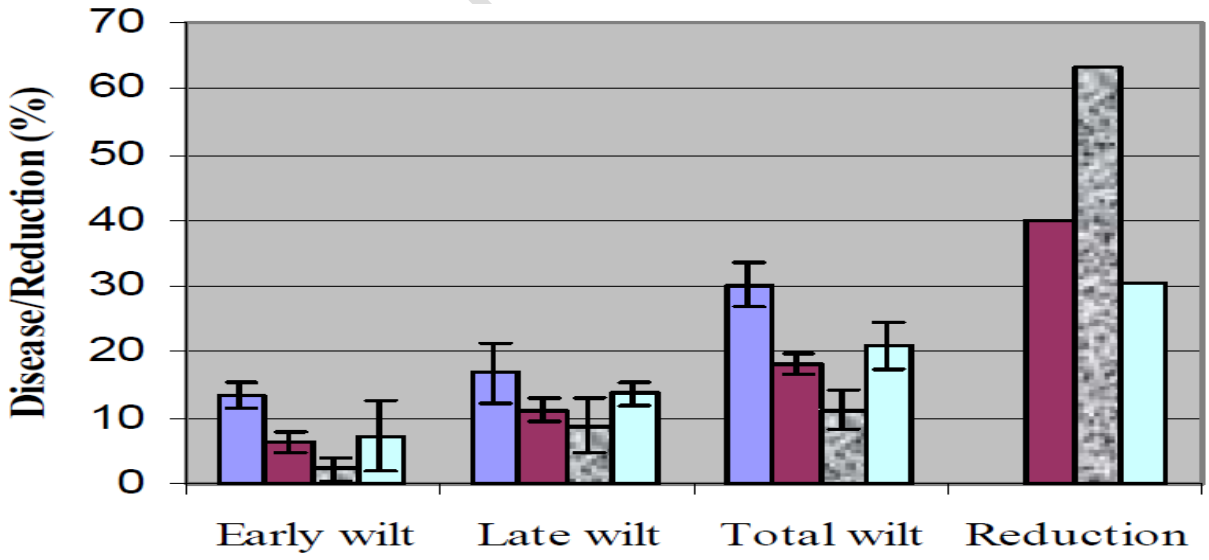


Fig.10: Effect of seed dressing with different chemicals on wilt disease incidence in chickpea grown in wilt sick field. Chickpea seeds were dressed with Salicylic acid (SA), Bezo (1,2,3)-thiadizole-7-carbothioic acid-S-methyl ester (Bion), K_2HPO_4 (Kp) and water (Con) for two hours before sowing

Fusarium wilt caused by *Fusarium oxysorum* f. sp. *ciceri* is one of the major diseases of chickpea. The fungus invades plant vascular tissues and induces severe wilting of the foliage by blocking xylem transport and impeding the movement of water (Beckman *et al*., 1989; Haq & Jamil, 1995). The pathogen is both seed and soil borne facultative saprophyte and can survive in soil up to six years in the absence of susceptible host (Haware *et al*., 1986).In resent years, the process of "immunization" or induced resistance to diseases has received increasing attention (Vallad & Goodman,2004). Induced resistance can be achieved with certain environmentally safe chemicals (Kuc, 2006). It has been proved that salicylic acid, acetyl salicylic acid and Bion haveinduced systemic resistance in chickpea against wilt disease under controlled environments (Saikia *et al*., 2003; Sarwar *et al*., 2005). Nighat sarwar *et al.,*2010 Induced systemic resistance in chickpea against wilt disease caused by *Fusarium oxysorum* f.sp. *ciceri* (FOC) was studied by treating the seeds with benzo (1,2,3)-thiadizole-7-carbothioic acid - s- methyl ester (Bion), salicylic acid (SA) and di-potassium hydrogen phosphate (K_2HPO_4). Reduction in disease was observed in both type of applications but seed dressing was found more effective than soaking method. Highest reduction i.e., 63% in wilt disease was observed with Bion dressing followed by SA, 40% and K_2HPO_4 , 30%. Bion and SA showed 41 & 24% reduction in the disease, respectively, when seeds were soaked in the respective chemicals but no reduction was found with K_2HPO_4 soaking. Slight increase in yield was observed with all the treatments in both applications but difference among them was statistically non-significant.

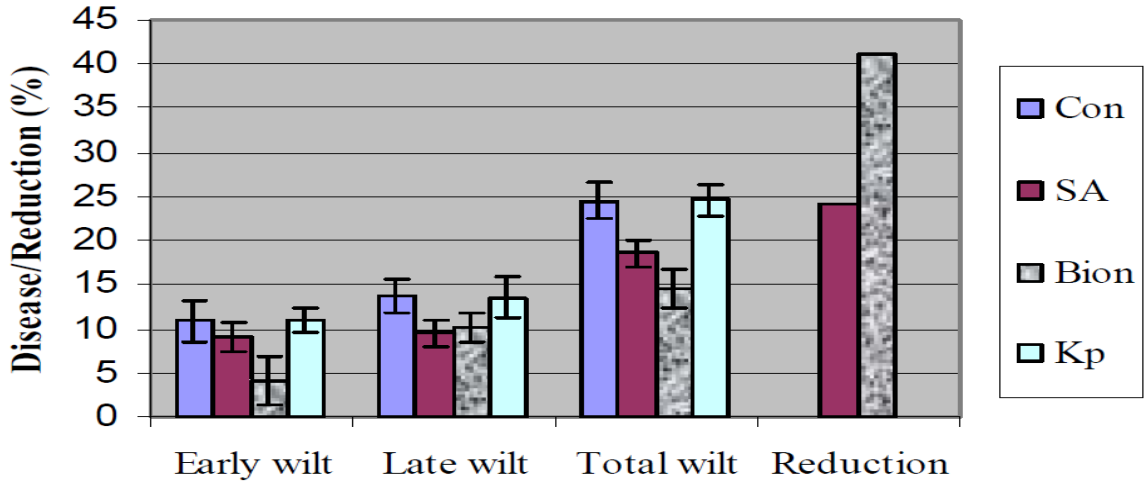


Fig.11: Effect of seed soaking with different chemicals on wilt disease incidence in chickpea grown in wilt sick field. Chickpea seeds were soaked in aqueous solutions of Salicylic acid (SA), Bezo (1,2,3)-thiadizole-7-carbothioic acid-S-methyl ester (Bion), K_2HPO_4 (Kp) and water (Con) for two hours before sowing

Hamid *et al*., 2008 conducted an experiment to study the effect of salicylic acid seed priming on growth by sand method of germination and some biochemical attributes in wheat grown under saline conditions. Wheat seeds of cv. Inqlab and S-24 were soaked in water and 100 mg L-1 salicylic acid solution for 24 hours and sown in sand salinized with 0, 50 or 100 m*M* NaCl. Pots were sprayed with ¼ strength Hoagland's nutrient solution. Growth parameters (shoot and root length, and shoot and root dry weights) were recorded and chlorophyll *a* and *b* contents ; soluble sugars (reducing and non-reducing) in the leaves were estimated from

fourteen days old seedlings. Salt stress significantly reduced all growth parameters (Table.5). However, salicylic acid treatment alleviated the adverse effect of salinity on growth. Salinity decreased the chlorophyll *a* and *b* content and chlorophyll a/b ratio in both the lines, but reduction in chlorophyll a/b ratio was recorded lower in salt tolerant wheat line S-24, which could be a useful marker for selection of salt tolerant wheat (Table.9). Salinity (NaCl) stress considerably increased the accumulation of reducing, non-reducing and total soluble sugars in the leaves of 14 days old wheat seedlings of both cultivars. Salt tolerant line S-24 accumulated higher sugar content which could also be a useful marker for salt tolerance in wheat (Table.10).

The sunflower seedlings were grown in dark conditions for 9 days. Etiolated cotyledons were transferred into Petri dishes containing 0.001 μM, 0.1 μM, 10 μM, 1000 μM SA and placed to incubation for 14 hours in the dark at room temperature; then they were incubated in light period for 3 hours. Chlorophyll, carotenoid content, protein amount and peroxidase (POD) activity in the cotyledons was examined. SA 1000 μM solution showed the toxic effect in growth considering the results of total chlorophyll, carotenoid content and protein amount. An increasing 2 fold of chlorophyll content in 10 μM SA and 3.5 fold of carotenoid content in 0.1μM SA treated cotyledons comparing to the control were observed. Protein amount increased in all concentrations except 1000 μM SA. POD activity was also stimulated in all concentrations of SA solutions. However, the clear difference in 0.001 μM SA was not seen. As a result, chlorophyll, carotenoid, protein contents and POD activity increased in exogenic SA applications.

Different induced chemicals responses on wheat

Table .5 Germination and seedling indices of wheat cultivar as affected by the interaction of PEG

PEG (%)	SA (mM)	Germination (%)	Mean germination time (d)	Vigor index	Shoot length (mm)	Root length (mm)	Shoot dry weight (mg/plant)	Root dry weight (mg/plant)	Electrolyte leakage (%)
0	0.0	92.50ab	1.42d	378.22abc	129.25a	280.75a	74.50b	27.46d	37.12f
	0.1	100.00a	1.54cd	410.50a	129.75a	280.75a	84.50a	34.13b	35.14f
	0.5	100.00a	1.34d	408.75a	128.75a	280.00a	83.83a	37.46a	35.52f
10	0.0	87.50bc	2.02b	361.57bcd	87.50d	326.75b	14.50f	25.20de	71.62b
	0.1	90.00abc	1.99b	391.72abc	93.50c	341.75b	37.83e	31.20 c	50.21d
	0.5	92.50ab	1.72c	4.6.40ab	99.00b	339.25b	41.16d	34.46ab	40.78f
20	0.0	75.00d	2.71a	295.75e	66.50g	324.25b	8.16i	24.60e	90.54a
	0.1	80.00cd	2.25b	330.23de	74.50f	334.75b	11.16h	34.40b	70.85b
	0.5	91.00ab	2.10b	356.87cd	79.00e	337.50b	13.50g	36.80ab	60.54c
In each column, means with similar letter are not significantly different (DMART, p > 0.05)									

Table .6 Effect of salicylic acid on germination of two wheat cultivars as affected by PEG.

SA (mM)	Germination (%)	Mean germination time (d)	Vigor index	Shoot length (mm)	Root length (mm)	Shoot dry weight (mg/plant)	Root dry weight (mg/plant)	Electrolyte leakage (%)
0.0	85c	2.04a	345.18cc	94.41c	309.58c	32.38c	25.73b	66.41a
01	90b	1.94a	3.77.48b	99.25b	319.08b	42.83b	33.12a	52.11b
0.5	95a	1.72b	390.67a	102.25a	328.91a	46.5a	35.30a	45.61c
In each column, means with similar letter are not significantly different (DMART, p > 0.05)								

Salicylic acid (SA), as a natural signal molecule has been shown to play important roles in regulating a number of physiological processes in plants and a common plant-produced phenolic compound known as an important signal molecule for modulating plant responses to environmental stresses (Senaratna *et al*., 2000). It is now clear that SA provides protection against a number of abiotic stresses such as heat stress in mustard seedlings (Dat *et al.,* 1998b), chilling damage in different plants (Kang and Salveit,2002; Tasgin *et al*., 2003), heavy metal stress in barley seedlings (Metwally *et al.,* 2003) and drought stress in wheat plants (Singh and Usha,2003)

Table 7. Effect of salisylic acid pre- treatment on root, shoot dry weights and root/shoot ratio of *T. aestivum* and *H. vulgare* after 8 days (g/ seedling).

	T. aestivum			***H. vulgare***		
Group	**Root(g)**	**shoot(g)**	**Root/Shoot**	**Root(g)**	**shoot(g)**	**Root/Shoot**
C	0.022	0.096	0.229	0.017	0.055	0.309
S	0.010	0.027	0.370	0.010	0.034	0.294
SAW	0.034	0.142	0.239	0.021	0.086	0.244
SAS	0.024	0.099	0.242	0.020	0.061	0.328

C=Control S=Treated with 150mM NaCl after 6 h in water SAW =Treated with 0.05 mM SA and then in water SAS= Treated with 0.05 mM SA and then in 150 mM NaCl *=Significant at P<0.5 **=Significant at P<0.1 ***=Significant at P<0.05

***Trichoderma spp*. as inducer**

Recently, several BCF, as well as some plant growth–promoting rhizobacteria (PGPR) have been shown to efficiently help plants overcome abiotic stresses, such as salinity and drought, in both field crops and trees (Adams P *et al*., 2007; Bae H *et al*., 2009; Mohamed Ha-La *et al.,*2006; Sherameti I. *et al.,* 2008; *Waller F et al*., 2005 and Yildrim E *et al.,* 2006). The ability of maize plants grown from seeds treated with *T. harzianum* to resist water deficit has been demonstrated in the field, and the enhanced deep rooting clearly contributes (Harman GE *et al.*, 2000). Moreover, in *Trichoderma* inoculated cacao seedlings, drought-induced changes such as stomatal closure and reduction of net photosynthesis were delayed under drought compared with non-inoculated plants, allowing plants to continue growing (Bae H *et al*., 2009). In maize, it has been shown that in addition to induction of carbohydrate metabolism and photosynthesis-related proteins, the stress factors in the field is water deficit. *T. harzianum* added as seed treatment (tomatoes) or as a soil treatment (*Arabidopsis*) largely improved the germination at osmotic potentials of up to 0.3 M Pa (F. Mastouri, T. Bjorkman, G. Harman, unpublished data) starch content of the leaves was higher in t*richoderma*-inoculated plants (Shoresh M *et al*., 2008). A number of other stresses are also alleviated. *T. harzianum* has recently been shown to improve resistance to heat and cold (seedlings of tomato were imbibed at 25◦Cfor 1 day, then exposed to either 10◦C or 35◦C, and then returned to 25◦C). Seedlings were much less damaged by the

temperature extremes in the presence of *T. harzianum.* (F. Mastouri, T. Birkman, G. Harman, unpublished data). *Trichoderma* also increased potassium content of plants (Yedidia I *et al*., 2005 and Yildrim E *et al.,* 2006). Salt stress is well known to reduce potassium uptake, and in several systems increasing potassium uptake ameliorated salt-induced damage (Shabala S *et al.*, 2008).

CONCLUSION :

Although not fully understood, induced resistance in plants opens new horizons in plant protection, being a promising tool for ecologically-friendly disease control and sustainable agriculture. It remains a challenge for both fundamental and applied research.

REFERENCES:

1. Adams P, *et al*., 2007. *Trichoderma harzianum* Rifai 1295-22 mediates growth promotion of crack willow (*Salix fragilis*) saplings in both clean and metal contaminated soil. *Microbial Ecol. 54*(2):306–13.
2. Alexander, D., R.M. Goodman, M. Gut-Rella, C. Glascock, K. Weyman, L. Friedrich, D. Maddox, P. Ahl-Goy, T. Luntz, E. Ward, J. Ryals, 1993. Increased tolerance to two oomycete pathogens in transgenic tobacco expressing pathogen-related protein 1a. *Proc. Natl. Acad. Sci*. USA, **90**, 7327-7331.
3. Amaral da Silva, E. A., P. E. Toorop, J. Nijsse, J. D. Bewley, and H. W. M. Hilhorst. 2005. Exogenous gibberellins inhibit coffee (*Coffea arabiga* cv. Rubi) seed germination and cause cell death in embryo. *J. Exp. Bot*. **56**(413): 1029–1038.
4. Anand, A., Z.T. Lei, L.W. Summer, K.S. Mysore, Y. Arakane, W.W. Backus, S. Muthukrishnan, 2004. Apoplastic extracts from a transgenic wheat line exhibiting lesion-mimic phenotype have multiple pathogenesis-related proteins that are antifungal. *Plant-Microbe Interact*., **17**, 1306-1317.
5. Antoniw, J.F., C.E. Ritter, W.S. Pierpoint, L.C. Van Loon, 1980. Comparison of three pathogenesis- related proteins from plants of two cultivars of tobacco infected with TMV. *J. Gen. Virol*., **47**, 79-87.
6. Ashraf, M.Y. 2007. Variation in nutritional composition and growth performance of some halophytic species grown under saline conditions. *African Journal of Range & Forage Science,* **24**: 19-23.
7. Bachmann, D., E. Rezzonico, D. Retelska, A. Chételat, S.Schaerer, R. Beffa, 1998. Improvement of potato resistance to *Phytophthora infestans* by overexpressing antifungal hydrolases. 5th International Workshop on pathogenesis-related proteins. Signalling pathways and biological activities. March 29-April 2, 1998, Aussois, France. Abstracts, P-57.
8. Bae H, Sicher RC, Kim MS, Kim S-H, Strem MD, *et al*. 2009. The beneficial endophyte *Trichoderma hamatum* isolate DIS 219b promotes growth and delays the onset of the drought response in *Theobroma cacao*. *J. Exp. Bot.* **60**:3279–95, 7-12, 2001.
9. Baltruschat H, Fodor J, Harrach BD, Niemczyk E, Barna B, *et al*. 2008. Salt tolerance of barley induced by the root endophyte *Piriformospora indica* is associated with a strong increase in antioxidants. *New Phytol.* **180**:501–10.

10. Beauverie J. Essais d'immunisation des végétaux contre les maladies cryptogamiques 1901. *CR Acad Sci Paris*. **133**: 107-110,
11. Beckman, C.H., P.A. Verdier and W.C. Mueller. 1989. A system of defence in depth provided by vascular parenchyma cells of tomato in respose to vascular infection with *Fusarium oxysporum* f. sp. *Lycopersici,* race 1. *Physiol. Mol. Plant Pathol*., **34**: 227-239.
12. Beckman, C.H., P.A. Verdier and W.C. Mueller. 1989. A system of defence in depth provided by vascular parenchyma cells of tomato in respose to vascular infection with *Fusarium oxysporum* f. sp. *Lycopersici,* race 1. *Physiol. Mol. Plant Pathol*., **34:** 227-239.
13. Bokshi AI, Morris SC, Deverall BJ. Effects of benzothiadiazole and acetylsalicylic acid on -1,3- glucanase activity and disease resistance in potato. *Plant Pathol*. **52**: 22-27, 2003.
14. Bol, J.F., H.J.M. Linthorst, B.J.C. Cornelissen, 1990. Plant pathogenesis-related proteins induced by virus infection. *Annu. Rev. Phytopathol*., **28**, 113-138.
15. Catinot J, Buchala A, Abou-Mansour E, M´etraux J-P. 2008. Salicylic acid production in response to biotic and abiotic stress depends on isochorismate in *Nicotiana benthamiana*. *FEBS Lett.* **582**:473–78
16. Chester KS. The problem of acquired physiological immunity in plants. *Quat Rev Biol*. **8:** 275-324, 1933.
17. Cohen Y 2001. The BABA story of induced resistance. *Phytoparasitica*. **29**: 375-378.
18. Cornelissen, B.J.C., R.A.M. Hooft van Huijuijnen, J.F. Bol, 1986. A tobacco mosaic virusinduced tobacco protein is homologous to the sweet tasting protein thaumatin. *Nature*, **321**, 531-532.
19. Cruickshank IAM and Mandryk M 1960. The effect of stem infestations of tobacco with Peronospora tabacina Adam on foliage reaction to blue mold. *J Aust Inst Agric Sci.* **26**: 369-372.
20. Cultivars K. Maghsoudia, M.J. Arvin, 2010. Salicylic acid and osmotic stress effects on seed germination andseedling growth of wheat (*Triticumaestivum L*.). *Plant Ecophysiology*. **2** (2010) 7-11.
21. Dann, E.K. and Deverall, BJ. 1996. 2, 6-dichloro-isonicotinic acid (INAJ induces resistance in green beans to the rnst pathogen, *Uromyces appendiculatus.* under tield conditions. *Australasian Plant Pathology*, **25**, 199-204.
22. Dann, E.K. and Deverall, BJ. 1995. Effectiveness of systemicresistance in bean against 1'oliar and soilborne pathogens as induced by biological and chemical means. *Plant Pathology*, **44**. 458-466.
23. Dat, J.F., Foyer, C.H., Scott, I.M., 1998b. Changes in salicylic acid and antioxidants during induced thermotolerance in mustard seedlings. *Plant Physiol*. 118.
24. Dat, J.F., Foyer, C.H., Scott, I.M., 1998b. Changes in salicylic acid and antioxidants during induced thermo tolerance in mustard seedlings. *Plant Physiol*. **118**, 1455-1461.
25. Datta, K., R. Velazhahan, N. Oliva, I. Ona, T. Mew, G.S. Khush, S. Muthukrishnan, S.K. Datta, 1999. Over-expression of the cloned rice thaumatin-like protein (PR-5) gene in transgenic rice plants enhances environmental friendly resistance to *Rhizoctonia solani* causing sheath blight disease. *Theor. Appl. Genetics*, **98**, 1138-1145.
26. Dean JV, Delaney SP. 2008. Metabolism of salicylic acid in wild-type, and glucose transferase mutants of *Arabidopsis thaliana*. *Physiol. Plant.* **132**:417–25.
27. Dean JV, Mills JD. 2004. Uptake of salicylic acid 2-*O*-*β*-D-glucose into soybean tonoplast vesicles by an ATP-binding cassette transporter-type mechanism. *Physiol Plant.* **120**:603–12.
28. Dean JV, Shah RP, Mohammed LA. 2003. Formation and vacuolar localization of salicylic acid glucose conjugates in soybean cell suspension cultures. *Physiol Plant.* **118**:328–36

29. Dordrecht, Kluwer. Staub, T., Ahl Goy, P. and Kessmann n.d. Chemically induced disease resistance in plants. Ciba's Technical Information. acid is a phenocopy of a genetically based mechanism governing race-specific powdery mildew resistance. *Plant Physiology*, **106**, 1269-1277.
30. Durrant WE, Dong X. 2004. Systemic acquired resistance. *Annu. Rev. Phytopathol.* **42**:185–209.
31. Edreva, A., 2004. A novel strategy for plant protection: induced resistance*. J. Cell Mol. Biol.*,**3**, 61-69.
32. Fagoaga, C., I. Rodrigo, V. Conejero, C. Hinarejos, J.J. Tuset, J. Arnau, J.A. Pina, L. Navarro, L. Peña, 2001. Increased tolerance to *Phytophthora citrophthora* in transgenic orange plants constitutively expressing a tomato pathogenesis related protein PR-5. *Mol. Breed*., **7,** 175-185.
33. Gaffney, T., L.Friederich, B. Vernooij, D. Negrotto, G. Nye, S. Uknes, E. Ward, H. Kessman and J. Ryals. 1993. Requirement of salicylic acid for the induction of systemic acquired resistance. *Science,* **261**: 754-756.
34. Garcion C, Lohman A, Lamodi`ere E, Catinot J, Buchala A, *et al*. 2008. Characterization and biological function of the *isochorismate synthase 2* gene of *Arabidopsis thaliana*. *Plant Physiol.* **147**:1279–87.
35. Garcion C M´etraux J-P. 2006. Salicylic acid. In *Plant Hormone Signaling*, **24**:229–255. Oxford: Blackwell Publishing Ltd.
36. Gau, A. E., M. Koutb, M. Piotrowski, K. Kloppstech, 2004. Accumulation of pathogenesisrelated proteins in apoplast of a susceptible cultivar of apple (*Malus domestica* cv. Elstar) after infection by *Venturia inaequalis* and constitutive expression of PR genes in the resistant cultivar Remo. Eur. *J. Plant Pathol.,* **110**, 703-711.
37. Gorlach, J., Volrath, S .. Knaufbeiter, G., Hengy, G., Beckhove, U., Kogel, K.H., Oostendorp, M., Staub, T., Ward, E., Kessmann, H. and Ryals, J. 1996. Benzothiadiazole. A novel class of inducers of systemic acquired resistance activates gene expression and disease resistance in wheat. *Plant Cell,* **8**, 629-643.
38. Gozzo F 2003. Systemic resistance in crop protection: from nature to a chemical approach. *J Agric Food Chem.* **51**: 4487-4503.
39. Hanan E. Deef,, 2007. Influence of Salicylic Acid on Stress Tolerance During Seed Germination of *Triticum aestivum* and *Hordeum vulgare* . *Advances in Biological Research* **1** (1-2): 40-48.
40. Haq, I. and F.F. Jamil. 1995. Comparison of vascular discoloration and growth of *Fusarium*
41. Harman GE. 2000. Myths and dogmas of biocontrol. Changes in perceptions derived from research on *Trichoderma harzianum* T-22. *Plant Dis.* **84**:377–93.
42. Haware, M.P., Y.L. Nene and S.B. Mathur. 1986. Survival of *Fusarium oxysporum* f. sp. *Ciceri* in soil in absence of chickpea. *Proc. Natl. seminar on Mangement of soil borne diseases of crop plants*. Tamilnadu Agricultural University, Coimbatore, Tamilnadu India, 8-10 Jan, 1986.
43. Haware, M.P., Y.L. Nene and S.B. Mathur. 1986. Survival of *Fusarium oxysporum* f. sp. *Ciceri* in soil in absence of chickpea. *Proc. Natl. seminar on Mangement of soil borne diseases of crop.*
44. Hennig J, Malamy J, Grynkiewicz G, Indulski J, Klessig DF. 1993. Interconversion of the salicylic acid signal and its glucoside in tobacco. *Plant J.* **4**:593–600.

45. Hijwegen, T. and Verhaar, M.A. 1995. Effects of cucumber genotype on the induction of resistance to powdery mildew, *Sphaerotheca fuliginea,* by 2, 6-dichloroisonicotinic acid. *Plant Pathology*, **44**, 756-762.
46. Hijwegen, T.. Verhaar, M.A. and Zadoks, lC. 1996. Resistance to *Sphaerotheca pannosa* in roses induced by 2,6- dichloroisonicotinic acid. Plant Pathology, 45,631-635. ICPN **2**: 3-32.
47. Kang, H.M., Salveit, M.E., 2002. Chilling tolerance of maize, cucumber and rice seedlings leaves and roots are differently affected by salicylic acid. *Physiology* **55**, 885-89.
48. Kawano, T., N. Sahashi, K. Takahashi, N. Uozumi, and S. Muto. 1998. Salicylic acid induced intracelular superoxide generation followed by an increase in cytosolic calcium ion in tobacco cell suspension culture: The earliest events in salicylic acid signal transduction. *Plant Cell Physiol*. **39**: 721–730.
49. Kogel. K.H., Beekhove, U., Dreschers, l, Munch, S. and Romme, Y. 1994. Acquired resistance in barley-the resistance mechanism induced by 2.6-dichloroisonicotinie acid is a phenocopy of a genetically based mechanism governing race-specific powdery mildew resistance. *Plant Physiology*, **106**, 1269-1277.
50. Kuc, J. 2006. What's old and what; new in concepts of induced systemic resistance in plants, and its applications. In: *Multigenic and Induced Resistance in Plants*. (Eds.): S.
51. Lawrence, C.B., N.P. Singh, J. Qiu, R.G. Gardner, S. Tuzun, 2000. Constitutive hydrolytic enzymes are associated with polygenic resistance of tomato to *Alternaria solani* and may function as an elicitor release mechanism. Physiol. Mol. *Plant Pathol*., **57**, 211-220.
52. Lee H-I, Raskin I. 1998. Glucosylation of salicylic acid in *Nicotiana tabacum* cv. Xanthi-nc. *Phytopathology.* **88**:692–97
53. Lee H-I, Raskin I. 1999. Purification, cloning, and expression of a pathogen inducible UDP-glucose: salicylic acid glucosyltransferase from tobacco. *J. Biol. Chem.* **274**:36637–42
54. Leslie, C.A. and R.J. Romani. 1988. Inhibition of ethylene biosynthesis by salicylic acid. *Plant Physiol*., **88**: 833-837.
55. Metraux, J.P., H. Singer, J. Ryals, E. Ward, M. Wyff-Panz, J. Gaudin, K. Rafehdorf, E. Fehmid, W. Blum and B. Inveradi. 1990. Increase in Salicylic acid at the onset of systemic acquired resistance in cucumber. *Science,* **250**: 1004-1106.
56. Metraux, lP., Ahl Goy, P., Staub, T., Speich, J., Steinemann, A., Ryals, J. and Ward, E. 1991. Induced systemic resistance in cucumber in response to 2,6-dichloro-isonicotinic acid and pathogens. *Plant Physiol*. **130**, 179-186.
57. Metwally, A., Finkimemeier, I., Georgi, M., Dietz, K.J., 2002. Salicylic acid alleviates the admium toxicity in barley seedlings. *Plant Physiol*. **132**, 272-281.
58. Metwally, A., Finkimemeier, I., Georgi, M., Dietz, K.J., 2002. Salicylic acid alleviates the cadmium toxicity in barley seedlings. *Plant Physiol*. **132**, 272-281.
59. Mohamed HA-LA, Haggag WM. 2006. Biocontrol potential of salinity tolerant mutants of *Trichoderma harzianum* against *Fusarium* oxysporum. *Braz. J. Microbiol.* **37**:181–91.
60. Mohammed LA, Fitzpatrick T. Dean JV2005. The formation, vacuolar localization, and tonoplast transport of salicylic acid glucose conjugates in tobacco cell suspension cultures. *Planta.* **221**:287–96
61. Mohsina hamid, m. Yasin ashraf*, khalil-ur-rehman and m. Arashad, 2008. Influence of salicylic acid seed priming on growth and some biochemical attributes in wheat grown under saline conditions. *Pak. J. Bot*., **40**(1): 361-367.
62. Nielsen, K.K., Bojsen, K., Collinge, D.B. and Mikkelsen, J.D. 1994. Induced resistance in sugar beet against *Cercospora beticola:* induction by dichloroisonicotinic acid is

independent of chitinase and beta-1, 3-glucanase transcript accumulation. Physiological and Molecular. *Plant Pathology*. **45**, 89-99.
63. Nighat sarwar, m. Hayat zahid ch. And ikramul haq, 2010. Seed treatments induced systemic resistance in *Fusarium oxysporum* in various segments of chickpea grown in wilt sick plot in Faisalabad, Pakistan. ICPN **2**: 3-32.
64. Ogawa, M., A. Hanada, Y. Yamauchi, A. Kuwahara, Y. Kamiya, and S. Yamaguchi. 2003. Gibberellin biosynthesis and response during Arabidopsis seed germination. The *Plant Cell*. **15**: 1591–1604.
65. Park, C.-J., J.-M. An, Y.-C. Shin, K.-J. Kim, B.-J. Lee, K.-H. Paek, 2004b. Molecular characterization of pepper germin-like protein as the novel PR-16 family of pathogenesisrelated proteins isolated during the resistance response to viral and bacterial infection. *Planta*, **219,** 797-806.
66. Pierpoint, W.S., N.P. Robinson, M.B. Leason, 1981. The pathogenesis-related proteins of tobacco: their induction by viruses in intact plants and their induction by chemicals in detached leaves. Physiol. *Plant Pathol*., **19**, 85-97.
67. Pieterse CMJ, van Wees SCM, van Pelt JA, Knoester M, Laan R, Gerrits H, Weisbeek PJ, van Loon LC 1998. A novel signaling pathway controlling induced systemic resistance in Arabidopsis. *Plant Cell*. **10**: 1571-1580.
68. Pinto, L. V. A., E. A. A. Da silva, A. C. Davide, V. A. Mendes De Jesus, P. E. Toorop, and H. W. M. Hilhorst. 2007. Mechanism and control of *Solanum lycocarpum* seed germination. *Ann. Bot.* **100**(6):1175–1187.
69. Poupard, P., L. Parisi, C. Campion, S. Ziadi, P. Simoneau, 2003. A wound- and ethephoninducible *PR-10* gene subclass from apple is differentially expressed during infection with a compatible and incompatible race of *Venturia inaequalis*. Physiol. Mol. *Plant Pathol*., **62**, 3-12.
70. Rai, V.K., S.S. Sharma and S. Sharma. 1986. Reversal of ABA-induced stomatal closure by phenolic compounds. *J. Exp. ot.,* **37**: 129-134.
71. Ray J. Les, 1901., maladies cryptogamiques des végétaux. *Rev Gen Bot.* **13**: 145-151.
72. Robert N., J. Ferran, C. Breda, P. Coutos-Thévenot, M. Boulay, D. Buffard, R. Esnault, 2001. Molecular characterization of the incompatible interactions of *Vitis vinifera* leaves with *Pseudomonas syringae* pv. *pisi*: expression of genes coding for stilbene synthase and class 10 PR protein. *Eur. J. Plant Pathol*., **107**, 249-261.
73. Ross AF. Localized acquired resistance to plant virus infection in hypersensitive hosts. *Virology*. **14**: 329-339, 1961.
74. Saikia, R., B.P. Singh, R. Kumar and D.K. Arora. 2005. Detection of pathogenesis-related proteinschitinase and β-1,3-glucanase in induced chickpea. *Current Science*, **89**: 659-663.
75. Saikia, R., T. Singh, R. Kumar, J. Srivastava, A. K Srivastava, K. Singh and D.K. Arora. 2003. Role of salicylic acid in systemic resistance induced by *Pseudomonas fluorescens* against *Fusarium oxysprum* f. sp. *Ciceri* in chickpea. *Microbiological Research*, **158**: 203-213.
76. Sarwar, N., M.H.Z.Ch, I. Haq and F.F. Jamil. 2005. Induction of systemic resistance in chickpea against *Fusarium* wilt by seed treatment with salicylic acid and Bion. *Pak. J. Bot*., **37**: 989- 995.
77. Sawada, Y., M. Aoki, K. Nakaminami, W. Mitsuhashi, K. Tatematsu, T. Kushiro, T. Koshiba, Y. Kamiya, Y. Inoue, E. Nambara, and T. Toyomasu. 2008. Phytochrome– and gibberellin–mediated regulation of abscisic acid metabolism during germination of photoblastic lettuce seeds. *Plant Physiol*. **146**: 1386–1396.

78. Schultheiss, H., C. Dechert, L. Király, J. Fodor, K. Michel, K.-H. Kogel, R. Hückelhoven 2003. Functional assessment of the pathogenesis-related protein PR-1b in barley. Plant Sci., 165, 1275-1280. *Science,* **24**: 19-23.
79. Selitrennikoff, C.P., 2001. Antifungal proteins. *Appl. Env. Microbiol*., **67**, 2883-2894.
80. Senaratna, T., Touchel, D., Bumm, E., Dixon, K., 2000. Acetyl salicylic acid induces ultiple stress tolerance in bean and tomato plants. *Plant Growth Regul*. **30**,157-161.
81. Senaratna, T., Touchel, D., Bumm, E., Dixon, K., 2000. Acetyl salicylic acid induces multiple stress tolerance in bean and tomato plants. *Plant Growth Regul*. **30**, 157-161.
82. Serap çag, gül cevahir-öz, mine sarsag and nihal gören-saglam. 2009, effect of salicylic acid on pigment, protein content and peroxidase activity in excised sunflower cotyledons. *Pak. J. Bot*.,**41**(**5**): 2297-2303.
83. Shabala S, Cuin TA. 2008. Potassium transport and plant salt tolerance. *Physiol. Plant.* **133**:651–69.
84. Sherameti I, Tripathi S, Varma A, Oelmueller R. 2008. The root-colonizing endophyte *Piriformospora indica* confers drought tolerance in *Arabidopsis* by stimulating the expression of drought stress–related genes in leaves. *Mol. Plant-Microbe Interact.* **21**:799–807.
85. Shoresh M, Harman GE. 2008. The molecular basis of shoot responses of maize seedlings to *Trichoderma harzianum* T22 inoculation of the root: a proteomic approach. *Plant Physiol.* **147**:2147–63.
86. Singh, B., Usha, K., 2003. Salicylic acid induced physiological and biochemical changes in wheat seedlings under water stress. *Plant Growth Regul*. **39**, 137-141.
87. Singh, B., Usha, K., 2003. Salicylic acid induced physiological and biochemical changes in wheat seedlings under water stress. *Plant Growth Regul.* **39**, 137-141.
88. Singh, S.K. and M. Kaur. 1980. Effect of growth regulators on podding and yield of mungbean (*Vigna radiata* L.). *Indian J. Plant Physiol.,* **23:** 366-370.
89. Song JT. 2006. Induction of a salicylic acid glucose transferase, AtSGT1, is an early disease response in *Arabidopsis thaliana*. *Mol. Cells* **22**:233–38
90. Soylu S, Baysal O, Soylu EM. Induction of disease resistance by the plant activator, acibenzolar-S methyl (ASM) against bacterial canker (Clavibacter michiganensis subsp. michiganensis) in tomato seedlings. *Plant Sci.* **165**: 1069-1076, 2003.
91. Sreenivasulu, N. , B. Usadel, A. Winter, V. Radchuk, U. Scholz, N. Stein, W. Weschke, M. Strickert, T. J. Close, M. Stitt, A. Graner, and U. Wobus. 2008. Barley grain maturation and germination: Metabolic pathway and regulatory network commonalities and differences highlighted by new MapMan/ PageMan profiling tools. *Plant Physiol.* **146**:1738–1758.
92. Strawn MA, Marr SK, Inoue K, Inada N, Zubieta C, *et al*. 2007. *Arabidopsis* isochorismate synthase functional in pathogen-induced salicylate biosynthesis exhibits properties consistent with a role in diverse stress responses. *J. Biol. Chem.* **282**:5919–33
93. Sung Y., D. J. R. Cantliffe, T. Nagata, and W. M. Nascimento. 2008. Structural changes in lettuce seed during germination at high temperature altered by genotype, seed maturation temperature, and seed priming. *J. Amer. Soc. Hort. Sci*. **133**: 167–311.
94. Tasgin, E., Atic, O., Nalbantoglu, B., 2003. Effect of salicylic acid on freezing tolerance in winter wheat leaves. *Plant Growth Regul.* **41**, 231-236.
95. Tonón, C., G. Guevara, C. Oliva, G. Daleo, 2002. Isolation of a potato acidic 39 kDa □-1,3- glucanase with antifungal activity against *Phytophthora infestans* and analysis of its expression in potato cultivars differing in their degrees of field resistance. *J. Phytopathol.*, **150**, 189-195.

96. Uppalapati SR, Ishiga Y, Wangdi T, Kunkel BN, Anand A, *et al*. 2007. The phytotoxin coronatine contributes to pathogen fitness and is required for suppression of salicylic acid accumulation in tomato inoculated with *Pseudomonas syringae* pv. *tomato* DC3000. *Mol. Plant-Microbe Interact.* **20**:955–65
97. Vallad, G.E. and R.M. Goodman. 2004. Systemic acquired resistance and induced systemic concepts and direction of induced systemic resistance in plants and its application. *Eur J Plant Pathol.* **107**: 7-12, 001.
98. Vallad, G.E. and R.M. Goodman. 2004. Systemic acquired resistance and induced systemic resistance in conventional agriculture. *Crop Science*, **44**: 1920-1934.
99. Van Loon LC, Bakker PAHM, Pieterse CMJ. 1998. Systemic resistance induced by rhizosphere bacteria. *Annu. Rev. Phytopathol.* **36**:453–83.
100. Van Loon, L.C., 1985. Pathogenesis-related proteins. *Plant Mol. Biol*., **4**, 111-116.
101. Van Loon, L.C., 1999. Occurrence and properties of plant pathogenesis-related proteins. In: Pathogenesis-related proteins in plants. Eds. S.K. Datta, S. Muthukrishnan, CRC Press LLC, Boca Raton, 1-19.
102. Van Loon, L.C., 2001. The families of pathogenesis-related proteins. *6th International Workshop on PR-proteins.* May 20-24, 2001, Spa, Belgium. Book of abstracts, p. 9.
103. Van Loon, L.C., E.A. Van Strien, 1999. The families of pathogenesis-related proteins, their activities, and comparative analysis of PR-1 type proteins. *Physiol. Mol. Plant Pathol.,* **55**, 85-97.
104. Verberne MC,Verpoorte R, Bol JF, Mercado-Blanco J, Linthorst HJM. 2000. Overproduction of salicylic acid in plants by bacterial transgenes enhances pathogen resistance. *Nat. Biotech.* **18**:779–83
105. Vernooij, B., L. Friedrich, A. Morse, R. Rest, R. Kolditz-Jawahar, E. Ward, S. Uknef, H. Kessman and J. Ryals. 1994. Salicylic acid is not the translocated signal responsible for inducing systemic acquired resistance but is required for signal transduction. *Plant Cell,* **6**: 959-965.
106. Vernooij, B., L. Friedrich, A. Morse, R. Rest, R. Kolditz-Jawahar, E. Ward, S. Uknef, H. Kessman and J. Ryals. 1994 Salicylic acid is not the translocated signal responsible for inducing systemic acquired resistance but is required for signal transduction. *Plant Cell,* **6**: 959-965.
107. Vleeshouwers, V.G.A.A., W. Van Dooijeweert, F. Govers, S. Kamoun, L.T. Colon, 2000. Does basal PR gene expression in *Solanum* species contribute to non-specific resistance to *Phytophthora infestans*? Physiol. Mol. *Plant Pathol*., **57**, 35-42.
108. Waller F, Achatz B, Baltruschat H, Fodor J, Becker K, *et al*. 2005. The endophytic fungus *Piriformospora indica* reprograms barley to salt-stress tolerance, disease resistance, and higher yield. *Proc. Natl. Acad. Sci. USA* **102**:13386–91.
109. Ward, E.R., S.J. Uknes, S.C. Williams, S.S. Dincher, D.L. Widerhold, D.C. Alexander, P. Ahl- Goy, J.P. Métraux, J.A. Ryals, 1991. Coordinate gene activity in response to agents that induce systemic acquired resistance. *Plant Cell*, **3**, 1085-1094.
110. Wildermuth MC, Dewdney J,Wu G, Ausubel FM. 2001. Isochorismate synthase is required to synthesize salicylic acid for plant defence. *Nature* **414**:562–71
111. Wildermuth MC. 2006. Variations on a theme: synthesis and modification of plant benzoic acids. *Curr. Opin. Plant Biol.* **9**:288–96
112. Wubben, J.P., C.B. Lawrence, P.J.G.M. de Wit, 1996. Differential induction of chitinase and 1,3-glucanase gene expression in tomato by *Cladosporium fulvum* and its racespecific elicitors. Physiol. Mol. *Plant Pathol*., **48**, 105-116.

113. Yasuda, M., Ishikawa, A., Jikumaru, Y., Seki, M., Umezawa, T., Asami, T., Maruyama-Nakashita, A., Kudo, T., Shinozaki, K., Yoshida, S. & Nakashita, H. Antagonistic interaction between systemic acquired resistance and the abscisic acid-mediated abiotic stress response in *Arabidopsis*. *The Plant Cell.* **20,** 1678–1692 (2008).
114. Yedidia I, Srivastva AK, Kapulnik Y, Chet I. 2001. Effect of *Trichoderma harzianum* on microelement concentrations and increased growth of cucumber plants. *Plant Soil* **235**:235–42.
115. Yildirim E, Taylor AG, Spittler TD. 2006. Ameliorative effects of biological treatments on growth ofsquash plants under salt stress. *Sci. Hortic.* **111**:1–6, 1455-1461.