FIELD EFFICACY OF RESISTANCE INDUCING CHEMICALS AGAINST TOMATO DISEASES

By

Miss. Thosar Ratna Uttam
(Reg. No. 013/50)

A Thesis submitted to the
MAHATMA PHULE KRISHI VIDYAPEETH
RAHURI – 413 722, DIST. AHMEDNAGAR
MAHARASHTRA, INDIA

in partial fulfillment of the requirements for the degree

of

DOCTOR OF PHILOSOPHY (AGRICULTURE)

in

PLANT PATHOLOGY

DEPARTMENT OF PLANT PATHOLOGY AND AGRICULTURAL MICROBIOLOGY

POST GRADUATE INSTITUTE
MAHATMA PHULE KRISHI VIDYAPEETH
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APPROVED BY

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POST GRADUATE INSTITUTE
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RAHURI – 413 722, DIST. - AHMEDNAGAR
MAHARASHTRA, INDIA.
2018

CANDIDATE'S DECLARATION

I hereby declare that this thesis or part
there of has not been submitted
by me or other person to any
other University or Institution
for a Degree or
Diploma

Place : MPKV, Rahuri

Date : / /2018 **(Ratna U. Thosar)**

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CERTIFICATE

This is to certify that the thesis entitled, "**FIELD EFFICACY OF RESISTANCE INDUCING CHEMICALS AGAINST TOMATO DISEASES"** submitted to the Faculty of Agriculture, Mahatma Phule Krishi Vidyapeeth, Rahuri Dist. Ahmednagar (M.S.) in partial fulfillment of the requirements for the degree of **DOCTOR OF PHILOSOPHY (AGRICULTURE)** in **PLANT PATHOLOGY,** embodies the results of a piece of *bona fide* research work carried out by **Miss. THOSAR RATNA UTTAM,** under my guidance and supervision and that no part of the thesis has been submitted for any other degree or diploma.

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Place: M.P.K.V., Rahuri.

Date: / /2018 (Ratna U. Thosar)

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ABBREVIATIONS

%	:	Per cent
/	:	Per
@	:	At the rate of
µg	:	Microgram
µl	:	Microlitre(s)
^{0}C	:	Degree celcius
BABA	:	β-aminobutyric acid
C.D.	:	Critical difference
CHT	:	Chitosan
cm	:	Centimeter(s)
Conc.	:	Concentration
CRD	:	Completely randomized block design
Cv.	:	Cultivar
DAT	:	Days after transplanting
et al.	:	And others
etc	:	*et cetera*
Fig.	:	Figure(s)
g	:	Gram
ha	:	Hectare(s)
hr	:	Hour
i.e.	:	That is
IR	:	Induced resistance
ISR	:	Induced systemic resistance
kg	:	Kilogram(s)
l	:	Litre(s)
M.S.	:	Maharashtra State
max.	:	Maximum
mg	:	Miligram(s)
min.	:	Minimum
ml	:	Mililiter(s)
mm	:	Milimeter(s)
mM	:	Milimole
MW	:	Meteorological week
No.	:	Number
PAL	:	Pheylalanine ammonia lyase
POD	:	Peroxidase
ppm	:	Part per million
PPO	:	Polyphenol oxidase
RBD	:	Randomised block design
RH	:	Relative humidity
RIC's	:	Resistance inducing chemicals
S.E.	:	Standard error
SA	:	Salicylic acid
SAR	:	Systemic acquired resistance
sp	:	Species
viz.,	:	Namely

ABSTRACT

FIELD EFFICACY OF RESISTANCE INDUCING CHEMICALS AGAINST TOMATO DISEASES

By

Miss. Thosar Ratna Uttam

A candidate for the degree of

DOCTOR OF PHILOSOPHY (AGRICULTURE)
in
PLANT PATHOLOGY
2018

Research Guide : Dr. K.R. Raghuwanshi

Department : Plant Pathology and Agril. Microbiology

Tomato is considered as a member of the family Solanaceae. The botanical name of tomato is *Solanum lycopersicum* L. It is a diploid plant with 2n = 24 chromosomes. Tomato is considered as the 2nd greatest significant vegetable crops in the world after potato. Over the last century, tomato (*Solanum lycopersicum* L.) as an important vegetable crop has attained a tremendous popularity due to its medicinal and nutritive value. It can be grown in most places all over the world, like growing in the field, greenhouses and net houses.

Induced systemic resistance represents a potentially attractive alternative disease management approach since SAR is known for growth promotion and disease reduction in crops. These are processes whereby the plant's own defense mechanisms are activated by biological or chemical resistance inducers. SAR is often associated with the priming, accumulation of pathogenesis-related (PR) proteins and defence against hemibiotrophic and biotrophic pathogens. Integration into existing crop management schemes or development of new crop management programs may be possible with this novel tool of induced plant defence.

In the light of above information on effective and environmentally safe methods of plant disease control, attempts were made in the present study to explore the potential use of abiotic agents to induce resistance in Tomato against diseases and to test the potential of resistance inducing chemicals for enhancing consistency and level of resistance by altering the anatomical and biochemical parameter against tomato diseases and to evaluate the efficacy of resistance inducing chemicals in the field.

Results indicated that all the tested RIC's along with their respective concentration reduced the incidence, severity and AUDPC of early blight, powdery mildew and tomato leaf curl virus disease. Among all treatments seed + seedling + spray treatment of salicylic acid was found most effective in reducing the early blight, tomato leaf curl virus disease incidence, severity and AUDPC value followed by spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. The highest reduction in powdery mildew disease incidence, severity and AUDPC was observed in seed + seedling + spray treatment of β aminobutyric acid followed by seed + seedling + spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. A trend of reduced incidence of tomato spotted wilt virus, although not significant, was also found on the salicylic acid -seed + seedling + spray treated plants.

Resistance inducing chemicals increased the yield of tomato during both season. The highest increase was observed in seed + seedling + spray treatment of salicylic acid followed by spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan.

Resistance inducing chemicals increased the thickness of epidermis cum cuticle layer and palisade mesophyll cells which was maximum for seed + seedling + spray treatment of salicylic acid concentration. Increase in thickness of epidermal layer and palisade mesophyll cell act as barrier for pathogen invasion of tomato leaves. The

critical analysis of stomatal characteristics of tomato leaves indicated that irrespective of RIC's treatments the stomatal frequency (No. of stomata/mm^2) was fairly high on lower (abaxial) leaf surface than the upper (adaxial) leaf surface for both studied season. The result also showed that different RIC's treatments have no significant effect on stomatal frequency of adaxial and abaxial surface. Relatively higher reduction in stomatal frequency was observed in salicylic acid - S+SL+SPY as compared to control. Though different RIC's have no effect on stomatal frequency, the result showed that it has a marked effect on stomatal size. The seed + seedling + spray treatment of β aminobutyric acid significantly reduces the stomatal length and width of stomata for both studied season which was followed by seed + seedling + spray salicylic acid treatment and seed + seedling + spray treatment of chitosan.

RIC's increased soluble protein content, total phenol content, phenylalanine ammonia lyase activity, peroxidase activity, polyphenol oxidase activity, chitinase activity and these were maximum for seed + seedling + spray treatment of salicylic acid @ 1.5mM concentration.

RIC's also increase the reducing sugar content, lignin content, β 1, 3 glucanase activity and these were maximum for seed + seedling + spray treatment of β–aminobutyric acid @ 15.0 mM concentration.

1. INTRODUCTION

Tomato is considered as a member of the family Solanaceae. The botanical name of tomato is *Solanum lycopersicum* L. It is a diploid plant with 2n = 24 chromosomes. Naturally, the tomato is a perennial plant, but it is cultivated annually because of having a great economical and commercial advantages.

Tomato is considered as the 2nd greatest significant vegetable crops in the world after potato (Anon., 2016). Over the last century, tomato (*Solanum lycopersicum*) as an important vegetable crop has attained a tremendous popularity due to its medicinal and nutritive value. It can be grown in most places all over the world, like growing in the field, greenhouses and net houses. The tomato crop is grown and used for both fresh market and processing and it is an adaptable crop. Over the past 25 years, the demand on tomato recorded highly data in both producing and consuming and it has grown quite rapidly. Nowadays, tomato occupied an area about 3.9 million hectares all over the world and annually about 108 million metric tonnes of tomato produced for both fresh market and processing (Bhatia *et al.*, 2004). In India it occupies an area of about 767 Mha with annual production of about 16385 MTs whereas in Maharashtra the area under Tomato cultivation is 35.5 Mha with production of 762.2 MTs and productivity is 21.5 MTs/ha during 2014-15 (Anon., 2016).

It is one of the most popular important "protective foods" because of its special nutritive value. Tomato fruits are rich in vitamin A, C, riboflavin and thiamine also having minerals like Mg, Ca, P, Fe, Na, K, Cu, S and Cl and contain fibers. Tomato is known as free in having cholesterol. Generally, the average size of tomato equal to (148 g) boasts only 35 calories. Approximately 20–50 mg of lycopene/100 g of fruit weight can be found in tomato. Lycopene is the best powerful antioxidant in the carotenoid family and it prevents humans from free radicals that

degrade many parts of the body, lycopene is all so known to protect human from cancer. Tomato is very good appetizer and its soup said to be a good remedy for patients suffering from constipation. At present, tomatoes are utilized at a higher rate in the developed countries than in the developing countries and hence it may be referred to as a luxury crop (Bhatia *et al.*, 2004).

It is one of the most versatile vegetable with wide usage in Indian culinary tradition. Tomatoes are served as salad, soup or baked, steamed, fried as well as used as sauce with various other foods. It is also used in pickle, ketchup, soup and other product. Tomato seeds contain 24 per cent oil used as salad oil and in the manufacture of margarine. Tomato has very few competitors in the value addition chain of processing. As a processing crop, it takes first rank among vegetable in the world (Thompson and Kelly, 1978). Fresh tomato rank as the leading green house produced vegetables in Europe. Italy, Spain and Greece are major suppliers of processed products to the world market (Tigchelaar, 1986). The leading producer of tomato in the world is USA followed by China, Italy, Turkey, Egypt, Spain, Romania, Brazil and Greece (Wani, 2011).

This crop is infected by several diseases *viz.* tomato leaf curl caused by tobacco, tomato and cucumber mosaic virus, bacterial wilt caused by *Pseudomonas solanacearum* and root knot caused by nematode *Meloidogyne incognita*, fungal disease early blight caused by *Alternaria solani*, late blight by *Phytophthora infestans*, powdery mildew by *Leveillula taurica*, *Fusarium* wilt by *Fusarium oxysporum* f.sp. *lycopersici*, root and collar rot by *Sclerotium rolfsii* and leaf spots caused by species of *Colletotricum, Ascochyta, Corynespora, Septoria, Cladosporium* and *Stemphylium* which causes heavy losses in yield. Among these fungal diseases, early blight caused by *Alternaria solani* (Elli. and Mart.) Jones and Grout, powdery mildew *Leveillula taurica*

(Lév.) G. Arnaud, tomato leaf curl and tomato spotted wilt disease are the most important one.

The pathogen *Alternaria* causing early blight was first noticed by Ellis and Martin in 1882 from drying leaves of tomato in New Jersey. Early blight was first recorded on tomato in Great Britain in epidemic form by Glasscock and Ware in 1944. The prevalence of this disease on tomato was first time reported in India by Butler (1918). Ramakrishnan *et al.* (1971) reported it's severity in Madras state causing 50 per cent losses. In Maharashtra state although tomatoes are grown around the year, the disease has been noticed to be quite severe during *kharif* as compared to *rabi* and *summer* crop. The incidence of early blight of tomato is most common in India and its severity limits crop yield significantly (Patel *et al.* 2011). Datar and Mayee (1981) observed 78.51 per cent fruit yield reduction due to *Alternaria* blight in Marathwada region of Maharashtra. Mathur and Shekhawat (1986) recorded losses to the extent of 50 to 80 per cent in tomato fruit yield due to early blight disease in Rajasthan. Infection of the plants can result in a complete loss of the crop as yields are reduced by the destruction of foliage and the fruits are damaged directly by the pathogen and by sun blotch on defoliated plants (Rotem, 1994). The disease progressively weakens the plant and increases its susceptibility to infection by reducing the photosynthetic leaf area and increasing the imbalance between nutrient demand in the fruits and nutrient supply from the leaves (Rowell, 1953). The disease can occur over a wide range of climatic conditions, but is most prominent in areas with heavy dew, rainfall and high relative humidity.

The disease Powdery mildew of tomato, caused by the fungus *Leveillula taurica*, was first reported in California in 1978 (Correlle *et al.*, 1988). The pathogen occurs frequently in home gardens. However, the disease can be very devastating in commercially-grown tomatoes where yield losses may exceed 50% in heavily infected fields.

The extent of loss depends on environmental conditions, date of disease onset, and effectiveness of fungicide control. Hot, dry days with an occasional rainstorm are conducive to disease development. The disease mainly affects leaves, causing yellowing, drying, necrosis and defoliation (Li, 2013).

Among virus diseases, Tomato leaf curl virus was first detected in Israel around 1930, and now it occurs in more than 30 countries around the world that grow tomatoes. Symptoms of ToLCV infection include severe stunting, reduction of leaf size, upward cupping/ curling of leaves, chlorosis on leaves and flowers, and reduction of fruit production. This virus causes significant yield losses from 90-100 per cent and it estimated that about 7 million hectares experience TLCV infection or mixed virus infections annually (Anon., 2016). The disease may cause up to 75 per cent or more reduction in fruit yield and due to its devastating nature it has become a national problem (Sastry and Singh, 1973). Saikia and Muniyappa (1989) reported that TLCV infected plants produced very few fruits when infected within 20 days after planting and resulting up to 92.3 per cent yield loss. Plants infected 35 and 50 days after transplanting resulted in 74 and 22.9 per cent yield loss, respectively.

Similarly, Tomato spotted wilt virus (TSWV) is one of the most important viruses affecting tomatoes, occasionally leading to yield losses of up to 100 per cent (Rosello *et al.* 1996). The "spotted wilt" disease of tomato was first described in Australia in 1915 and it was first reported in tomato by Brittlebank in 1919. The disease was determined to be of viral etiology in 1930 (Sherwood *et al.*, 2003). In India the virus (TSWV) was first reported on tomato in 1964 (Todd *et al.*, 1975). It is common in temperate, subtropical, and tropical regions around the world. Heavy crop losses *i.e.* 75-100 per cent in the field were reported during 1980s in tomato in Louisiana and in tomato and lettuce in Hawaii (Zitter and Daughtrey, 1989). Sevik and Arli-Sokmen (2012) reported

that TSWV caused 42.1 and 95.5 per cent reduction in yield and marketable value of tomato, respectively.

Under favourable climatic conditions the diseases occurs in severe form leading to low yield and economic losses. Several approaches have been attempted to manage the diseases. For controlling these diseases chemical control measures were followed under field conditions, but these traditional fungicides had their residues on tomato fruits and thus harmful to human health. Chemical control measures create imbalances in the microbial community, which may be unfavourable to the activity of the beneficial organisms and may also lead to the development of resistant strains of pathogen. The level of awareness for the safe and effective application of these fungicides is low among the farmers, thus creating dangers to human health and the environment. Developing resistant varieties is difficult in the absence of dominant genes and development of new races of the pathogen overcoming host resistance, thus it is difficult task to control the disease.

Hence, to employ the induced systemic resistance represents a potentially attractive alternative disease management approach since SAR is known for growth promotion and disease reduction in crops. These are processes whereby the plant's own defense mechanisms are activated by biological or chemical resistance inducers. SAR is often associated with the priming, accumulation of pathogenesis-related (PR) proteins and defence against hemibiotrophic and biotrophic pathogens (Glazebrook, 2005; Durrant and Dong, 2004). With ISR, the disease susceptibility of treated plants is reduced for several weeks and the growing parts are protected. In many plants, induced systemic resistance (ISR) stimulated by abiotic or biotic elicitors that increase the capacity of the plant to resist pathogens (Beckers and Conrath, 2007). As ISR has only protective and no curing effects, it could be necessary to combine the resistance inducer with low-dosage of conventional fungicide

application in order to stop pathogen's development during the build-up of the induced resistance.

Chemical activation of disease resistance in plants represents an additional option for growers to protect their crops from losses due to plant diseases. It may be the best option for chemical control of diseases where genetic resistance is not available or not sufficient. Against dynamic fungal pathogens with a history of adaptation of fungicides resistance or limited resistant cultivars and harmful effect of these fungicide residues on human health, the integration of this new technology of activating broad-spectrum plant defences with resistance inducing chemicals is likely to offer a more sustainable plant health management system. Plants have defense genes that need appropriate stimuli or signals to activate them. Inducing the plants for their own defense mechanisms by prior application of abiotic or biotic inducer is a novel technique for plant protection. The inducers are known to accumulate signalling molecules, leading to increased expression of defense genes encoding chitinase, β 1, 3- glucanase, peroxidase and other enzymes involved in the synthesis of Phytoalexins (M'Pinga *et al.*, 1997). Induce systemic resistance is a wide spread phenomenon that has been intensively investigated with respect to the underlying signalling pathways as well as to its potential use in plant protection. Elicited by abiotic or biotic elicitors, plants respond with a salicylic-dependent signalling cascade that leads to the systemic expression of a broad spectrum and long lasting disease resistance that is efficient against fungi, bacteria and viruses. There are indications from other pathosystems that such adaptation of pathogens to genetically resistant cultivars can indeed be slowed down by plant activators. A synthetic resistance activator must fit the criteria concerning environmental and toxicological safety and reliability under practical conditions and it must be commercially interesting for agrochemical producer, farmer and supplier. Integration into existing crop management schemes or

development of new crop management programs may be possible with this novel tool of induced plant defence. The effect of SAR can be long lasting and recent reports indicate that the memory of SAR even can be inherited to the next generation (Luna *et al.*, 2012; Slaughter *et al.*, 2012).

In the light of above information on effective and environmentally safe methods of plant disease control, attempts were made in the present study to explore the potential use of abiotic agents to induce resistance in Tomato against diseases and to test the potential of resistance inducing chemicals for enhancing consistency and level of resistance against Tomato diseases and to evaluate the efficacy of resistance inducing chemicals in the field with following objectives.

1. To study the efficacy of resistance inducing chemicals in tomato plant against tomato diseases.
2. To study the anatomical changes at cellular level due to resistance inducing chemicals.
3. To study the quantitative biochemical changes in host plant due to resistance inducing chemicals.
4. To study the quantitative changes in enzymes of phenolic synthesis pathways.
5. To study the pathogenesis related proteins *viz*. β-1, 3-glucanase and chitinase, as affected by RIC.

2. REVIEW OF LITERATURE

Tomato is the most popular vegetable in the world because of its taste, colour and high nutritive value and also for its diversified use. There are many factors involved in low yield of tomato; among them are infestations by fungi, bacteria, nematodes or viruses and the competing weeds are predominant. The most urgent need is to develop eco friendly control measures that can resist the ravage of important diseases like early blight, powdery mildew, tomato leaf curl disease, tomato spotted wilt virus because, crop rotation, breeding for resistant plant varieties and application of pesticides are insufficient to control. Since the phenomenon of SAR was described and phrased in the 1960s (Ross, 1961; Chester, 1933), intense research has been conducted to understand which components that are involved in SAR activation and signal transmission. Most of the questions still need to be answered, but recent progress has been made. Several articles have been published to summarize the latest findings and address the fundamental questions regarding SAR. Hence earlier research reports relevant to present attempt are collected and presented here.

2.1 The pathogen

2.1.1 *Alternaria*

The pathogen *Alternaria* plays a major role in causing leaf blight in tomato crop. Two species *viz., Alternaria solani* (Ellis and Martin, 1882), Jones and Grout, 1897) and *A. alternata* (Fries) Kessler (*A. tenuis*) have been observed to be an important pathogens on tomato from all over India. A stem canker disease of tomato caused by *A. alternata* f. sp. *lycopersici* is also reported by Gragon *et al.* (1975).

The causal organism of early blight of tomato was described by Ellis and Martin (1882) for the first time from the drying leaves of tomato collected at New Jersey and named it as *Macrosporium solani*. In the year 1896-97, they described this pathogen critically and proposed new combination of nomenclature. Jones and Grout (1897) transferred it

into the genus *Alternaria* because in one of their culture, spores were found in chains of two. Butler (1903) reported this pathogen for the first time in India on potatoes from Farukabad district of Uttar Pradesh.

Ellis and Gibson (1974) recorded in CMI description that *A. solani* may persist in infected debris in soil up to 3 years under temperate conditions and is known to be seed borne pathogen in tomato.

Patterson (1991) stated that chlamydospores of *A. solani* remained in soil along with tomato residue. These were responsible as a source of primary soil borne inoculum for infection of collar rot and early blight of tomato.

Alternaria has the ability to grow over a wide range of temperatures from 4 to 36°C (Pound, 1951) and requires only a short wet period of at least four hours for successful infection (Vloutoglou and Kalogerakis, 2000).

Typically, weakened plant tissues, either due to stress, senescence, or wounding, are more susceptible to *Alternaria* infection than healthy tissues (Thomma, 2003).

2.1.2 *Leveilleula taurica*

Powdery mildew of Tomato was caused by *Leveillula taurica*, the incomplete stage of *Oidiopsis taurica* (Correlle *et al.*, 1988).

Powdery mildew of tomato, caused by the fungus *Leveillula taurica*, occurs infrequently in home gardens. However, the disease can be very devastating in commercially-grown tomatoes where yield losses may exceed 50 per cent in heavily infected fields. The extent of loss depends on environmental conditions, date of disease onset, and effectiveness of fungicide control. Hot, dry days with an occasional rainstorm are conducive to disease development (Thomson and Ockey, 1994).

Tomato powdery mildew caused by three pathogens worldwide *viz. Leveillula taurica* (*Oidiopsis taurica*) is a pathogen of a wide range of host species in warm arid to semiarid climates in Asia, the

Mediterranean, Africa, and more recently the southwest United States; *Erysiphe orontii* (*E. cichoracearum* and *E. polyphaga*) is another species common to many host plants in both temperate and tropical regions. *Odium lycopersicum* reported in eastern North America (Anon., 1998).

The disease Powdery mildew of Tomato, caused by *Leveillula taurica* (Lév.) G. Arnaud, is a major problem in several countries and causes heavy losses in yield (Ilhe *et al.*, 2008, Aydin and Gore, 2010).

Schultz and French (2011) stated that the fungus was survived between crops as mycelium in volunteer hosts and weeds. Conidia (asexual spores) were easily dislodged by wind and rain, and carried long distances. The disease was favoured by high humidity, high density stand and excess nitrogen fertilization contribute. Conidia were also spread within the same crop by insects (thrips, aphids and whiteflies).

The genus *Leveillula* is distributed in warm, arid areas of Africa, Asia, South America, southern Europe, and the western parts of North America. Species within the genus are adapted to xerophytic conditions, exemplified by the ability of their conidia to germinate rapidly and at any relative humidity (Braun and Cook, 2012).

2.1.3 Tomato Leaf curl Virus

In all reported viral diseases in tomato, tomato leaf curl virus (ToLCV), a geminivirus (Geminiviridae : subgroup-III), is the most important and destructive viral pathogen in many parts of India and can reach up to 100 per cent incidence of disease with yield losses often exceeding 90 per cent, the disease first reported in the Sudan (Cowland, 1932; Saikia and Muniyappa, 1989; Harrison *et al.*, 1991). In India, it was first reported by Vasudeva and Sam Raj (1948) from Delhi.

Chavan *et al.* (2013) reported that Indian ToLCV isolates are mostly monopartite (DNA-A) in nature with few isolates possessing bipartite (DNA-A and DNA-B) genome organization. Both DNA-A and

DNA-B are single-stranded (ss) DNA genomes of approximately 2.7 kb size.

Mishra *et al.* (2014a) stated that of all the disease reported in tomato, the tomato leaf curl virus (ToLCV) disease is cited to be the most devastating both in terms of quantitative and qualitative yield losses. Often, the loss reaches to the extent of 100% during summer throughout India

2.1.4 Tomato spotted wilt virus

The TSWV disease caused by the TSWV was first observed in 1906 (Sakimura, 1962). Brittlebank (1919) published the first description of TSWV disease detected on tomatoes in 1915 in the state of Victoria (Australia) and he called it "spotted wilt of tomato". The first characterization of this virus as the causal agent of the disease was reported by Samuel *et al.* (1930), who gave it its current name "Tomato spotted wilt virus".

Osborn (1919) also observed this disease on tomatoes in the south of Australia and in 1920 its presence was reported in all the Australian states (Best, 1968).

The TSWV reported from many parts of the world and causes about 75 to 100 per cent losses in tomato yield, if the infection starts 10 to 30 days after transplanting, the yield loss will be 100 per cent (Rosello *et al.*, 1996 and Anon., 1997).

Tomato spotted wilt virus (TSWV)was found in almost all continents and was the type species of the plant viruses belonging to the genus Tospovirus within the arthropod-borne Bunyaviridae family (Van Regenmortel *et al.*, 2000).

Kulshrestha *et al.* (2013) stated that TSWV disease was reported from several tropical and temperate regions and it was considered worldwide in distribution.

2.2 Symptoms induced by pathogen

2.2.1 *Alternaria species*

Borksdale and Stoner (1973) observed the syndrome consisting of leaf spotting that leads to defoliation, stem cankers occasionally leading to stem and fruit rot caused by *Alternaria solani* in tomato.

Ellis and Gibson (1974) described the symptoms on foliage as small, dark, circular lesions, brown to dark brown becoming distinctly zonate at advance stages. Conidia are formed on necrotic lesions, later the spots become light coloured in centre. Under severe conditions the spot may enlarge rapidly, coalescing to give complete blighting and wilt condition. Stem lesion were roughly circular, sunken dark and zonate.

Wilson (1943) and Basu (1974) reported that floral infection and black rot of fruit of tomato are caused by *Alternaria solani*. The disease appears throughout the fruit growth and affects quality of fruits.

Waraitch *et al.* (1975) recorded that the pathogen *A. solani* attacks the plants at all its growth stages. The disease on leaves, stem and fruits under favourable condition result in defoliation, drying of twigs and premature drop, depending upon its severity.

Coffey *et al.* (1975) studied the effect of early blight disease caused by *A. solani* on shoot growth of young tomato plants. The changes in growth were related to the severity of infection with increasing inoculums which leads to premature leaf fall. Healthy leaves expanding soon after inoculation were markedly affected by the disease.

Gragon *et al.* (1975) reported that *Alternaria alternata* f.sp. *lycopersici* was shown to be the cause of a serious stem canker disease of tomatoes. The fungus was a distinct pathotype capable of primary infection of leaves, stem and fruit of susceptible tomato cultivars.

Pearson and Hall (1975) studied that free moisture as dew deposition was essential for rapid germination of conidia in the absence of rainfall. However water soluble nutrient *viz.* glucose and fructose on

the fruit surface dissolved in the dew stimulates the germination of conidia at 6-15^{0}C, causes infection and symptom development under field conditions. But these conditions were not required for disease development under greenhouse conditions. Inoculation of ripe fruits frequently resulted in large sunken lesions whereas, inoculations of green fruit resulted in quiescent lesions that failed to enlarge after the fruit ripened. Sporulation was optimum at 27^{0}C and was inhibited below 15^{0}C and above 33^{0}C.

Reda *et al.* (1986) studied the response of three cultivars of tomato against *Alternaria solani* and showed fruits of redstone, Roma VF and marmande early were susceptible to rot caused by this pathogen. Plants and fruits of redstone were most susceptible. Diseased fruits weighed only half as much as healthy one, especially during the riped stage in Egypt.

Gleason *et al.* (1993) observed first initiation of symptoms on older leaves after fruit setting. Early blight caused dark brown leaf spot with concentric rings on tomato by *A. solani*.

2.2.2 *Leveillula taurica*

The most common symptoms of tomato powdery mildew are yellow and necrotic spots on the upper leaf surface and powdery white spots on the lower side of the leaves (Holliday, 1980).

Thomson *et al* (1994) reported that the first symptoms occur on lower leaves as bright yellow spots that range from 1/8 to 1/2 inch in diameter. The spots enlarged and eventually turn brown. With progress in infections, the entire leaf withers and dies but remains attached to the stem. No symptoms were observed on the stems or fruit. However, with extensive loss of foliage, many exposed fruit were sunburn. Heavy sporulation of the fungus on leaves was rarely observed in Utah.

In addition, The commonly observed symptoms of powdery mildew disease caused by *L. taurica* as light green to yellowish spots or blotches on the upper tomato leaf surface. While on the lower surface,

fine whitish grey to purplish growth of the causal agent arises from the lower stomata. Later, infected areas of leaves dry up and turn into dark brown. Finally, the leaves may defoliate. Generally, the symptoms are considerably more visible on older leaves than on younger ones (Correlle *et al.*, 1988; Haroun, 2002; McGrath *et al.*, 2001 and Reis *et al.*, 2004). On the other hand, this disease does not infect the stem or fruit but causes general declines in growth, chief foliage damage, reduce the quality of fruits and finally, it sharply decreases tomato yield (Natour *et al.*, 1971; Schickedanz, 1989; Jones and Thomson, 1987).

2.2.3 Tomato leaf curl virus

Vasudeva and Sam Raj (1948) reported that Tomato Leaf Curl Virus Disease (ToLCV) was characterized by the curling and twisting of leaves followed by marked reduction in leaf size. The diseased plants looks pale and stunted due to shortening of inter nodal length with more lateral branches resulting in a bushy appearance.

Gaikwad *et al.* (2011) studied the occurrence, detection and molecular characterization of leaf curl virus infecting tomato, under open and net house conditions and recorded the symptoms include leaf curling, yellowing, purpling, blistering, puckering, malformation, vein clearing, vein thickening and/or stunting of plants. The most occurring symptoms were puckering and malformed leaves followed by typical leaf curl type symptoms.

2.2.4 Tomato spotted wilt virus

On tomatoes, plants show bronzing, curling, necrotic streaks and spots on the leaves. Dark-brown streaks also appear on leaf petioles, stems and growing tips. The plants are small and stunted as compared with healthy plants. The ripe fruit shows paler red or yellow areas on the skin. Sometimes, affected plants are killed by severe necrosis (Anon., 1997).

Sherf and MacNab (1986) reported that the first symptoms of TSWV infection on tomato were small, orange-coloured flecks on some

middle or lower leaves or on the calyx. However, as new spots appear, older leaves turn brown, die, and droop. Similar spots or streaks occurred on the stems and petioles. The entire plant becomes dwarfed, and with its drooping leaves it resembles a plant affected by a wilt. On the green fruits, yellowish spots up to 10 mm in diameter usually with distinct concentric zones of shades of yellow or brown alternating with green and later with pink or red were appeared. These zoned fruit spots were the most striking symptom of spotted wilt on red tomato fruits.

2.3 Induced systemic resistance (ISR) : The phenomenon

Gaffney *et al.* (1993) stated that systemic acquired resistance was thought to be activated by pathogens causing cell death reactions, ranging from single-cell hyper sensitive reactions (HR), a plant specific type of programmed cell death, (PCD) to necrotic disease lesions.

Kuc (1995) stated that in incompatible reaction between plants and the pathogen, pathogen will elicit a set of localized responses in and around the infected host cell which leads to cell death, thus the pathogen may be trapped in dead cells and appears to be preventing from spreading from the site of initial infection. Further local responses in the surrounding cells include changes in cell wall composition that can inhibit penetration by the pathogen and *de novo* synthesis of antimicrobial compounds such as phytoalexins.

Schneider *et al.* (1996) stated that systemic resistance, phenotypically, was manifested as a protection of the plant not only against the attacking pathogen but also other types of pathogen. Although it posses some specificity, the resistance seems to be rather non specific and long lasting.

Kuc (2001) stated that resistance to plant disease was often specific and metabolites and receptors contributing to this specificity may have specific structures. However, simple, structurally-unrelated compounds induce systemic resistance in unrelated plants to diverse pathogens including fungi, bacteria and viruses. Both resistance and

induced systemic resistance (ISR) were associated with the rapid accumulation of the same structurally unrelated putative defence compounds that have diverse functions. These compounds could not be released or synthesized by a plant until an appropriate signal had been received for its synthesis or release.

Heil and Bostock (2002) reported that ISR of plants against pathogen was a widespread phenomenon elicited by a local infection. Plants respond with a salicylic dependant signaling cascade that leads to the systemic expression of a broad spectrum and long lasting disease resistance that was efficient against fungi, bacteria and viruses. Changes in cell wall composition, *de novo* production of pathogenesis related proteins such as chitinases and glucanases and synthesis of phytoalexins were associated with resistance.

Edreva (2004) defined an increased expression of natural defense mechanisms of plants against various type of pathogens, provoked by a range of factors: pathogens causing hypersensitive necrotic reaction, avirulent or attenuated pathogenic strains, elicitors of pathogenic origin (glucans, proteins, lipids *etc.*), abiotic elicitors, including synthetic harmless chemical products such as 2, 6-dichloroisonicotinic acid (INA), β-aminobutyric acid (BABA), benzothiadiazole (BTH) *etc*.

Vallad and Goodman (2004) stated that systemic acquired resistance (SAR) and induced systemic resistance (ISR) were two forms of induced resistance. In both SAR and ISR plant defenses were preconditioned by prior infection or treatment that results in resistance or tolerance against subsequent challenge by a pathogen or parasite. These defenses could be actively expressed in response to pathogen and parasite of various scales ranging from microscopic virus to insect herbivores.

Kogal and Langen (2005) stated that disease resistance strategies reduced the chemical input into the environment and are

therefore powerful approaches to sustainable agriculture. Induced resistance (IR) was emerged as a potential alternative or a complementary strategy for crop protection. IR signifies the control of pathogen and pests by prior activation of plant defense pathways.

2.4 Efficacy of different resistance inducing chemicals in the plant against the pathogen

2.4.1 Salicylic acid (SA)

Exogenous application of SA induced the resistance against TMV and PR-protein accumulation in tobacco that accompanied increased resistance (Van Loon *et al.* 1998). Conversely, some workers suggested that SA may have not translocated primary signal for SAR, but may only play a regulatory role in the expression of SAR genes (Vernooij *et al.*, 1994).

Exogenous application of salicylic acid or structural analogues of SA such as benzo (1,2,3) thiadiazole-carbothioic-acid-S-methylester (BTH) and 2,6-dichloro isonicotinic acid (INA) that appear to act similarly to salicylic acid can induce SAR (Hammerschmidt, 1999).

Spletzer and Enyedi (1999) investigated the effect of exogenous application of salicylic acid (SA) against *Alternaria solani* in tomato leaves and it was found that the addition of 200 µM SA to the root system significantly increased the endogenous SA content of leaves. Challenge inoculation of SA-treated tomato plants using conidia of *A. solani* resulted in 83% fewer lesions per leaf and a 77% reduction in blighted leaf area as compared with control. Also the expression of PR-1B gene was strongly induced within 24 hr of the addition of 200 µM SA. Hence it was indicated that root feeding 200 µM SA to tomato plants significantly elevate foliar SA levels, induce PR-1B gene expression and activate SAR that was effective against *A. solani*.

Salicylic acid (SA) is phenolics in nature that affects a variety of biochemical and molecular events associated with induction and

expression of disease resistance controlled by major genes (Hammerschmidt *et al.*, 2000).

Ju *et al.* (2002) studied the effect of seedling treatment of salicylic acid (SA) on *Pseudoperonospora cubensis* resistant development in cucumber and stated that the challenge inoculation of *Pseudoperonospora cubensis* 4 days after treatment reduced the disease index of the first and the second leaves by 8.9 and 22.1 per cent, respectively.

Cheng-bo and Hua-zhi (2005) tested the effect of 9 chemical compounds for the induction of resistance against *Curvularia lunata* in maize at different growth stages. The results showed that all the chemical compounds could reduce the disease by a range of 9.8-49.6 per cent, but SA was the most effective compound which reduced the disease severity by up to 49.6 per cent relative to control. Also the resistance induction in less susceptible cultivars was more significant than in more susceptible cultivars and the resistance induction was more significant at seedling stage than at jointing stage or ripening stage.

Wang *et al.* (2005) reported that SA (1 to 5 mM) induced pathogenesis-related (PR) genes expression led to acquired resistance against a variety of microbial pathogens, and even the lower concentrations of SA (10 to 50 µM) were also found effective.

Madhusudhan *et al.* (2005) reported the effect of seed and seedling treatment of salicylic acid (50 mM), neem oil (5 %) and *Pseudomonas fluorescens* (slurry) on the concentration of *Tomato mosaic tobamovirus* (ToMV) and *Tobacco mosaic tobamovirus* (TMV) in tomato and bell paper. The concentration was assessed based on the number of local lesions on *Nicotiana glutinosa.* The results showed that the SA was an effective inducer in reducing the number of local lesions and concentration of *tobamovirus*.

Tosun *et al.* (2006) applied the plant disease resistance elicitors, salicylic acid (SA) and $Harpin_{Ea}$ alone and in combination with

systemic fungicide phosphorus acid (Agriphos 400) to tomato plants, which were inoculated with *Phytophthora infestans* after treatments. The average disease reduction was about 47 per cent after treatment with SA and 55 per cent with $Harpin_{Ea}$.

Awadalla (2008) investigated the effect of five antioxidants(citric acid, salicylic acid, benzoic acid, ascorbic acid, and sodium citrate) on the resistance of tomato plants (*Lycopersicon esculentum* Mill) to early blight disease incited by *Alternaria solani in vitro* and *in vivo*. The results indicated that all of the antioxidants tested markedly inhibited the mycelial growth of *A. solani in vitro*. The degree of inhibition was directly proportional with the antioxidant concentrations. The mycelial growth was greatly inhibited at the highest concentration (10.0 mM) of all antioxidants tested. Salicylic acid, ascorbic acid, and citric acid were more effective throughout the concentration ranges in inhibiting the mycelial growth than sodium citrate and benzoic acid respectively. Soaking tomato seeds in the antioxidants prior to sowing markedly increased tomato resistance to early blight disease by *A. solani*. All antioxidantse employed at different concentrations, caused a marked reduction in the appearance of early blight disease on tomato plants and the disease incidence was completely inhibited at the highest concentrations of every antioxidants tested. Salicylic acid, ascorbic acid, and citric acid were the most effective in controlling the disease; followed by sodium citrate, and benzoic acid. Also data indicated that the increasing in stem average length and leaves number per plant was corresponded with the reduction of disease severity.

Esmailzadeh *et al.* (2008) investigated the effect of foliar applications of salicylic acid (SA) with two concentrations 200 µM and 400 µM against tomato stem canker caused by *Alternaria alternata*. The application of SA (400 µM) significantly reduced the disease index as compared with infected control. SA pretreated plants were identified with higher number single cell necrosis (HR), but lower blighted areas and

discolourations as compared to non-treated control. The results of HPLC analysis indicated that there was highly correlation between increasing of the endogenous level of free SA in treated plants and reduction of the blighted leaf areas which results in systemic resistance induction.

Farouk *et al*. (2008) stated that exogenous foliar application of chitosan (CHT) at 0.05 %, salicylic acid (SA) at 100mg/l and methyl jasmonate (MeJA) were effective in reducing downy mildew (caused by *Pseudoperenospora cubensis*) occurrence and severity in cucumber plants under field conditions as well as improving its growth and yield in addidtion to decrease of the fungicide used.

Hadi and Baladi (2010) evaluated the effects of salicylic acid (SA) in Marfona potato cultivar for reducing the pathogenesis of *Rizoctonia solani* under greenhouse conditions. They treated the seedlings with 0 (control), 0.1, 0.2, 0.5, 1, 2 and 4 mM of salicylic acid every week. The results showed that treatment with 0.2 mM salicylic acid resulted in 73 per cent reduction in the infection symptoms on the potato tubers. Also, the number of potato tubers was increased by the application of 2 mM salicylic acid to plants that had been infected with the pathogen.

Atia and Ahmed (2011) investigated the effect of different concentration of salicylic acid and Chitosan on controlling of tomato early blight disease. The result showed that *in vitro* experiments, Chitosan and salicylic acid (SA) 4 % were the effective concentrations on reducing growth of *A. solani* (84.44 and 67.77 %) and reducing spores germination (55.31 and 81.56 %). However, all the tested concentrations of Chitosan (1, 2 and 4 g/l) and SA (0.1, 0.2, 0.5, 1, 2 and 4 g/l) as inducer of resistance in tomato plants, which reduced number and diameter of lesion, percentage of disease and increase percentage of protection 7 days after treatment using detached and intact leaf techniques.

Agamy *et al*. (2013) evaluated the ability of salicylic acid (SA) and the newly prepared bioactive matter "Agrileen" (AG) to suppress the leaf spot disease of tomato caused by *A. tenuissima*. The result showed that exogenous application of SA (0.5 and 1.0 mM) or AG (2.5 and 5 %) enhanced the growth and plant yield of tomato in addition to the reduction of infection with *A. tenuissima*. Also the photosynthetic pigments, total proteins, free proline and the relative water content of leaves were also enhanced when either SA or AG was applied. The protein pattern showed an increase in band intensity in plants treated with SA that express the increase in gene expression involved in resistance against stress conditions. Hence, the results confirmed that the application of SA or AG could protect tomato plants against *A. tenuissima* infection either through the direct strength of the defense system and a reduction of the severity of the pathogen.

Abada and Ahmed (2014) evaluated the effect of combination between bioagents and antioxidants on management of tomato powdery mildew. The result showed that in plastic house experiments, spraying of the bioagents *B. thuringienesis* and *T. harzianum* and the IRC salicylic acid, each alone or in different combinations, resulted in significant reduction to the severity of the disease with significant increase to the produce fruit yield. Furthermore, spraying any of these compounds alone was of less effect in this regard compared with spraying their combinations.

Jabnoun-Khiareddine *et al.* (2015) assessed two resistance inducers (RIs), Chitosan and salicylic acid *in vitro* for their antifungal activity against ten tomato phytopathogenic fungi *i.e. Fusarium oxysporum* f.sp. *lycopersici, Fusarium oxysporum* f.sp. *redicis-lycopersici, F. solani, Verticillium dahlia, Rhizoctonia solani, Collitotrichum coccodes, Pythium aphanidermatum, Sclerotinia sclerotiorum, Botrytis cinerea* and *Alternaria solani.* Results from this investigation showed that SA and

Chitosan may be used as potential inducers of systemic acquired resistance for successfully controlling fungal tomato diseases in Tunisia.

Kumar *et al.* (2015) reported that the exogenously applied SA first accumulates in host tissues and induces resistance; however, endogenous SA accumulation in plant tissue is considered to be involved in induced systemic resistance to early blight disease of tomato caused by *A. alternata*.

López-Gresa *et al.* (2016) studied the role of SA in the basal resistance of tomato plants. They have observed that the NahG tomato plants were unable to accumulate SA, hypersusceptible to CEVd and TSWV, presenting an earlier accumulation of the pathogens, which was accompanied by an early and more intense accumulation of hydroxycinnamic amides (*p*-coumaroyltyramine, feruloyltyramine, caffeoylputrescine, and feruloylputrescine), ethylene and the defence-related proteins basic PR1 and P23. In addition, BTH treatments completely or partially rescue the hypersusceptibility phenotype of NahG plants. The results clearly indicated that SA is an important component of basal resistance of tomato plants.

Ong and Cruz (2016) studied the effect of SA treatment on the severity of leaf curl disease of tomato (*Solanum lycopersicum* L.) under screen house conditions in two experimental trials from 2012 to 2013. The study sought to determine the concentration of SA applied at different time of induction which can effectively reduce the severity of the disease. Healthy seedlings of susceptible tomato variety, Apollo White were treated by spraying with 50, 250 or 500µM SA at 5, 10 or 15 days before inoculation (dbi). At induction time of 5 dbi, treatment with 250µM SA had lowest leaf curl infection compared with the untreated control, while at 10 and 15 dbi, leaf curl infection was lowest with treatment of 50µM SA. Likewise, treatment with 50µM SA regardless of induction time had consistently delayed and reduced the severity of leaf curl disease. Generally, plants treated with 50µM SA had reduced

amount of disease (AUDPC values), lower symptom severity score and lower disease index (DI) than the untreated control. The severity of the disease was also reduced with 250 and 500 µM SA treatment but the effect was more consistent with 50µM.

2.4.2 β-amino butyric acid (BABA)

Papavizas and Davey (1963) and Papavizas (1964) reported the role of BABA in the protection of pea plants against the oomycete pathogen *Aphanomyces euteiches*. They demonstrated that from 10 amino compounds and related substances tested in the greenhouse against root rot of peas, only DL-β-aminobutyric acid and DL-methyl-β-aspartic acid effectively reduced the root rot severity. The substances showed the highest activity when applied as a soil drench three days prior to inoculation with *Aphanomyces* at a concentration of 100 ppm in the soil. It was claimed that the two amino acids did not act directly on the pathogen but prevented expression of disease symptoms.

Arici and Dehne (2007) evaluated the effect of chemical inducers BION and BABA against *Phytophthora infestans* in tomato plants for induction of resistance and stated that the physiological activities in the plants was affected and changed. *In vivo* test with BION showed reduction in necrotic symptoms on leaves of tomatoes caused by *Phytophthora infestans* and complete inhibition of symptom expression on the upper younger leaves as compared to the necrotic control plants. Similar results were also observed for BABA. However, BABA produced the toxic effect on the leaves of the variety Balkonstar, but had no inhibitory effect on the growth of the plant. *In vitro* test showed that BABA with 10000 ppm and BION with 1000 ppm reduced about 10-15 % of the mycelial growth to V-8 juice medium as compared to the control. Thus it indicates that the application of both BTH and BABA weakened the pathogen virulence, which was in correlation with a plant defense mechanism reducing and preventing the disease expression.

Cohen (1994) and Cohen *et al.* (1994) ruled out the possibility of a direct toxicity of BABA on plant pathogens, since no toxic effects have ever been observed.

Cohen and Gisi (1994) studied an *in vivo* toxicity based action of metabolites of BABA and reported no probable action since experiments using 14C-labeled BABA clearly demonstrated that the substance does not undergo any changes while in the plant as shown for tomato.

Zimmerli *et al.* (2000) stated the mode of action of BABA, that it does not directly activate the plants defense arsenal but rather conditions the plant for a faster and stronger activation of stress-specific defense mechanism upon exposure to stress.

Jakab *et al.* (2001) presented an overview of BABA – induced protection in various pathosystems and stated that the protective effect of BABA is due to a potentiation of natural defense mechanisms against biotic and abiotic stresses.

The xenobiotic amino acid β-amino butyric acid (BABA) has emerged as a priming agent with an exceptionally wide range of effectiveness against tomato diseases, including biotrophic and necrotrophic pathogens (Cohen, 2002).

BABA also has been shown to directly inhibit some of these pathogens. Furthermore, depending on the plant species and the concentration used, BABA has also been shown to cause growth defects in plants (Jakab *et al.*, 2001). Plants have been shown to activate an array of defence responses ranging from induction of reactive oxygen species to production of secondary metabolites in response to BABA treatment (Justyna and Ewa, 2013, Liljeroth *et al.*, 2010). Post-infection, BABA treated plants have been shown to activate defence responses faster than untreated plants (Balmer *et al.*, 2015).

Shailasree *et al.* (2001) tested the various concentrations of β-aminobutyric acid *viz.* 25, 50, 75 and 100 mM against downy mildew

of pearlmillet caused by *Sclerospora graminicola* and reported that seeds treated with 50 mM for 6 hr resulted in the maximum seedling vigour with only 23 per cent disease incidence as compared to control which recorded less seedling vigour (98 %) and disease incidence i.e. 75 per cent protection from disease. They also tested the durability of induced resistance in plants raised from seed treated with the inducer and resistance induced in seeds with BABA remained operative through vegetative and reproductive growth of pearl millet plants.

BABA treated Potato plants also show improved protection to *Phytophthora infestans* (Cohen, 2002). Field studies have shown that BABA treated Potato plants display 40-50 per cent reduction in lesion size in comparison to untreated plants (Liljeroth *et al.*, 2010). Interestingly, BABA is also non-toxic to *Phytophthora infestans* (Justyna and Ewa, 2013), suggesting that the observed protection can be attributed to BABA dependent induction of Potato defence. In similarity to Arabidopsis, a functional SA regulated defence pathway has been shown to mediate BABA dependent induced response to *P. infestans* in Potato (Eschen-Lippold *et al.*, 2010). Furthermore, it has also been shown that BABA treatment induces reactive oxygen species production, PR-1 and hypersensitive like response (HR -like) in Potato. In addition, differential phenotypic responses and phenolic composition were observed among Potato cultivars in response to BABA treatment (Bengtsson *et al.*, 2014).

Si-Ammour *et al.* (2003) analyzed the efficacy of two chemical inducers of disease resistance against *Phytophthora* of Arabidopsis (*P. brassicae*) and potato (*P. infestans*), a functional SA-analogue, benzothiadiazole (BTH), and β-aminobutyric acid (BABA). BABA pre-treatment (300 µM) via soil drench applied 24 hr before inoculation completely protected the susceptible *Arabidopsis* accession *Landsberg erecta* (Ler) from infection with *P. brassicae* whereas similar treatment with BTH (330 µM) did not induce resistance. Spraying the

susceptible potato cultivar Bintje with BABA (1 mM) 2 days before inoculation resulted in a phenocopy of the incompatible interaction shown by the resistant potato cultivar Matilda while BTH (1.5 mM) did not protect Bintje from severe infection.

Ton and Mauch-mani (2004) examined the efficacy and mode of action of BABA on resistance against two necrotrophic pathogens, *Alternaria brassicicola* and *Plectosphaerella cucumerina* of *Arabidopsis*. Treatment of BABA induced resistance to these pathogens to a similar level by jasmonic acid (JA). Conversely, treatment with benzothiadiazole (BTH), a functional analogue of salicylic acid (SA), had no significant effect on the resistance against both pathogens.

Ton *et al.* (2005) stated that plants treated with the non-protein amino acid β–amino butyric acid (BABA) develop an enhanced capacity to resist biotic and abiotic stresses. This BABA induced resistance was associated with an augmented capacity to express basal defense responses.

Polyakovskii *et al.* (2008) investigated the mechanism of action of β-aminobutyric acid (BABA) as an inducer of resistance to the necrotrophic fungi *Botrytis* spp. in three different varieties of onion and showed that low concentrations of BABA are capable of inducing resistance in onion to neck rot. By means of fluorescence microscopy it is established that treatment of the bulb of onion with BABA leads to priming defensive callose deposition, the principal mechanical barrier against penetration of the pathogen.

Zhang *et al.* (2009) stated that the xenobiotic compound β-aminobutyric acid (BABA) primes Arabidopsis innate immunity against attack by microbial and herbivorous insects. This defense priming results in faster, stronger and probably more sustainable defense reactions once the plant was exposed to pathogen or insect attack.

Korosi (2011) studied the effect of β-amino butyric acid (BABA) and 2, 6-dichloroisonicotinic acid (INA) on *Plasmopara holstedii* in

comparision to that of benzothiadiazole on susceptible, partially resistant and completely resistant sunflower under greenhouse and field condition and it was found that BTH, INA and BABA treatments decreased the appearance of the disease in all the sunflowers in greenhouse conditions. Under field conditions INA and BABA effectively enhanced the plant growth.

β-amino butyric acid (BABA), a non-proteinaceous amino acid has shown to induce resistance against various pathogens in several different plant systems with a great degree of success (Justyna and Ewa, 2013).

BABA has not been detected in plants with the exception of few reports (Justyna and Ewa, 2013, Bengtsson, 2013). Use of BABA as a protective agent was first described in 1963 wherein BABA treated pea plants showed significant reduction in root rot symptoms caused by *Aphanomyces euteiches* (Jakab *et al.*, 2001). Since then, BABA has been shown to provide protection against numerous diseases in many different plant systems both in lab and field studies (Liljeroth *et al.*, 2010, Justyna and Ewa, 2013).

Li *et al.* (2016) investigated the duration of action, optimal processing mode and disease related mechanisms of BABA (500 $mg \cdot L^{-1}$) and BTH (50 $mg \cdot L^{-1}$) with tomato seedling by measuring disease index, relative control effect, H_2O_2 and NO content and powdery mildew resistance related gene expression levels and stated that the best time of BABA was the second day and the effective duration was about 10 days, the best time of BTH was the first, third and fifth day and the effective duration was up to 15 days. The contents of H_2O_2 and NO were significantly increased. The expression patterns of CHI3, GLUCA, GLUCB, PR1A genes all showed similar trend, but had differences at different treatment time in different materials thus BABA and BTH could be used to improve the disease resistance of tomato to tomato powdery mildew.

Luna *et al.* (2016) evaluated the different application methods of β-amino butyric acid (BABA) and jasmonic acid (JA) for long-lasting induced resistance in tomato against *Botrytis cinerea*. Result shows that germinating seeds for 1 week in BABA- or JA-containing solutions promoted seed germination efficiency, did not affect plant growth, and induced resistance in 4-week-old plants. When formulating BABA and JA in carboxymethyl cellulose seed coating, only BABA was able to induce resistance in 4-week-old plants. Also root treatment of 1-week-old seedlings with BABA or JA also induced resistance in 4-week-old plants. However, this seedling treatment repressed plant growth at higher concentrations of the chemicals, which was particularly pronounced in hydroponically grown plants after BABA treatment.

2.4.3 Chitosan (CHT)

Allan and Hadwigar (1974) reported that applied chitosan at a rate of 1 mg/ml, was able to reduce the *in vitro* growth of a number of fungi and oomycetes except zygomycetes, which have chitosan as a component of their cell walls.

Pospieszny *et al.* (1991) stated that spraying or inoculation of leaves by chitosan protected various plant species against local and systemic infection caused by alfalfa mosaic virus (ALMV), tobacco necrosis virus (TNV), tobacco mosaic virus (TMV), peanut stunt virus (PSV), cucumber mosaic virus (CMV) and potato virus X (PVX). The efficiency of chitosan in inhibition of viral infection depends upon the host-virus combination, chitosan concentration and mode of its application. The highest protection was observed in the treated leaves, but the effect also appeared in the non-treated parts of the treated plants.

Benhamou and Theriault (1992) studied the effect of chitosan on the induction of host cell reactions at concentrations ranging from 0·5 to 2 mg ml^{-1} with an optimal effect at 2 mg ml^{-1}. Chitosan was applied to tomato plants prior to inoculation with the root pathogen,

Fusarium oxysporum f.sp. *radicis-lycopersici* and it was observed that there was reduction in the root lesions formed by the fungus and increase in the formation of physical barriers like papillae in the infected root tissues.

Lafontaine and Benhamou (1996) investigated the potential of chitosan for controlling *Fusarium* crown and root rot of greenhouse-grown tomato caused by *Fusarium oxysporum* f.sp. *radicis-lycopersici* (FORL) and it was found that the amendment of plant growth substratum with chitosan at concentrations of 12.5 or 37.5 mg l^{-1} significantly reduced plant mortality, root rot symptoms and yield loss attributed to FORL. Maximum disease control was achieved with chitosan at 37.5 mg l^{-1}, when plant mortality was reduced by more than 90 per cent as compared to control.

Reddy *et al.* (1999) reported that seed borne *Fusarium graminearum* infection was reduced to less than 50 per cent at higher chitosan treatment. It was also seen that chitosan also inhibits fungal transmission to the primary roots of germinating seedlings.

Muzzarelli *et al.* (2001) tested the effectiveness of five chemically modified chitosan derivatives in restricting the growth of *Saprolegnia parasitica*. Results indicated that methylpyrrolidinonec-hitosan, *N*-phosphonomethylchitosan, and *N*-scarboxymethylchitosan as opposed to *N*-dicarboxymethylchitosan, did not allow the fungus to grow normally.

The mode of action of chitosan is to stimulate natural defence response systems in treated plants. Poly-D-glucosamine binds to fungal receptor sites, mimicking an attack by fungal spores. This in turn results in signals being sent to the nuclei of the plant and triggering signals which elicit multiple genetic and biological responses, including the production of phytoalexins (anti-microbial compounds produced in plants), aimed at inhibiting infections (Anon., 2002).

Rabea *et al.* (2005) reported the fungicidal activity of 24 new derivatives of chitosan (*i.e.*, *N*-alkyl, *N*-benzylchitosans) using a radial hyphal growth bioassay of *B. cinerea* and *P. grisea* and showed that all derivatives have a higher fungicidal action than the native chitosan. *N*-dodecylchitosan, *N*-(*p*-isopropylbenzyl) chitosan and *N*-(2,6-dichlorobenzyl) chitosan were the most active against *B. cinerea*, with EC50 values of 0.57, 0.57 and 0.52 $g.L^{-1}$, respectively. Against *P. grisea*, *N*-(*m*-nitrobenzyl)chitosan was the most active with 77 per cent inhibition at 5 $g.L^{-1}$. *O*-(decanoyl)chitosan at mol ratio of 1:2 (chitosan to decanoic acid) was the most active compound against *B. cinerea* (EC50 = 1.02 $g.L^{-1}$) and *O*-(hexanoyl)chitosan displayed the highest activity against *P. grisea* (EC50 = 1.11 $g.L^{-1}$).

Bautista-Banos *et al.* (2006) stated that Chitosan is an insoluble cationic polysaccharide of high molecular weight, usually extracted from crustacean shell waste, which has been claimed to control fungal proliferation in tomato crops.

Palma-Guerrero *et al.* (2009) demonstrated that chitosan is able to permeabilize the plasma membrane of *Neurospora crassa* and kills the cells in an energy-dependent manner.

Vasil'ev *et al.* (2009) stated that chitosan induces programmed-cell death (PCD) and hypersensitive-associated responses in plants.

Feliciano-Rivera (2011) evaluated the field efficacy of OMRI-certified and other materials for control of early blight and septoria leaf spot of tomato in 2009 and 2010. Nine fungicides, ammonium bicarbonate and chitosan were evaluated in an organic production system. The results have been showed that copper-based fungicides were most effective fungicides to manage septoria leaf spot and early blight of tomato. Also, none of the biological-based products (Sonata® and Serenade Max®), plant-based extracts (Trilogy® and Regalia® SC), chitosan, ammonium bicarbonate nor horticultural lime sulphur

provided a significant (*P*. > 0.05) reduction in disease severity However, in spite of significant (P < 0.05) disease control in plots treated with copper based products, no significant (P > 0.05) improvement in yield over the untreated control was observed during the first two experiments, in which the initial symptoms of foliar disease were observed after fruits were set. In the third field trial, in which initial symptoms were observed before fruit set, Serenade Max®, Bordeaux mixture, Regalia® SC, water-soluble chitosan and lime sulfur improved yield, although none provided significant disease control.

Abdel-Kader *et al.* (2012) evaluated the efficacy of bio-agents, resistance chemical inducers and/or thyme oil application as foliar spray against vegetables foliar diseases incidence in open greenhouse conditions. Results suggested that application with either *T. harzianum* and *B. subtilis* showed significant reduction in diseases incidence of foliar diseases, *i.e*. Powdery, Downy mildews of Cucumber, Cantaloupe and Pepper as well as Early, Late blights of Tomato. Under artificial infestation the most significant reduction in diseases incidence of tested vegetables were recorded in combined treatments of chemical inducers and *S. serevisiae*, *i.e*. (Chitosan + Thyme oil); (Chitosan + Saccharin); (Chitosan + Calcium chloride + *S. serevisiae*); (Chitosan + Potassium monohydrogen phosphate); (Saccharin + Potassium monohydrogen phosphate) ; (Humic and folic + Thyme oil) and (Chitosan + *S. serevisiae*) comparing with untreated control and the usage of combined application of the bio-agents with resistance plant chemicals might be used as easily applied, safely and cost effective control methods against such foliar plant diseases.

El-Mougy *et al*. (2012) evaluated the efficacy of different plant resistance inducers treatments against Early and Late blight foliar diseases incidence as spray treatments on growing Tomato plants under commercial plastic houses. The evaluated foliar spray treatments were applied four times with fifteen days intervals starting at thirty days after

transplanting. The obtained results revealed that plant spray with treatments, [Calcium chloride (20mM) + *S. cerevisiae* 10 x 1010 cfu/mL (10 ml/L) + Chitosan (0.05 mM)] and [Potassium bicarbonate (20 mM) + Thyme oil (5 ml/L)] resulted in the highest reduction in foliar diseases (Early and Late blight diseases) incidence and severity of Tomato plants grown under plastic houses conditions and increased produced yield. The usage of combined application of the bio-agent *S. cerevisiae* and/or resistance plant chemicals might be used as easily applied, safely and cost effective control methods against such foliar plant diseases.

Ribas-Agusti *et al*. (2013) compared the effects of wettable sulphur and two elicitors (Chitoplant® i. e. Chitosan treatment and Milsana®) on the productivity and nutritional quality of tomato (*Solanum lycopersicum* L cv. Belladonna). The result indicated that both elicitors, and a conventional sulphur treatment showed a similar degree of protection against powdery mildew. One or more of these three treatments positively affected the concentrations of some natural phytochemicals in tomato fruit (e.g. ascorbic acid, quercetin trisaccharide, quercetin-3-*O*-rutinoside, -carotene, and potassium) compared with untreated control plants.

Abada and Ahmed (2014) stated that the inducer resistant chemicals (IRCs) bion, Chitosan, humic acid, salicylic acid and zinc sulphate resulted in significant reduction to the germinated conidia of the powdery mildew fungus compared with control treatment. This reduction was gradually increased by increasing the tested concentration.

Mishra *et al*. (2014) conducted the study to evaluate the comparison between the histological and histochemical changes in leaves of tomato due to Tomato leaf curl virus (ToLCV) with or without plant growth promoting rhizobacteria (PGPR) and Chitosan treatments. The results showed that the combined application reduce the disease severity (%) of ToLCV in Tomato plant.

Ramkissoon *et al.* (2016) studied the commercial chitosan formulation (Armour-Zen®) for its ability to control two important tomato pathogens: *Alternaria solani* and *Xanthomonas vesicatoria* and mechanism of induced resistance by examining induction of enzymes and genes involved in plant defense. Results showed that applications of the chitosan extract significantly reduced the incidences of both diseases in greenhouse and field trials and also displayed a positive effect in growth and yields of tomato, also recorded elevated levels of defense enzymes and upregulation of the PIN II marker gene for defense signaling pathways when compared to the control. It was therefore proposed that chitosan would able to provide broad-range protection through induced systemic resistance mechanisms.

2.5 Yield

Vasudevan *et al.* (2002) suggested that application of chitosan formulations can serve as increase in root and shoot length, and grain yield. It also increases in the growth of nursery-raised plants such as cucumber, pepper and tomato among others.

Yildırım and Dursun (2009) determined the effect of foliar salicylic acid (SA) applications on fruit quality, growth and yield of tomato under greenhouse conditions Tomato plants were treated with foliar SA applications at different concentrations (0.00, 0.25, 0.50 and 1.00 mM). SA was applied with spraying four times during the vegetation at 10-days interval two weeks after planting. In the study, it was determined that foliar applications of SA showed positive effect on some fruit characteristics, plant growth, chlorophyll content in leaves, early yield and total yield. SA treatments had no effect on pH, AA and TA of tomato. Total soluble solids (TSS) increased with foliar SA applications. The greatest stem diameter, leaf dry matter and chlorophyll content were obtained from 0.50 mM SA treatment. SA treatments increased the early yield of tomato compared to the control. The yield of tomato was

significantly influenced by foliar SA applications. The highest yield occurred in 0.50 mM SA treatment.

Anonymous (2011) investigated the effect of Chitosan growth regulator on morphological characters, growth, biochemical, reproductive, yield attributes and yield in summer tomato cultivar cv. BINA tomato-6. The experiment comprised five levels of Chitosan concentrations *viz*. 0 (control), 25, 50, 75 and 100 ppm. The Chitosan was applied two times, at vegetative (25 days after transplanting, DAT) and flower initiation stages (40 DAT). Result showed that application of Chitosan increased plant height, branch and leaf number, leaf area (LA), total dry matter (TDM), absolute growth rate (AGR), relative growth rate (RGR), nitrate reductase (NR) activity and total sugar in leaves, number of effective flower clusters plant^{-1}, flowers plant^{-1}, fruits plant^{-1} and fruit yield over control. 75 ppm. The highest fruit yield was recorded in 75 ppm of Chitosan solution sprayed on tomato plants at 25 and 40 days after transplanting due to increased number of fruits plant^{-1}.

El-Mohamedy *et al.* (2014) investigated the effect of some chemical inducers such as potassium salts, salicylic acid and sorbic acid on control of root rot pathogens and their impact on growth, quantity and quality parameters of tomato and suggested that application of plant chemical resistance inducers could be commercially used for controlling tomato root rot diseases and increasing both quality and quantity of tomato since they are safe, less expensive and effective against these diseases even under field conditions.

Kazemi (2014) studied the effect of salicylic acid in 2 levels (0.5 and 0.75 mmol L^{-1}) and methyl jasmonate in 3 levels (0.25, 0.5 and 0.75 mmol L^{-1}) as pre- harvest treatments on the tomato vegetative growth, yield and fruit quality. Results indicated that salicylic acid (0. 5 mmol L^{-1}) and methyl jasmonate (0.25 mmol L^{-1}) either alone or in combination (0.5 mmol L^{-1} SA + 0.25 mmol L^{-1} MJ) increased vegetative and reproductive growth, yield and chlorophyll content. The application

of salicylic acid (0. 5 mmol L^{-1}) alone significantly increased the leaves-NK content and dry weight and decreased the incidence of blossom end rot, but methyl jasmonate application alone or in combination had not significant effect on blossom end rot and leaves -NK content. The TSS, TA and vitamin C content of tomato fruit had significantly affected by the application of salicylic acid and methyl jasmonate either alone or in combination (0.5 mmol L^{-1} SA + 0.25 mmol L^{-1} MJ). Application of salicylic acid with methyl jasmonate improved the yield contributing factors that resulted in significant increase in tomato fruit yield.

Abd El-Gawad and Bondok (2015) investigated the effect of foliar application with salicylic acid (2 mM/l) alone or combined with chitosan (0.1 %) with or without TMV inoculation on improving resistance, growth, productivity and quality of tomato Hybrid Super Jackal F1. Result showed that all inoculated plants with TMV after 14 and 30 days from foliar application with combination of salicylic acid and chitosan, exhibited symptoms later and less severe than the plants inoculated with TMV in the other treatments. The SA plus CH foliar application without TMV inoculation gave the highest significant values of vegetative growth in both seasons. Combination treatment of SA plus CH increased significantly N, P, K, Fe and Zn concentration. This treatment was also effective in increasing tomato yield compared with treatment of infection alone. Also showed that SA or CH alone or combined significantly increased ascorbic acid concentration compared with control treatment.

Javanmardi and Akbari (2016) evaluated the effect of SA foliar application of SA at different plant growth stages and SA concentrations (0, 150, 300 and 450 mg/L) on fruit yield, secondary metabolites and quality features of tomato (*Solanum lycopersicum* L. cv. Kardelen) under greenhouse conditions. Result showed that the highest fruit yield per plant (about 1.3-fold greater than control) was obtained from 300 mg/L SA when applied three weeks after fruit set. However,

comparing to control plants, the highest fruit firmness, 10 days prolonged storability, highest total phenolics (22.6 mg gallic acid equivalent per 100 g fw); and highest antioxidant activity (65.11) were observed when 450 mg/L SA applied at fruiting stage and 3 weeks later. Also suggested that the effect of exogenous SA application on tomato plant depends on the developmental stage and SA concentrations tested and improved fruit quality factors may happen in a certain concentration range, while over that may have negative or adverse effect.

2.6 Anatomical changes induced due to application of resistance inducers

Lee *et al*. (1999) examined the effect of oligogalacturonic acid (OGA) and Chitosan (b-1,4-linked glucosamine), a component of the fungal cell wall, on stomatal movements in leaf epidermis of tomato (*Lycopersicon esculentum* L.) and *Commelina communis* L. Result showed that these elicitors reduced the size of the stomatal aperture. Chitosan inhibited light-induced stomatal opening in tomato epidermis. The effects of OGA and Chitosan were suppressed when EGTA, catalase, or ascorbic acid was present in the medium, suggesting that Ca^{2+} and H_2O_2 mediate the elicitor-induced decrease of stomatal apertures. Also suggested that the H_2O_2 that is involved in this process was produced by guard cells in response to elicitors. The guard cells infected by pathogens may close their stomata via a pathway involving H_2O_2 production, thus interfering with the continuous invasion of pathogens through the stomatal pores.

It has been suggested that BABA potentiates salicylic acid (SA) and abscisic acid (ABA) dependent defence responses that leads to increased PR-1 expression in response to *Pseudomonas syringae* and enhanced callose deposition in response to *Hyloporenospora arabidopsidis*, leading to improved resistance (Ton *et al.*, 2005).

Huckelhoven (2007) reported that the plant cuticle and cell wall separate entry of microbial pathogens. While microbial pathogen try to breach these barriers for colonization, plants respond to attempted

penetration by a battery of wall associated defense reactions. Both early plant defense to fungal penetration and host reprogramming for susceptibility could function at the host cell periphery.

Farouk *et al.* (2008) reported that application of elicitor chitosan @ 0.05 % and salicylic acid @ 100 mg/lit increased leaf anatomical characters, i.e. thickness of leaf blade, thickness of palisade and spongy parenchyma as well as thickness of midrib region and dimension of vascular bundles as a means of protection against cucumber downy mildew pathogen.

Cárcamo *et al.* (2012) investigated the impact of exogenous application of SA on the tissue morphology of lluteño maize under Nacl stress. The treatment consisted of salt and SA (0.1 mM). Result showed that salt stress negatively affected leaf anatomy and application of SA showed positive effect of SA on the composition of structures such as xylem vessels, Kranz anatomy, mesophyll cells, epidermal tissues, leaf blades and chloroplast development. It also increased the number of stomata per unit area. Finally, the presence of salicylic acid in saline conditions has beneficial effects on the morphology of lluteño maize, likely contributing to the tolerance to salinity.

Gomma *et al.* (2015) studied the influence of foliar spray with various concentrations of salicylic acid (0, 25, 50, 75, 100 and 150 ppm) on stem and leaf anatomy of Egyptian lupine 'Giza 2'. The obtained results revealed that the median used concentrations of Salicylic acid (SA) induced promotive effect on vegetative and reproductive characters of Egyptian lupine 'Giza 2'. The treatment of 75 ppm SA conc. induced favorable changes in anatomical structures of vegetative growth. Foliar application with 75 ppm SA induced prominent increase in stem diameter due mainly to the prominent increases induced in thickness of stem wall and in diameter of hollow pith. The increase in stem wall thickness could be attributed to the induced increase in thickness of epidermis, cortex, fiber strands, phloem and xylem tissues as well as in

thickness of parenchymatous area of the pith and in vessel diameter. Likewise, such treatment increased thickness of both midvein and lamina of leaflets due to increase induced in thickness of both palisade and spongy tissues and to increase induced in size of midvein bundle.

2.7 Biochemical changes induced due to application of resistance inducers in host plant

2.7.1 Salicylic acid (SA)

White (1979) stated that the exogenous application of SA to tobacco plants results in tobacco mosaic disease resistance which was correlated with PR-gene expression.

Mur *et al.* (1996) showed that tobacco (*Nicotiana tabacum*) plants protected with SA displayed augmented expression of defense related genes that are usually not directly responsive to SA itself. They proposed a dual role for SA in induced resistance: a direct one leading to the expression of some PR-proteins and an indirect one regulating augmented expression of defense related genes such as phenyl alanine ammonia lyase, upon elicitation of defense.

Conrath *et al.* (2002) demonstrated that pretreatment of SAR inducers like salicylic acid (SA) or benzothidiazole (BTH) leads to the direct activation of certain defense related genes and primes the cells for stronger elicitation of other defense related genes including those encoding phenyl alanine ammonia lyase (PAL).

Ju *et al.* (2000) studied the effect of seedling treatment of salicylic acid (SA) on the activity of peroxidase (POD), polyphenol oxidase (PPO) and phenyl alanine ammonia lyase (PAL) at two leave stage in cucumber. The activity of POD changed at 24 hr after treatment and increased steadily till 144 hr after treatment with 193 per cent increment as compared with control. Similarly the activity of PPO increased by 61.7 per cent at 144 hr after treatment. The PAL activity reached a little peak 72 hr after treatment and then declined, another peak appeared in 120 hr after treatment.

Feng *et al.* (2004) studied the effect of salicylic acid (2 mM) on cotton against *Verticilium* wilt under greenhouse condition. The activity of PAL, POD, PPO and glucanase was increased significantly compared with control within 7 days and it was considered that these increased activities of enzymes was involved in the biochemical mechanism of SA induced resistance to *Verticilium* wilt.

Tosun *et al.* (2006) stated that salicylic acid induced the accumulation of pathogenesis related proteins (chitinase and β-1, 3 glucanase) and peroxidase, while $Harpin_{Ea}$ was failed to provide significant increase in chitinase activity when SA and $Harpin_{Ea}$ alone and in combination with systemic fungicide phosphorus acid (Agriphos 400) were applied to tomato plants to induce the resistance against *Phytophthora infestans*. SA and $Harpin_{Ea}$ treatment also exerted pleotrophic effects on tomato growth and development and photosynthetic pigments contacts.

Houssien *et al*. (2010) used *Trichoderma harzianum*, salicylic acid and low dose of thiophanate methyl as recommended fungicide to enhance tomato defense response against wilt disease caused by *Fusarium oxysporum* f.sp. *Lycopersici* under greenhouse conditions and investigated the changes in various physiological defenses including enzymes like polyphenol oxidase (PPO), peroxidase (POD) and acid invertase (AI); total soluble phenols; protein and chlorophyll content. The tomato plants infected by *Fusarium oxysporum f. sp. Lycopersici*, one week after inoculated with *Trichoderma harzianum* (TH) fungi (seedling root dipping and/or soil treatment) and/ or sprayed daily for one week with hormonal inducer (SA). Plants were harvested at 35 days after pathogen infection. All applied treatments completely protected tomato seedlings against *Fusarium* wilt. Disease index percentage (DI %) was significantly reduced up to zero per cent. Level of all the determined physiological parameters greatly changed in tomato plants in response to *Fusarium oxysporum* (FO), TH fungi and hormonal elicitor and reflected

many components of defense signals which leading to the activation of power defense system in tomato against pathogen attack. Application of *Trichoderma harzianum* and salicylic acid stimulated all these parameters not only to reach but also exceed their content in healthy control.

Abada and Ahmed (2014) evaluated the effect of combination between bioagents and antioxidants on management of tomato powdery mildew. The result showed that plants sprayed with salicylic acid recorded the highest activity of the three oxidative – reductive enzymes, i.e. PAL, PO and PPO followed by that sprayed with B. thuringiensis then T. harzianum, meanwhile untreated tomato leaves (control) recorded the lowest activity followed by that sprayed with Sumi-8.

2.7.2 β-amino butyric acid (BABA)

Oostendorp *et al.* (2001) stated that DL- β-aminobutyric acid (BABA) or its 3-(s)-enantiomer have been reported to activate disease resistance against downy mildew in various crops, when used at relatively high rates. PR-1 induction has been reported after application of high concentration of the compound in tomato and activation has been reported to depend on salicylic acid accumulation and lesion formation.

Toquin *et al.* (2002) stated that the chemical inducer BABA has showed to prime disease resistance without direct induction of PR-genes in *Arabidopsis* and was therefore an ideal tool to analyze the process of priming.

The partial resistance against systemic viruses has been obtained in the lab by using BTH, BABA and chitosan (Schreiber and Desveaud, 2008). However, the efficacy of these compounds in open field was acceptable only when treatments were coupled with specific agricultural practices (i.e. UV-reflective plastic mulch, non-woven covers, and insecticides). By this strategy the most successful results were obtained in controlling tomato spotted wilt virus (TSWV) and tomato

yellow leaf curl sardinia virus (TYLCS) and their vectors (Momol *et al.*, 2004; Fanigliulo *et al.*, 2009)

Korosi (2011) reported that the activator treatment *viz.* INA, BABA and BTH increased the defense related polyphenol oxidase and peroxidase enzyme activity in both susceptible and resistant sunflowers against *Plasmopara holstedii.*

Raut and Borkar (2014) studied the effect of application of resistance inducing chemicals on PR-proteins accumulation in tomato plant during period of induced resistance against *alternaria* leaf blight and reported that the application of RIC SA and BABA elicits the increased activity of PR-proteins *viz*. chitinase and β 1, 3-glucanase which inhibit the process of pathogenesis in susceptible tomato cultivars to exhibit the resistance.

2.7.3 Chitosan (CHT)

Tiuterev *et al.* (1996) stated that the mechanism of action of natural disease resistance inducer chitosan was based on the increase of lignin biosynthesis and plant cell wall lignifications.

Chitosan treatment enhanced the content of phenolic compounds in post-harvest tomato fruit and elicited a specific anti-fungal activity (Liu *et al.*, 2007).

Hassni *et al.* (2004) studied the effect of chitosan in date palm in response to *Fusarium oxysporum* f.sp. *albedinis*, the causal agent of a major wilt. When injected into the roots at various concentrations chitosan elicited date palm peroxidase and polyphenoloxidase activities with increased level of phenolic compound hydroxycinnamic acid derivatives.

Iriti *et al.* (2009) stated that chitosan (non-toxic and low expensive polyamines), act as pathogen associated molecular patterns (PAMPs), stimulate plants own defense mechanism by means of oxidative burst, hypersensitive response (HR), callose apposition, phytoalexin and pathogenesis related (PR) protein synthesis. They also

showed that CHT when applied to bean leaves increase the ABA level over three fold. This ABA increment mediates both callose depositions involved in the resistance to viruses.

Hadrami *et al.* (2010) stated that chitin and chitosan are naturally-occurring compounds, display toxicity and inhibit fungal growth and development. These were reported to be active against viruses, bacteria and other pests. These compounds have eliciting activities leading to a variety of defense responses in host plants in response to microbial infections including the accumulation of phytoalexins, pathogen related (PR) proteins and proteinase inhibitors, lignin synthesis, and callose formation.

Sujeeth *et al.* (2010) stated that maximum hydroxyproline rich glycoproteins (HRGP) accumulation was observed in seedlings raised from susceptible seeds treated with chitosan. Peroxidase and hydrogen peroxide, essential components for HRGP cross-linking, were also increased in samples treated with chitosan.

Falcioni *et al.* (2013) showed that SA treatment increased the expression of PR2, the activity of phenylalanine ammonia lyase (PAL) and the concentration of antioxidant compounds at 7 days post-treatment. SA treatment delayed the detection of PVX by ELISA in uninoculated leaves of mechanically infected tomato plants. Although the effect of PVX infection on physiological parameters was weak, moderate SA treatments showed enhanced photosynthesis, particularly for infected plants. The results obtained confirm that SA promotes major changes in the induction of resistance in tomato plants and suggest that treatment with exogenous SA could be considered to reduce the infections caused by PVX.

Mishra *et al.* (2014) conducted the study to evaluate the comparison between the histological and histochemical changes in leaves of tomato due to Tomato leaf curl virus (ToLCV) with or without plant growth promoting rhizobacteria (PGPR) and Chitosan treatments. The

results showed that the combined treatment of Chitosan and *Pseudomonas sp.* induce a significant (P=0.05) increase in the activities of Polyphenol oxidase (PPO), chitinase, Phenylammonialyase (PAL) and enhanced the content of phenolic compounds in Tomato leaves.

2.8 Role of sugar, protein, phenols, lignin in resistance to fungal pathogen

Vance *et al.* (1980) stated that lignin, a major component of cell walls of vascular plants, accumulated in cell wall apposition mediated defense and was considered as first line defense against successful penetration of invasive pathogens. Lignification renders the cell wall more resistant to mechanical pressure applied during penetration by fungal appressoria as well as more water resistant and thus less accessible to cell wall-degrading enzymes.

Bennett and Wallsgrove (1994) reported that an enormous variety of secondary metabolites were derived from shikimic acid or aromatic amino acids, many of which have important roles in defense mechanisms. Several classes of secondary products were induced by infection, wounding or herbivory.

Barcelo (1997) stated that cell wall lignification is a complex process occurring exclusively in higher plants; its main function is to strengthen the plant vascular body. This process involves the deposition of ill-defined phenolic polymers, the so-called lignins, on the extracellular polysaccharidic matrix. These polymers arise from the oxidative coupling of three cinnamyl alcohols in a nonrandom reaction, in which cell wall polysaccharides appear to influence the freedom of cinnamyl alcohol radicals, giving rise to a highly orchestrated process.

Samia and Khallal (2007) treated the *Fusarium oxysporum* infected tomato plants with arbuscular mycorrhizal (AM) fungi and/or sprayed three times with inducer (JA and SA) and showed that total soluble sugars, free amino acids and total soluble proteins increased in

both leaves and roots of AM and/ or JAand SA- treated plants as compared with infected control.

Freeman and Beattie (2008) stated that many cell walls also contain lignin, a heterogeneous polymer composed of phenolic compounds that gives the cell rigidity. Lignin is the primary component of wood and cell walls that become "lignified" are highly impermeable to pathogens and difficult for small insects to chew. Cutin, suberin, and waxes are fatty substances that may be deposited in either primary or secondary cell walls (or both) and outer protective tissues of the plant body, including bark.

Rodaki *et al.* (2009) examined the impact of glucose upon the *Candida albicans* transcriptome by testing the sensitivity of this pathogen to wide ranging glucose levels (0.01, 0.1 and 1.0 %) and showed that *C. albicans* was extremely sensitive to glucose, regulating central metabolic genes even in response to 0.01 % glucose. Thus suggested that *C. albicans* cells respond to glucose increasing their resistance to the oxidative and cationic stresses central to the armory of immunoprotective phagocytic cells.

Ashry and Mohammad (2012) studied the changes in various physiological defenses like secondary metabolites, proline, total soluble protein in leaves and stems of 18 flax lines either resistant or susceptible to powdery mildew. Results showed that Stems of the resistant flax lines had a higher content of total phenols than susceptible ones upon pathogen inoculation. Total soluble protein content in flax leaves increased significantly in resistant lines when compared with resistant parent but decreased significantly in susceptible lines when compared with resistant parent.

2.9 Role of phenyl alanine ammonia lyase, peroxidase, polyphenol oxidase in resistance to pathogen

Modafar *et al.* (2001) inoculated the roots of resistant and susceptible cultivars of date palm seedlings by *Fusarium oxysporum* f. sp.

albedinis (Foa) and observed that there was an increase in activity of phenylalanine ammonia-lyase. The post-infectional response in the PAL activity in the resistant cultivar roots was faster and higher than that in the susceptible cultivar.

Gerasimova *et al.* (2005) stated that biogenic elicitors (chitosan and its complex with salicylic acid) and an immunosuppressor (laminarin) were shown to increase the activity of L-phenylalanine ammonia lyase and protein synthesis in potato tubers.

Joseph *et al.* (2005) analysed the preoxidase and polyphenol oxidase activities in fifteen clones of *Theobroma cacao* after inoculation with *Phytophtora megakarya*. It was found that soluble peroxidase activity on inoculated leaves increased in three clones. The increase was higher in the clone Playa $Alta_2$ than in clone UPA 143 and PA 107. At the same time polyphenol oxidase activity in 5 day old inoculated leaves increased by 92 and 47 per cent in clones PA 107 and UPA 143 respectively.

Wen *et al.* (2005) carried an experiment of an in vivo incubation of grape berry tissues in the salicylic acid contained medium and showed that SA could induced the accumulation of PAL mRNA, the synthesis of new PAL protein and increase the activity.

Karthikeyan *et al.* (2005) studied the induction of defense related proteins *viz.* chitinases, β-1, 3 glucanases, peroxidase and polyphenol oxidase by application of *Pseodomonas fluorescens* Pf1 against *Alternaria palandui* in resistant (IHR-56) and susceptible (MDU-1) cultivars of onion. Chitinase in both the cultivars, with or without challenge inoculation of the pathogen revealed changes in isoform pattern. Isoform analysis of peroxidase and polyphenol oxidase exhibited induction in response to pathogen. Similarly the activity of β-1,3 glucanase was greatly induced in Pf1 treated plants challenged with the pathogen as compared to control.

Raj *et al.* (2006) analysed the polyphenol oxidase (PPO) activity in resistant and susceptible pearlmillet cultivars with or without inoculation of downy mildew pathogen *Sclespora graminicola* and observed that the seedlings of resistant varieties had greater PPO activity than susceptible seedlings and inoculated seedlings had significantly higher PPO levels than uninoculated seedlings.

Saikia *et al.* (2006) studied the elicitor properties of cell-wall protein (CWP) of *Fusarium oxysporum* f.sp. *ciceri* and *Macrophomina phaseolina*. The chickpea seedlings exposed to CWP showed enhanced synthesis of phenol, pathogenesis related proteins and activities of phenyl alanine ammonia lyase and peroxidase relative to water treated control. Both *Fusarium* wilt and charcoal rot diseases of chickpea was significantly reduced in CWP treated seedlings.

Koc and Uston (2011) investigated the biochemical response of two cultrivars of pepper (resistant CM-334 and susceptible KM-Hot) to *Phytophthora capsici* by comparing *P. capsici* inoculated and non-inoculated roots, stems and leaves and observed the highest PAL content in control and infected leaves and stems of CM-334 cultivar. They also observed the highest rate of phenolic content in stems and leaves of resistant genotypes compared with control. These results suggested that induction of PAL and accumulation of phenolics might have contributed to restrict the invasion of *P. capsici*.

Xu *et al.* (2011) stated that on the basis of expression of genes, enzyme activity and lignin levels revealed differences between resistant and susceptible cotton plants. Both types of plants showed an increased level of expression of lignin synthesis related genes and increased phenyl alanine ammonia lyase (PAL) and peroxidase enzyme activity after inoculation with *Verticilium dahliae*, but the increase was greater and faster in the resistant line. Histochemical analysis of lignin revealed that the resistant cotton not only retains its vascular structure but also accumulates high levels of lignin. Also quantitative analysis

demonstrated increased lignification and cross linking of lignin in resistant cotton stems. Thus lignin was believed to play a critical role in resistance of cotton to disease.

Ashry and Mohammad (2012) studied the changes in antioxidant enzymes in leaves and stems of 18 flax lines either resistant or susceptible to powdery mildew. Results showed that infection with powdery mildew significantly increased peroxidase, polyphenol oxidase and catalase activities in leaves of flax lines when compared with either resistant or susceptible parents.

Ngadze *et al.* (2012) studied the role of polyphenol oxidase (PPO), peroxidase (POD), phenylalanine ammonia lyase (PAL), chlorogenic acid, and total soluble phenols in imparting resistance to soft rot pathogens. The results showed significantly higher enzyme activity of PPO and PAL as well as higher concentrations of chlorogenic acid and total soluble phenols in Vanderplank, Pentland Dell, M69/11, Romano, M59/20, and Mondial$_{Zw}$. PAL activity increased significantly with time in all varieties, and the highest activity was recorded 8 hr after cutting. The resistance of the varieties was correlated with high PPO and PAL enzyme activity as well as increased concentrations of chlorogenic acid and total soluble phenols. PPO, POD, and PAL activities increased significantly in wounded and inoculated tubers. These findings show that PAL, PPO, POD, chlorogenic acid, and total soluble phenols play a role in imparting resistance to potato soft rot infection.

2.10 Role of β -1,3 glucanase and chitinase in resistance to pathogen

Wyatt *et al.* (1991) stated that β-1,3-glucanase, chitinase, and peroxidase activities were higher in leaf tissue from the main stalk of tobacco (resistant to blue mould) as compared to leaf tissue from suckering stems (susceptible to blue mould) on the same plant. Isozyme patterns of β-1,3-glucanase and chitinase in all resistant tissues are

typical of those of tissues systemically protected by either *stem injection with Peronospora tabacina.*

Krishnaveni *et al.* (1999) studied the sorghum genotypes that differ in their resistance to the fungus, *Fusarium moniliforme.* These genotypes were analysed for their response to stress induced by infection with the fungus. The pathogenesis related proteins *viz.* chitinases and β-1,3 glucanases were induced in the plant tissues in all inbreds under the stress condition and this induced enzyme activities were found in the apoplastic space and intracellular locations, but the distribution varied depending on the genotype.

Christopher *et al.* (2000) stated that expression of PR-proteins *viz.* chitinase and β-1, 3 glucanase exhibited antifungal activity against *Alternaria solani* in response to the release of hyper sensitive response elicitors from fungal cell wall in tomato.

Wrobel-Kwiatkowska *et al.* (2004) reported that the ectopic expression of a β-1, 3-glucanase cDNA in flax has improved the plant's defense against pathogen. The resistance of transgenic lines against *Fusarium oxysporum* and *Fusarium culmorum* was about threefold higher than for the non-transformed plants.

Shrestha *et al.* (2008) observed that chitinase activity was detected 24 hr after inoculation of moderately resistant cultivars but in the susceptible cultivars chitinase activity was detected 36 hr after inoculation and thus stated that moderately resistant cultivars had higher levels of chitinase activity and lower disease severity and number of infection cushions formed compared to susceptible ones.

Freeman and Beattie (2008) stated that hydrolytic enzymes are produced by some plants in response to pathogens and often accumulate in extra cellular spaces where they degrade the cell walls of pathogenic fungi. Chitinases are enzymes that catalyze the degradation of chitin, a polymer with a backbone similar to cellulose that is present in the cell walls of true fungi. Glucanases are enzymes that catalyze the

degradation of glycosidic linkages in glucans, a class of polymers similar to cellulose that is present in the cell walls of many oomycetes (water molds). *In vitro* analysis has verified the anti-fungal properties of these compounds and transgenic plants expressing high levels of these enzymes exhibit increased resistance to a wide range of both foliar and root pathogens.

2.11 Role of epidermal layer thickness and palisade tissues in resistance to pathogen

Sigee and Epton (1976) studied the infiltration of resistant (Red Mexican) and susceptible (Prince) varieties of *Phaseolus vulgaris* with a suspension containing 10^{10} cells/ml of *Pseudomonas phaseolicola* (race 1). The resistant host showed degenerative changes in palisade mesophyll cells and some of the spongy mesophyll cells that were similar to natural senescence and involved a fall in the frequency of free ribosomes without gross initial disruption of chloroplasts. These gradual changes may have been due to the isolation of living cells by dead cells rather than the direct effect of the pathogen.

Kaur and Dhillon (1985) studied structural characteristics of wheat leaves in relation to resistance against leaf rust pathogen. In general, in resistant genotypes, the thickness of epidermal cell wall, number of sclerenchyma layers and their cell wall thickness were significantly higher.

Yang *et al.* (1992) conducted an experiment to determine whether the cuticles in two genera of the family cruciferae are effective barriers to infection by *Rhizoctonia solani*. Seven days old seedlings of mustard cultivars are invariably more resistant than rapeseed cultivars. *B. napus* cultivar did not showed an obvious cuticle layer at one week stage but at three week stage, the presence of cuticle is seen with concomitant increase in resistance to *R. solani*. Three week old plants of *S. alba* had a much lower per cent disease rating and a significantly thicker cuticle layer than similar age of *B. napus* and thus suggested that

the cuticle plays an important role in the resistance of *S. alba* and older plants of *B. napus* to infection by *R. solani*.

Gelelcha and Hanchinal (2003) stated that wheat rust resistant genotypes had thicker epidermis, more number of epidermal cells, and thicker leaf lamina than the corresponding susceptible genotypes. These leaf morphophysiological traits had significant contribution in rust resistance mechanism.

Smith *et al*. (2007) studied the anatomical changes in juvenile leaves of two *Eucalyptus* species, with contrasting susceptibility to infection by *Mycosphaerella* leaf disease. The results showed that there was an enhanced resistance of *Eucalyptus nitens* attributed to the formation of an effective lignified and suberized necrophylactic periderm, to restrict pathogen spread. Whereas *Eucalyptus globulus* formed a slower, distorted necrophylactic periderm through hypertrophic changes to existing mesophyll.

Freeman and Beattie (2008) stated that the cell wall was a major line of defense against fungal and bacterial pathogens. It provides an excellent structural barrier that also incorporates a wide variety of chemical defenses that can be rapidly activated when the cell detects the presence of potential pathogens. All plant cells have a primary cell wall, which provides structural support and is essential for turgor pressure, and many also form a secondary cell wall that develops inside of the primary cell wall after the cell stops growing. The primary cell wall consists mostly of cellulose, a complex polysaccharide consisting of thousands of glucose monomers linked together to form long polymer chains. These chains are bundled into fibers called microfibrils, which give strength and flexibility to the wall. The cell wall may also contain two groups of branched polysaccharides: cross-linking glycans and pectins. Cross-linking glycans include hemicellulose fibers that give the wall strength via cross-linkages with cellulose.

Azevedo *et al.* (2011) studied the leaf morphological characteristics in sugarcane cultivars against brown rust and observed that thick cuticle and thick epidermis prevents the penetration of fungi. The thickest cuticle observed in resistant cultivar PR 980. These results indicate that morphological characteristics such as thick cuticle, thick epidermis was in relation with rust resistant cultivars, so could be used in the selection of resistant cultivars against brown rust.

Lazniewska *et al.* (2012) stated that highly hydrophobic cuticle was an outermost shield separating plant cells from harmful external environment. However, suitable cuticle chemistry was also a necessary factor for enabling host recognition and development of infection structures by pathogenic fungi. Fortification of the plant cell wall in the form of callose deposition at the site of fungal penetration restricts the pathogen to the infection area.

2.12 Biochemical molecules induced/increased due to resistance inducing chemicals

Reddy *et al.* (1999) stated that the synthesis of precursors of lignin such as p´-coumaric, ferrulic and sinapic acids and phenolic acids having antimicrobial activity such as benzoic, p´-coumaric, caffeic protocatechuic, chlorogenic, ferrulic and gallic acid was also stimulated by chitosan treatment.

Prithiviraj *et al.* (2007) studied the effect of an allelochemical catechin produced by the invasive weed *Centaurea maculosa* (spotted knapweed) against *Pseudomonas syringae* pv. *tomato* DC3000 in *A. thaliana* leaves and it was observed that pretreatment of *A. thaliana* roots with low concentrations of catechin induced resistance to the pathogen. The experiments with *A. thaliana* mutants indicated that catechin induces pathogen resistance by up-regulating defense genes via the salicylic acid pathways.

Panina *et al.* (2007) studied the changes in phenolic compounds in tomato in response to biocontrol and plant pathogenic

Fusarium oxysporum and noted the quantitative changes in the phenolic compounds *viz.* ferrulic, vanillic and caffeic acid as well as in unidentified phenolic compounds with an HPLC profile.

Mandal *et al.* (2009) demonstrated that exogenous application of 200 µM salicylic acid through root feeding and foliar spray could induce resistance against *Fusarium oxysporum* f.sp. *Lycopersici* (Fol) in tomato. Endogenous accumulation of free salicylic acid in tomato roots was detected by HPLC and identification was confirmed by LC-MS/MS analysis. Also showed the activities of phenylalanine ammonia lyase (PAL, EC 4.3.1.5) and peroxidase (POD, EC 1.11.1.7) were 5.9 and 4.7 times higher, respectively than the control plants at 168h of salicylic acid feeding through the roots. The increase in PAL and POD activities was 3.7 and 3.3 times higher, respectively at 168h of salicylic acid treatments through foliar spray than control plants. Significant increase in basal level of salicylic acid in noninoculated plants indicated that tomato root system might have the capacity to assimilate and distribute salicylic acid throughout the plant. The results indicated that the induced resistance observed in tomato against *Fol* might be a case of salicylic acid-dependent systemic acquired resistance.

Ghareib *et al.* (2010) evaluated the allelopathic potential of the acetone fraction of *Chenopodium murale* and vanillic acid through laboratory bioassays against tomato plants. Results showed that the allelopathic potential induced by low concentrations of the acetone fraction of *Chenopodium murale* and vanillic acid stimulated the germination and growth of tomato and had stimulating effects on the activity of some antioxidant enzymes. The activity of catalase, peroxidase, superoxide dismutase and polyphenol oxidase as well as the content of soluble protein and phenolic glycoside had been found to be enhanced.

3. MATERIALS AND METHODS

The details of materials and the methods adopted during the course of investigation are as given below-

3.1 Experimental site and material

3.1.1 Location

The present investigation on "Field efficacy of resistance inducing chemicals against Tomato diseases" was carried out at Mahatma Phule Krishi Vidyapeeth, Rahuri. Field experiments were conducted at experimental field of Department of Plant Pathology and Agricultural Microbiology, Post Graduate Institute, MPKV, Rahuri during *Kharif* season of the year 2014-15 and 2015-16.

3.1.2 Geography and climate

Geographically, the Mahatma Phule Krishi Vidyapeeth, Rahuri is situated between 19°47' and 19°57' North latitude and 74°19' and 74°32' East longitude. The altitude above mean sea level is 495 to 569 meter of Arabian Sea. This tract is lying on the eastern side of Western ghat which falls under rain shadow area. Climatologically, this area falls in the Scarcity Zone (Semi-arid tropics) with annual rainfall ranging between 307 and 619 mm. The average annual rainfall is 520 mm. The rainfall is erratic and ill distributed in 15 to 45 rainy days in different years. Out of total rainfall, about 80 per cent is received from South West monsoon (June to September), while the rest from North - East monsoon (October and November). The annual mean maximum temperature was 37.9^{0}C with a range between 33 and 43^{0}C and annual mean minimum temperature was 17.2^{0}C with a range between 3^0 and 18^{0}C. The mean relative humidity in the morning and evening was 59 and 35 per cent, respectively. Agro-climatically the location is in the drought prone areas of Maharashtra state, characterized by low, erratic rainfall with less rainy days coupled with long dry spells.

3.1.3 Seed material

Seeds of tomato *var. Dhanashree* were procured from Tomato Improvement Scheme, MPKV, Rahuri.

3.1.4 Resistance inducing chemicals

The resistance inducing chemicals used during the course of research work were of Glaxo, Sigma-aldrich, E-merk and HI-media. Chemicals *viz.,* salicylic acid (SA), β-aminobutyric acid (BABA), chitosan (CHT) was used during the study.

3.1.5 Glasswares

The glasswares used during the course of research work were of Borosil make.

3.1.6 Instruments

The instruments *viz.* laminar air flow, autoclave, hot air oven, electronic balance, light microsope, refrigerator, spectrophotometer, centrifuge, incubator and water bath were used from Department of Plant Pathology and Agril. Microbiology for research work.

3.1.7 Miscellaneous materials and tools

Fertilizers, insecticides, labels, sprayer, Plastic crates, cotton, etc. were used during the course of investigation.

3.2 Experimental details

3.2.1 Land preparation

The land selected for experimental purpose was ploughed once and two harrowing were given. The appropriate quantity of Farm Yard Manure (FYM) was uniformly spread all over the land before preparatory tillage operation. The soil was brought to fine tilth condition. The experiment was carried out in *Kharif* 2014 and *Kharif* 2015 in RBD design with three replication. The gross and net plot area were 697.5 sq. m and 548 sq. m.

3.2.2 Layout and plan

Location - PGI farm, Central campus, MPKV, Rahuri

Year - *Kharif* 2014 and *Kharif* 2015

Crop - Tomato

Variety - Dhanashree

Spacing - 75cm × 30cm

Plot size - 11.25 sq.m.

Total no. of treatments -16

No. of replications - 3

Design - Randomised Block Design (RBD)

3.2.3 Treatment details

Treatment No.	Treatment	Concentration
T_1	Salicylic acid- seed treatment (SA-S)	1.5mM
T_2	Salicylic acid- seedling treatment (SA-SL)	1.5mM
T_3	Salicylic acid- spray treatment (SA-SPY)	1.5mM
T_4	Salicylic acid- seed + seedling treatment (SA- S+SL)	1.5mM
T_5	Salicylic acid- seed + seedling + spray treatment (SA- S+SL+SPY)	1.5 mM
T_6	BABA- seed treatment (BABA- S)	15 mM
T_7	BABA- seedling treatment (BABA-SL)	15 mM
T_8	BABA- spray treatment (BABA- SPY)	15 mM
T_9	BABA- seed + seedling treatment (BABA-S+SL)	15 mM
T_{10}	BABA- seed + seedling + spray treatment (BABA- S+SL+SPY)	15 mM
T_{11}	Chitosan- seed treatment(CHT-S)	15 mM
T_{12}	Chitosan- seedling treatment(CHT-SL)	15 mM
T_{13}	Chitosan- spray treatment (CHT-SPY)	15 mM
T_{14}	Chitosan- seed + seedling treatment (CHT-S+SL)	15 mM
T_{15}	Chitosan- seed + seedling+ spray treatment (CHT-S+SL+SPY)	15 mM
T_{16}	Control	

3.2.4 Raising of nursery, transplanting and intercultural operations

The seeds of the Tomato were sown in raised nursery bed. The seedlings were raised up to one month.

The transplanting of one month old tomato seedling was done with 75 cm × 30 cm spacing on second week of July in *Kharif* 2014 and *Kharif* 2015.

At an each hill single plant was maintained. Forty plants were maintained in each plot. The recommended fertilizer dose 200:100:100 kg/ha (N:P:K) was applied to tomato crop. Hand weeding was carried out to control weeds in field and irrigations were given in spell. The field was exposed to natural infection of the diseases. However no fungicide spray was given in whole cropping period however insecticides were sprayed to manage *Helicoverpa* fruit borer and serpentine leaf miner.

3.3 Methods

3.3.1 Efficacy of resistance inducing chemicals to induce resistance into tomato plant against diseases under field condition

3.3.1.1 Seed treatment

Seeds of tomato *var. Dhanashree* were first surface sterilized with 0.1 % $HgCl_2$ for 30 sec. followed by three subsequent washings with sterilized distilled water. The sterilized seeds were dipped in the solution of resistance inducing chemicals for 8 hr. Seeds dipped in distilled water served as a control. After 8 hr, the treated seeds were sown in the nursery for raising the seedlings. The raised seedlings were exposed to natural infection.

3.3.1.2 Seedling treatment

Tomato seedlings of *var. Dhanashree* were raised in nursery. The 21 days old seedlings were uprooted gently after adding sufficient quantity of water to loosen the soil. The roots of seedling were washed

with distilled water and then dipped in resistance inducing chemical (RIC) of required concentrations separately for 2 hr for each treatment and transplanted in field. The seedlings dipped in sterile distilled water served as a control.

3.3.1.3 Spray treatment

The seedlings were planted in field in different sets. These set were sprayed with RIC at 1.5 mM concentration of salicylic acid or 15.0 mM concentration of β-amino butyric acid and 15.0 mM concentration of chitosan at 15, 30, 45, 60, 75 and 90 days intervals and the efficacy of RIC's and maintenance of induced resistance throughout the crop growth period was observed.

3.3.1.4 Observations of Disease incidence, severity, area under disease progress curve and infection rate

The observations of disease incidence were recorded from all plants in per plot per treatment in the field trial at 15 days interval. The calculation were made by using the following formula :

$$\text{Per cent disease incidence} = \frac{\text{Number of infected plants}}{\text{Total number of plants}} \times 100$$

The per cent disease severity was calculated. In each field ten plants were randomly selected and for measuring disease severity at lower, middle and upper crop canopy were observed and evaluated individually using 0-9 rating scale (Mayee and Datar, 1986) based on leaf area covered by blight as mentioned below in Table 1.

Table 1. Rating scale (grade) used for Early blight of tomato

Rating scale (Grade)	Disease severity	Level of resistance/ susceptibity
0	No symptom	Immune
1	Small circular, scattered, brown spots, covering 1 per cent or less of the leaf area	Highly Resistant
3	Spots enlarging, dark brown in colour covering 1 to 10 per cent of leaf area and infection on the lower most leaves of the plant	Resistant
5	Spots enlarging, dark brown in colour covering 11 to 25 per cent of leaf area and infection on the lower most leaves of the plant	Moderately Resistant
7	Spots dark brown in colour covering 26 to 50 per cent of leaf area and covering one third of the plant	Susceptible
9	Spots uniformly dark brown, coalescing, covering 50 per cent or more leaf area and sever infection on all leaves	Highly Susceptible

Table 2. Rating scale (grade) used for Powdery Mildew disease severity (Mayee and Datar, 1986)

Rating Scale (Grade)	Disease severity	Level of resistance/ susceptibity
0	No visible symptom of powdery mildew on leaves	Immune
1	Small white specks on the leaf covering 1 % or less of the leaf area	Highly Resistant
3	Small Powdery patches covering 1-10 % of the leaf area	Resistant
5	Powdery lesions enlarged covering 11-25 % of the leaf area.	Moderately Resistant
7	Powdery patches irregular in size covering 26-50 % of the leaf area.	Susceptible
9	Big white powdery growth covering more than 50 % or more of leaf area and defoliation occurs.	Highly Susceptible

Table 3. Rating scale (grade) used for Leaf curl disease severity (Friedmann *et al.*, 1998)

Rating scale (Grade)	Disease severity
0	Symptoms absent
1	very slight yellowing of leaflet margins on apical leaf
2	some yellowing and minor curling of leaflet ends
3	a wide range of leaf yellowing, curling and cupping, with some reduction in size, yet plants continue to develop
4	very severe plant stunting and yellowing, pronounced leaf cupping and curling, and plant growth stops

Further these scales were converted to per cent disease severity using formula given by Ayyangar (1928):

$$\text{Disease severity (\%)} = \frac{\text{Sum of all numerical ratings}}{\text{Total number of observations} \times \text{Maximum rating grade}} \times 100$$

The area under disease progress curve was calculated by using the formula given by Johnson and Wilcoxson 1982.

$$\text{AUDPC} = \sum_{i=1}^{N_i - 1} \left(\frac{Y_{i+1} + Y_i}{2(X_{i+1} - X_i)} \right)$$

Where,

AUDPC = Area under disease progress curve,

Yi = Severity at i^{th} observation,

Xi = Time (days) at i^{th} observation,

N = Total number of observations.

3.3.2 Studies on anatomical changes in tomato leaf due to application of RIC

The leaf samples treated with the RIC's were collected after 10 days of treatment and were cut into 0.5 x 1 cm rectangle segments

including midrib portion. This was used for micrometry. The thin sections of 10 µm thickness were obtained, mounted under DPX and observed under the microscope.

Observations on the thickness of cuticle and palisade mesophyll were recorded with the help of ocular and stage micrometer.

3.3.3 Studies on biochemical changes in RIC treated and non-treated tomato plant

3.3.3.1 Soluble proteins

Soluble protein content was determined by the method of Lowry *et al.* (1951).

Reagents

1. Alkaline copper tartarate solution: It was prepared on the day of use by mixing 50 ml of solution A with 1 ml of solution B.

Solution A. Alkaline sodium carbonate solution (2% w/v): It was prepared by dissolving 2 g Na_2CO_3 in 100 ml of 0.1 N NaOH solution.

Solution B. Copper sulphate (0.5 %): It was freshly prepared by dissolving 0.5 g $CuSO_4.5H_2O$ in 1 per cent Na-K tartarate solution.

1. Folin-Ciocalteau reagent : It was prepared by diluting commercial grade reagent with equal volume of water on the day of use.
2. Standard protein solution : It was prepared by dissolving 100 mg bovine serum albumin in 100 ml distilled water. From the stock solution , 10 ml solution was diluted to 100 ml with distilled water. The solution contained 100 µg ml^{-1} of bovine serum albumin.

Extraction

0.25 g tomato leaf sample of each treatment was weighed and macerated separately in a mortar and pestle in 10 ml of water and centrifuged at 10000 x g for 20 min. the supernatant obtained was used for protein estimation.

Procedure

One ml of test solution after dilution was mixed with 50 ml of alkaline solution and kept for 10 min at room temperature. Folin-Ciocalteau reagent (0.5 ml) was rapidly added with immediate mixing and the colour intensity was measured after 30 min at 660 nm against blank. The protein content was calculated from the standard curve prepared by using various concentrations of BSA.

Calibration of standard curve

An aliquot of 0.0,0.1,0.2,0.3……..1.0 ml of working solution of BSA was pipette in a series of test tube in a triplicate set. The volume was made 1 ml with distilled water in each of these test tubes. The protein estimation procedure was followed as described above and the standard curve was prepared by plotting absorbance against concentration of BSA solution.

3.3.3.2 Reducing sugar

Reducing sugar content was determined by Nelson Somogyi's method (Somogyi, 1952).

Reagents

1. Reagent 'A': This contained 2.5 g anhydrous sodium carbonate, 2.5 g Rochelle salt, 2.0 g sodium bicarbonate and 20 g anhydrous sodium sulphate in a final volume of 100 ml distilled water.
2. Reagent 'B': Exactly 15 g of copper sulphate was dissolved in distilled water containing 1-2 drops of concentrated sulphuric acid and the volume was made to 100 ml.
3. Alkaline copper tartarate reagent: It was a mixture of 25 parts of reagent 'A' and 1 part of reagent 'B' (v/v). a freshly mixed reagent was used for analysis.
4. Arsenomolybdate reagent: this was prepared by dissolving 25 g of ammonium molybdate in 450 ml distilled water. To this 25 ml of

concentrated sulphuric acid was added. 3 g of sodium arsenate was dissolved in 25 ml distilled water separately and poured in to above solution. The final volume was made to 500 ml with distilled water. This reagent was incubated at 37^0C for 24-48 hr and then used for colour development.

5. Standard D-glucose solution : 100 mg of D-glucose was dissolved in 100 ml distilled water.
6. Working standard D- glucose solution: 10 ml of the stock solution was pipette out into a 100 ml volumetric flask and diluted to 100 ml with distilled water. This working solution contained 100 µg ml^{-1} of glucose.

Extraction

0.25 g of tomato leaf samples weighed and macerated in mortar and pestle in 10 ml of 80 % alcohol and centrifuged at 10000 x g for 15 min. The supernatant thus obtained was used as a enzyme extract for estimation of reducing sugar.

Colour development

One ml of enzyme extract was pipette in a test tube and 1 ml of alkaline copper tartarate reagent was added to it. The contents were mixed and heated for 10 min in boiling water bath. After cooling, 1 ml of arsenomolybdate reagent was added and the contents were diluted to 8 ml by adding 5 ml distilled water and the intensity of the colour was read at 520 nm.

Calibration of standard curve

An aliquot of 0.0,0.1,0.2,0.3……..1.0 ml of working solutionof D-glucose was pipette in a series of test tubes in triplicate. The volume was made to 1 ml with distilled water in each of these test tubes. The sugar estimation procedure was followed as described above and the

standard curve was prepared by plotting absorbance against concentration of D-glucose solution.

3.3.3.3 Polyphenols

Total polyphenol content was determined by using Folin-Denis reagent as described by Swain and Hills (1959).

Reagents

1. Folin-Denis reagent: Sodium tungstate (100 g) and phosphomolybdic acid (20 g) were dissolved in 750 ml water, to which 50 ml of conc. phosphoric acid was added. The contents were refluxed for 2 hr and the volume was made to 1000 ml.
2. Alkaline reagent: This was prepared by dissolving 350 g of sodium carbonate in 1000 ml water at 80^0 C. The solution was allowed to stand overnight and filtered through glass wool.
3. Standard tannic acid solution (0.1 %, w/v): 100 mg of tannic acid was dissolved in 100 ml of distilled water. Then 10 ml of this solution was diluted to 100 ml with distilled water. 1 ml of this solution contained 100 µg of tannic acid.

Extraction of plant phenols

0.25 g of tomato leaf sample of each treatment was separately macerated in a mortar and pestle in 10 ml of water and phenols were extracted by boiling in hot water bath. The contents were centrifuged at 10,000 x g for 15 min. The extraction was repeated two more times and the supernatant was diluted to 10 ml.

Colour development

1 ml of extract was mixed with 7 ml of water and 0.25 ml Folin-Denis reagent and kept for 10 min. After 10 min 1 ml of the alkaline reagent was added in each tube and the contents were mixed thoroughly. After 20 min, the contents were made to 10 ml and the extinction was measured at 650 nm on a Spectronic-20.

Calibration of a standard curve

It was prepared by using 0,10,20,30.....100 µg of tannic acid concentrations corresponding to 0, 0.1, 0.2, 0.3.....1 ml of standard solution in separate test tubes in duplicate. The phenol estimation procedure was followed as described above for colour development. The concentration of total phenolics was calculated from a standard curve and expressed as mg 100 g^{-1} on a fresh weight basis.

3.3.3.4 Peroxidase activity (POD)

The peroxidase activity in the leaf sample was assayed by the method of Kumar and Khan (1982).

Reagents

1. 0.1 M phosphate buffer (pH 7.0): It was prepared by mixing 47.8 ml of 0.2 M $NaH_2PO_4.2H_2O$ solution and 76.3 ml 0.2 M $Na_2HPO_4.2H_2O$. The pH was adjusted to 7.0 and the final volume was made to 250 ml.
2. Pyrogallol reagent (0.01 M): It was prepared by dissolving 0.126 g of pyrogallol in 100 ml of distilled water.
3. Hydrogen peroxide solution (0.005 M): 103 µl of 30% (v/v) hydrogen peroxide was pipetted in a 100 ml volumetric flask and the volume was made to 100 ml with distilled water. From this stock solution (1 M), 0.5 ml was pipetted in 100 ml volumetric flask and the volume was made to 100 ml with distilled water. This solution had the concentration of 0.005 M. The solution was prepared freshly at the time of experiment.

Procedure

0.25 g of tomato leaf samples was macerated separately with 6 ml of 0.1 M phosphate buffer in pre-chilled mortar and pestle. The homogenate was centrifuged at 15,000 x g at 4^0C for 30 min. 1 ml supernatant was diluted to 10 ml with distilled water and was used as

the enzyme source. The assay mixture of peroxidase contained 3.6 ml of 0.1 M phosphate buffer (pH 7.0), 1 ml of 0.005 M hydrogen peroxide, 1 ml of 0.01 M pyrogallol and 1 ml of well diluted enzyme extract. The absorbance was read at 420 nm on a Spectronic-20 Spectrophotometer for every 30 sec. up to 3 min and the reaction was stopped by adding 2.5 N H_2SO_4 exactly after 3 min.

One unit of peroxidase activity was determined as an increase in O.D. by 0.001. The enzyme activity was calculated for one g of sample and expressed as unit's $min^{-1}mg^{-1}$ soluble protein.

3.3.3.5 Polyphenol oxidase activity (PPO)

The polyphenol oxidase activity in the leaf sample was assayed by the method of Kumar and Khan (1982).

Reagents

1. 0.1 M phosphate buffer (pH 7.0): It was prepared by mixing 47.8 ml of 0.2 M $NaH_2PO_4.2H_2O$ solution and 76.3 ml 0.2 M $Na_2HPO_4.2H_2O$. The pH was adjusted to 7.0 and the final volume was made to 250 ml.
2. Pyrogallol reagent (0.01 M): It was prepared by dissolving 0.126 g of pyrogallol in 100 ml of distilled water.

Procedure

The enzyme extract was prepared as described under the assay of peroxidase and was used as the enzyme source. The assay mixture of polyphenol oxidase contained 2.0 ml of 0.1 M phosphate buffer (pH 7.0), 1 ml of 0.01 M pyrogallol and 1 ml of well diluted enzyme extract. The absorbance was read at 420 nm on a Spectronic-20 Spectrophotometer for every 30 sec up to 3 min and the reaction was stopped by adding 2.5 N H_2SO_4 exactly after 3 min.

One unit of polyphenol oxidase activity was calculated as the change in absorbance by $min^{-1}mg^{-1}$ soluble protein.

3.3.3.6 Phenylalanine ammonia lyase activity

Phenylalanine ammonia lyase activity was assayed by Campos *et al*. (2004).

Reagents

1. 0.05 M borate buffer (pH 8.5) containing 5 mM 2-mercaptoethanol and 0.4 % (w/v) PVP: Exactly 1.545 g of boric acid and 1.825 g sodium hydroxide were weighed, dissolved separately and mixed together. The pH was adjusted to 8.5 and the volume was made to 400 ml with distilled water. Then 2 g of insoluble polyvinyl pyrrollidone and 0.18 ml 2-mercaptoethanol were mixed in it and the final volume was made to 500 ml with distilled water.
2. 100 mM L-phenylalanine (1.66 g) was dissolved in distilled water and the volume was made to 100 ml.
3. TCA (4 % w/v): Four mg of trichloroacetic acid was dissolved in distilled water and the volume was made to 100 ml.

Procedure

Fresh leaf samples (0.25 g) was macerated separately with 6 ml of 0.05 M borate buffer (pH 8.5) containing 2-mercaptoethanol and in soluble polyvinyl pyrrolidone in pre-chilled mortar and pestle and was used as an enzyme source.

The assay mixture containing 1 ml aliquots of supernatant and 110 µl of 100 mM L-phenylalanine were incubated at 40^0c for 30 min. Then 1 ml of 4 % TCA was added in it to terminate the reaction. Similarly the TCA was added in one of the test tube at 0 min to serve as a blank. The assay mixture was incubated with TCA for 5 min at room temperature and centrifuged at 10,000 x g for 5 min and the absorbance of the supernatant was read at 290 nm.

PAL activity was calculated as µ moles of cinnamic acid produced g^{-1} soluble protein min^{-1} under the specific condition.

3.3.3.7 β-1, 3-glucanase activity

The assay of β-1, 3-glucanase was carried out as per the method described by Rashkit *et al.* (2000).

Reagents

1. 0.2 M Tris-HCl buffer (pH 7.5): 50 ml of Tris solution was mixed with 40 ml of 0.4 M HCl and the volume made to 100 ml with distilled water.
2. 333330.1 M potassium acetate buffer (pH 5.2): 50 ml of 0.1 M acetic acid was mixed with 79 ml of 0.1 M potassium acetate.
3. Laminarin (0.1 % w/v): 0.1 % in 0.1 M potassium acetate buffer, pH 5.2.

Procedure

The fresh tomato leaf samples (0.25 g) were macerated with chilled 6 ml of 0.2 M Tris-HCl buffer (pH 7.5) in pre-chilled mortar and pestle. The homogenate was centrifuged at 11,000 x g at 4^0C for 30 min and the supernatant was used as the enzyme source of crude enzyme. One ml of reaction mixture contained 950 µl of laminarin and 50 µl of crude enzyme extract, was incubated at 37^0C for 30 min and the reducing sugar released in to the solution at the end of reaction was estimated by Nelson-Somogyi's method (Somogyi, 1952). The protein content in the crude enzyme extract was estimated according to the method of Lowry *et al.* (1951).

The β-1, 3-glucanase activity was expressed as mg of glucose released g^{-1} soluble protein min^{-1}.

3.3.3.8 Chitinase activity

The chitinases activity was estimated according to the methods described by Thimmaiah (1999) and Giri *et al.* (1998).

Reagents

1. 0.1 M sodium citrate buffer (pH 5.0): 70 ml of 0.1 M citric acid and 130 ml of 0.1 M sodium citrate were mixed together.
2. Colloidal chitin : Chitin (1.83 mg) was dissolved in 1 ml of sodium acetate buffer (pH 5.2).

3. 0.1 M sodium acetate buffer (pH 5.2): 2.1 ml of 0.1 M acetic acid and 7.9 ml of 0.1 M sodium acetate were mixed together.
4. Standard 0.4 M N-acetyl glucosamine solution- 100 mg of N-acetyl glucosamine dissolved in 100 ml water which acts as a stock solution. From this stock solution 10 ml was diluted to 100 ml with distilled water. 1 ml of this working solution contains 100 µg of N-acetyl glucosamine.

Procedure

Fresh leaf samples (0.25 g) were macerated with 6 ml of 0.1 M sodium citrate buffer in pre-chilled mortar and pestle. The homogenate was centrifuged at 10,000 x g for 10 min at 10^0C and the supernatant was used as crude source of chitinase.

For the assay, 1 ml of supernatant, 4 ml of chitin suspension containing 15 mg of BSA were boiled in water bath at 37^0C for 3 hr. One ml water and one ml reaction mixture was boiled in centrifuge tube for 10 min and was centrifuged. An aliquot of 0.5 ml was taken for the estimation of N-acetylglucosamine as per the method of Nelson-Somogyi's (Somogyi, 1952). The protein content in the crude enzyme extract was estimated according to the method of Lowry *et al.* (1951).

The chitinase activity was expressed as mg of N-acetylglucosamine released g^{-1} soluble protein min^{-1}.

3.3.3.9 Lignin content

Lignin content in the powdered leaf sample was determined by the method of Van Soet (1963).

Reagents

1. Acid detergent solution: 20 g acetyl trimethyl bromide solution was dissolved in 1000 ml of 1 N H_2SO_4.
2. Decahydronaphthalene (R garde)
3. Acetone
4. 72 % H_2SO_4 by weight – 61 g of H_2SO_4 was dissolved in distilled water and final volume was made to 100 ml.

Procedure

One g of leaf sample was accurately weighed and transferred to a beaker refluxing apparatus. To this 100 ml of acid detergent solution was added and heated upto boiling for 10 min. Heat was reduced to avoid foaming. From the onset of boiling, it was refluxed for 60 min. It was filtered through previously weighed glass funnel by light suction. Two washings with hot water (90-100^{0}C) and acetone were given and then dried at 100^{0}C for 8 hr, cooled in a desiccator.

The residue left was placed in sintered glass crucibles in a tray and 72 % H_2SO_4 was added to these crucibles to break the lumps with a glass rod. After draining the acid, crucibles were again filled with acid and kept at 20-23^{0}C for 3 hr. Crucible were subjected to light suction. Then the contents were washed with hot water (95-100^{0}C) to make it free of sintered acid. The sintered glass crucibles were dried for 8 hr, cooled in a desiccator.

The residue left in the crucible was ignited in a muffle furnace at 50^{0}C for 3 hr, it was then cooled first in a desiccator and weighed.

Calculation

$$\text{Lignin (\%)} = \frac{\text{(Wt. of crucible and lignin) – (Wt. of crucible and ash)}}{\text{Weight of sample}} \times 100$$

3.4 Statistical analysis

The statistical analysis will be done as per the methods given by Panse and Sukhatme (1985).

4. EXPERIMENTAL RESULTS

Studies on the efficacy of resistance inducing chemicals against tomato diseases were undertaken during 2013-14 to 2014-15. The results obtained on all these aspects are being interpreted under following sub-heads.

4.1 Studies on effect of RIC's on diseases of Tomato

The results obtained from the effect of different RIC's on diseases of tomato var. *Dhanashree* have been presented under the following headings. There are four major diseases observed in field during field trial *viz.,* Early blight, Powdery mildew, Leaf curl virus disease and tomato spotted wilt virus disease of tomato (Plate 1).

4.1.1 Effect of RIC's on Early blight disease incidence of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatments on early blight disease incidence during *kharif* 2014 and 2015 crop season is presented in Table 4 and Table 5.

The data on percent disease incidence of early blight during *kharif* 2014 and *kharif* 2015 was recorded periodically from 30 to 105 days after transplanting (DAT) with an interval of 15 days (Table 4 and 5). It has been found that in all treatments percent disease incidence increased with age of the plants *i.e*. from 30 DAT to 105 DAT. However the per cent disease incidence were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence increase was comparatively low in case of RIC's treated plots over control. Data given in Table 4 and 5 revealed that incidence of early blight of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 30, 45, 60, 75, 90 and 105 DAT. Data on disease incidence also showed that among all RIC's tested, treatment T_5 (SA-S+SL+SPY) reduced the disease incidence significantly compared to control and showed the least disease incidence from 30 DAT to 105 DAT.

***Kharif* 2014**

Data recorded during *Kharif* 2014 indicated that effect of RIC's treatments on incidence of early blight disease of tomato was significant up to 105 DAT. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease incidence reduction in different treatments varied from 7.09 to 20.16 per cent in comparison with control. The incidence of early blight disease was significantly minimum in plots treated with treatment T_5 (SA-S+ SL + SPY) at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease incidence was continuously observed in unsprayed control plot.

At 30 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e.10.68 per cent which was at par with treatment T_3 (SA-SPY) i.e. 11.06 per cent, T_4 (SA-S+SL) i.e. 11.24 per cent, T_{15} (CHT-S+SL+SPY) i.e. 11.29 per cent, T_2 (SA-SL) and T_{13} (CHT-SPY) i.e. 11.41 per cent. Whereas the maximum disease incidence was observed in T_{16} i.e. control (12.20 %). In rest of the treatments the disease incidence was ranged from 11.64 (T_1) to 12.19 (T_6) per cent.

At 45 DAT, Tomato plants treated with treatment T_5 (SA-S+SL+SPY) exhibited least disease incidence i.e. 21.43 per cent which was at par with treatment T_3 (SA-SPY) i.e. 22.09 per cent, treatment T_4 (SA-S+SL) i.e. 22.62 per cent followed by treatment T_{15} (CHT-S+SL+SPY) i.e. 22.73 per cent against untreated control i.e. 24.76 per cent.

The percent disease incidence in different treatments at 60, 75, 90 and 105 days after transplanting (DAT) demonstrated that the treatment T_5 (SA-S+SL+SPY) was significantly superior over rest of the treatment except treatment T_3 (SA-SPY). However the remaining treatments performed comparatively better than control.

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 70.15 per cent. Whereas the

Table 4. Effect of RIC's on Early blight disease incidence of tomato *var. Dhanashree* during *Kharif* 2014

Tr. No.	Treatment	Early blight disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	11.64 (19.896)	23.47 (28.935)	46.04 (42.700)	60.46 (51.048)	71.25 (57.632)	78.63 (62.588)	10.51
T_2	SA-SL	11.41 (19.629)	22.90 (28.547)	45.44 (42.358)	59.12 (50.253)	69.67 (56.609)	75.52 (60.399)	14.05
T_3	SA-SPY	11.06 (19.306)	22.09 (27.963)	43.65 (41.327)	57.15 (49.100)	67.34 (55.150)	72.73 (58.536)	17.22
T_4	SA-S+SL	11.24 (19.472)	22.62 (28.330)	44.68 (41.925)	58.43 (49.839)	68.96 (56.136)	74.60 (59.738)	15.09
T_5	SA-S+SL+SPY	10.68 (19.016)	21.43 (27.501)	42.25 (40.523)	55.12 (47.934)	65.16 (53.809)	70.15 (56.868)	20.16
T_6	BABA-S	12.19 (20.385)	24.70 (29.762)	48.49 (44.113)	63.42 (52.805)	75.35 (60.319)	81.63 (64.790)	7.09
T_7	BABA-SL	12.14 (20.287)	24.38 (29.548)	48.04 (43.855)	63.04 (52.566)	74.35 (59.619)	80.76 (64.080)	8.08
T_8	BABA-SPY	11.88 (20.055)	23.81 (29.142)	47.21 (43.380)	61.49 (51.637)	73.36 (58.944)	78.93 (62.717)	10.16
T_9	BABA-S+SL	12.01 (20.171)	24.20 (29.405)	47.57 (43.588)	62.17 (52.032)	73.94 (59.304)	79.10 (62.808)	9.97
T_{10}	BABA-S+SL + SPY	11.68 (19.932)	23.48 (28.918)	46.00 (42.687)	60.40 (51.013)	71.81 (57.917)	77.26 (61.509)	12.06
T_{11}	CHT-S	11.98 (20.199)	24.11 (29.393)	47.52 (43.554)	61.98 (51.947)	74.23 (59.571)	79.83 (63.452)	9.14
T_{12}	CHT-SL	11.76 (19.947)	23.69 (29.084)	46.53 (42.986)	60.89 (51.292)	72.04 (58.113)	78.13 (62.190)	11.07
T_{13}	CHT-SPY	11.41 (19.629)	23.01 (28.598)	45.44 (42.361)	58.74 (50.025)	70.01 (56.804)	75.45 (60.324)	14.12
T_{14}	CHT-S+SL	11.71 (19.902)	23.50 (28.932)	46.09 (42.738)	60.56 (51.084)	71.15 (57.509)	77.24 (61.513)	12.09
T_{15}	CHT-S+SL + SPY	11.29 (19.579)	22.73 (28.405)	44.82 (42.008)	58.22 (49.735)	68.85 (56.059)	74.60 (59.723)	15.09
T_{16}	Control	12.20 (20.341)	24.76 (29.817)	48.96 (44.385)	65.15 (53.846)	79.04 (62.938)	87.86 (69.990)	0.00
	S.E. $\pm$	0.261	0.356	0.469	0.414	0.650	0.728	--
	C.D. at 5 %	0.757	1.003	1.361	1.201	1.887	2.113	--

*: Mean of three replications, Figures in parentheses are angular transformed values

maximum disease incidence was continuously observed in T_{16} i.e. Control (87.86 %). In rest of the treatments the percent disease incidence was ranged from 72.73 (T_3) to 81.63 (T_6) per cent. The percentage disease reduction was highest recorded in T_5 i.e. SA-S+SL+SPY (20.16 %) followed by T_3 (17.22 %).

***Kharif* 2015**

The data recorded on percent disease incidence of early blight on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 5) followed the same pattern as recorded during previous crop season. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease incidence reduction in different treatments varied from 7.56 to 21.50 per cent in comparison with control.

Result also indicated that the effect of RIC's treatments on early blight disease incidence of tomato was significant up to 105 DAT. The incidence of early blight disease was significantly minimum in plots treated with treatment T_5 (SA-S+SL+SPY) which was at par with treatment T_3 (SA-SPY) at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease incidence was continuously observed in unsprayed control plot.

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 64.65 per cent which was at par with treatment T_3 of SA-SPY (67.23 %). Whereas the maximum disease incidence was continuously observed in T_{16} i.e. Control (82.36 %). In rest of the treatments the percent disease incidence was ranged from 69.10 (T_4) to 76.13 (T_6) per cent. The percentage disease reduction was highest recorded in T_5 i.e. SA- S+SL+SPY (21.50 %) followed by T_3 (18.37 %).

Table 5. Effect of RIC's on Early blight disease incidence of tomato *var. Dhanashree* during *Kharif* 2015

Tr. No.	Treatment	Early blight disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	9.44 (17.622)	20.27 (26.635)	39.84 (39.092)	55.26 (48.002)	66.05 (54.346)	73.13 (58.764)	11.21
T_2	SA-SL	9.21 (17.384)	19.70 (26.321)	39.24 (38.767)	53.92 (47.244)	64.47 (53.452)	70.02 (56.878)	14.98
T_3	SA-SPY	8.86 (17.016)	18.89 (25.671)	37.45 (37.699)	51.95 (46.101)	62.14 (52.021)	67.23 (55.082)	18.37
T_4	SA-S+SL	9.04 (17.336)	19.42 (26.094)	38.48 (38.316)	53.23 (46.835)	63.76 (52.976)	69.10 (56.223)	16.10
T_5	SA-S+SL+SPY	8.48 (16.751)	18.23 (25.217)	36.05 (36.855)	49.92 (44.936)	59.96 (50.745)	64.65 (53.530)	21.50
T_6	BABA-S	9.99 (18.178)	21.50 (27.514)	42.29 (40.526)	58.22 (49.714)	70.15 (56.868)	76.13 (60.742)	7.56
T_7	BABA-SL	9.94 (18.128)	21.18 (27.374)	41.84 (40.285)	57.84 (49.522)	69.15 (56.330)	75.26 (60.300)	8.62
T_8	BABA-SPY	9.68 (17.867)	20.61 (26.920)	41.01 (39.793)	56.29 (48.602)	68.16 (55.654)	73.43 (58.990)	10.84
T_9	BABA-S+SL	9.81 (18.110)	21.00 (27.227)	41.37 (40.008)	56.97 (48.992)	68.74 (56.000)	73.60 (59.082)	10.64
T_{10}	BABA-S+SL + SPY	9.48 (17.782)	20.28 (26.715)	39.80 (39.078)	55.20 (47.976)	66.61 (54.717)	71.76 (57.933)	12.87
T_{11}	CHT-S	9.78 (17.968)	20.91 (27.095)	41.32 (39.960)	56.78 (48.896)	69.03 (56.170)	74.33 (59.546)	9.75
T_{12}	CHT-SL	9.56 (17.745)	20.49 (26.886)	40.33 (39.405)	55.69 (48.270)	66.84 (54.896)	72.63 (58.554)	11.81
T_{13}	CHT-SPY	9.21 (17.384)	19.81 (26.344)	39.24 (38.757)	53.54 (47.015)	64.81 (53.614)	69.95 (56.767)	15.07
T_{14}	CHT-S+SL	9.51 (17.812)	20.30 (26.729)	39.89 (39.144)	55.36 (48.061)	65.95 (54.292)	71.74 (57.884)	12.89
T_{15}	CHT-S+SL + SPY	9.09 (17.387)	19.53 (26.174)	38.62 (38.383)	53.02 (46.718)	63.65 (52.930)	69.10 (56.253)	16.10
T_{16}	Control	10.00 (18.296)	21.56 (27.592)	42.76 (40.800)	59.95 (50.748)	73.84 (59.313)	82.36 (65.349)	0.00
	S.E. $\pm$	0.301	0.422	0.507	0.575	0.638	0.697	--
	C.D. at 5 %	0.874	1.223	1.470	1.667	1.850	2.022	--

* : Mean of three replications, Figures in parentheses are angular transformed values

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on disease incidence presented in Table 6 revealed that the data followed the same pattern as recorded during *kharif* 2014 and *kharif* 2015 crop season. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all the pooled treatments the percent disease incidence increased with age of the plants *i.e.* from 30 DAT to 105 DAT. However the per cent disease incidence were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence increase was comparatively low in case of RIC's treated plots over control. The disease incidence was observed increase constantly throughout the crop growth period in control as well as in other treatments also, whereas magnitude of increase in RIC's treatment was less over control.

Pooled result also indicated that the effect of RIC's treatments on early blight disease incidence of tomato was significant up to 105 DAT. The incidence of early blight disease was significantly minimum in plots treated with treatment T_5 (SA-S+SL+SPY) which was at par with treatment T_3 (SA-SPY) at 30, 45, 60, 75 and 90 DAT, whereas maximum disease incidence was continuously observed in unsprayed control plot.

At 30 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 9.57 per cent which was at par with treatment T_3 (SA-SPY) i.e. 9.96 per cent, T_4 (SA-S+SL) i.e. 10.14 per cent, T_{15} (CHT-S+SL+SPY) i.e. 10.19 per cent and T_2 (SA-SL) i.e. 10.30 per cent. Whereas the maximum disease incidence was observed in T_{16} i.e. Control (11.10 %). In rest of the treatments the disease incidence was ranged from 10.31 (T_{13}) to 11.09 (T_6) per cent (Table 6).

The pooled result of percent disease incidence in different treatments at 60, 75, 90 and 105 days after transplanting (DAT)

Table 6. Pooled data on effect of RIC's on Early blight disease incidence of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Early blight disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	10.54 (18.817)	21.87 (27.809)	42.94 (40.905)	57.86 (49.512)	68.65 (55.957)	75.88 (60.612)	10.84
T_2	SA-SL	10.30 (18.550)	21.30 (27.449)	42.34 (40.570)	56.52 (48.745)	67.07 (55.016)	72.77 (58.612)	14.50
T_3	SA-SPY	9.96 (18.207)	20.49 (26.834)	40.55 (39.525)	54.55 (47.597)	64.74 (53.572)	69.98 (56.786)	17.78
T_4	SA-S+SL	10.14 (18.432)	21.02 (27.227)	41.58 (40.130)	55.83 (48.333)	66.36 (54.541)	71.85 (57.954)	15.58
T_5	SA-S+SL+SPY	9.57 (17.926)	19.83 (26.376)	39.15 (38.707)	52.52 (46.432)	62.56 (52.266)	67.40 (55.180)	20.81
T_6	BABA-S	11.09 (19.333)	23.10 (28.659)	45.39 (42.325)	60.82 (51.243)	72.75 (58.549)	78.88 (62.681)	7.32
T_7	BABA-SL	11.04 (19.245)	22.78 (28.475)	44.94 (42.074)	60.44 (51.037)	71.75 (57.953)	78.01 (62.149)	8.34
T_8	BABA-SPY	10.78 (19.000)	22.21 (28.046)	44.11 (41.593)	58.89 (50.112)	70.76 (57.277)	76.18 (60.815)	10.49
T_9	BABA-S+SL	10.91 (19.166)	22.60 (28.329)	44.47 (41.804)	59.57 (50.504)	71.34 (57.629)	76.35 (60.907)	10.29
T_{10}	BABA-S+SL + SPY	10.58 (18.892)	21.88 (27.831)	42.90 (40.895)	57.80 (49.486)	69.21 (56.297)	74.51 (59.688)	12.45
T_{11}	CHT-S	10.87 (19.138)	22.50 (28.279)	44.42 (41.764)	59.38 (50.414)	71.63 (57.829)	77.08 (61.428)	9.43
T_{12}	CHT-SL	10.66 (18.886)	22.09 (28.000)	43.43 (41.202)	58.29 (49.776)	69.44 (56.487)	75.38 (60.339)	11.43
T_{13}	CHT-SPY	10.31 (18.550)	21.41 (27.487)	42.34 (40.568)	56.14 (48.515)	67.41 (55.192)	72.70 (58.516)	14.58
T_{14}	CHT-S+SL	10.61 (18.883)	21.90 (27.845)	42.99 (40.949)	57.96 (49.566)	68.55 (55.882)	74.49 (59.665)	12.48
T_{15}	CHT-S+SL + SPY	10.19 (18.521)	21.13 (27.305)	41.72 (40.210)	55.62 (48.221)	66.25 (54.479)	71.85 (57.961)	15.58
T_{16}	Control	11.10 (19.343)	23.16 (28.724)	45.86 (42.600)	62.55 (52.286)	76.44 (61.081)	85.11 (67.559)	0.00
	S.E. ±	0.206	0.229	0.358	0.406	0.464	0.523	--
	C.D. at 5 %	0.598	0.665	1.040	1.179	1.347	1.518	--

* : Mean of three replications, Figures in parentheses are angular transformed values

demonstrated that the treatment T_5 (SA-S+SL+SPY) was significantly superior over rest of the treatments except treatment T_3 (SA-SPY). However the remaining treatments performed comparatively better than control.

At 105 DAT, minimum pooled disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 67.40 per cent which was significantly superior over other. Whereas the maximum pooled disease incidence was continuously observed in T_{16} i.e. Control (85.11 %). In rest of the treatments the pooled percent disease incidence was ranged from 69.98 (T_3) to 78.88 (T_6) per cent. The percent disease reduction was highest recorded in T_5 i. e. SA-S+SL+SPY (20.81 %) followed by T_3 (17.78 %).

A perusal of the data of the effect of RIC's on early blight incidence under field conditions indicated that the different treatments has effect on early blight disease incidence and considerable reduction in disease incidence was observed when the number of sprays was increased. Critical analysis of the data revealed that the salicylic acid was found effective in reducing the disease incidence as compared to other chemicals.

4.1.2 Effect of RIC's on early blight disease severity of tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different RIC's treatments on early blight disease severity indices during *kharif* 2014 and 2015 crop season is presented in Table 7 and Table 8.

The data on percent disease severity of early blight during *kharif* 2014 and *kharif* 2015 was recorded periodically from 30 to 105 days after transplanting (DAT) with an interval of 15 days (Table 7 and 8). It has been found that in all treatments percent disease severity increased with age of the plants. It was observed that disease severity was increased during 30 to 105 DAT in all treatments but the rate of percent disease severity increase was comparatively low in case of RIC's

treated plots. Data given in Table 7 and 8 revealed that severity of early blight of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 30, 45, 60, 75, 90 and 105 DAT. Data on disease severity also showed that among all RIC's tested, treatment T_5 (SA-S+SL+SPY) reduced the disease severity significantly compared to control and showed the least disease incidence from 45 DAT to 105 DAT.

***Kharif* 2014**

The data on percent disease severity during *kharif* 2014 (Table 7) demonstrated that severity of early blight disease of tomato *var. Dhanashree* was significantly differ due to different treatments of RIC's from 30 DAT to 105 DAT. However the severity of early blight disease was comparatively minimum in plots treated with treatment T_5 (SA-S+SL+SPY) at 45, 60, 75, 90 and 105 DAT, whereas at 30 DAT the percent disease severity was found minimum in treatment T_3 of (SA-SPY). However the treatment T_5 (SA-S+SL+SPY) was statistically at par with treatment T_3 (SA-SPY), treatment T_{15} (CHT-S+SL+SPY), treatment T_{13} (CHT-SPY), treatment T_{10} (BABA-S+SL+SPY), treatment T_2 (SA-SL) and treatment T_4 (SA-S+SL) at different period (from 30 DAT to 105 DAT) in their effectiveness against disease control. The remaining treatments performed comparatively less. Maximum disease severity was continuously observed in unsprayed control plot.

The data recorded at 105 DAT showed minimum disease severity i.e. 35.78 per cent in the plots protected with T_5 (SA-S+SL+SPY) treatment which was at par with T_3 (SA-SPY), T_{15} (CHT-S+SL+SPY) and T_4 (SA-S+SL) with disease severity 37.06, 37.48 and 37.91 per cent, respectively. While maximum disease severity 42.60 per cent was recorded in T_{16} i.e. unsprayed control plot. In rest of the treatments the per cent disease severity was ranged between 38.34 (T_{13}) to 41.32 (T_6) per cent and most of them were at par with each other. The percent disease severity reduction was found maximum in treatment T_5 i.e. SA-S+SL+SPY

Table 7. Effect of RIC's on Early blight disease severity of tomato *var. Dhanashree* during *Kharif* 2014

Tr. No.	Treatment	Early blight disease severity* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	4.24 (11.858)	9.99 (18.287)	16.69 (24.047)	26.24 (30.777)	33.59 (35.352)	39.19 (38.709)	8.00
T_2	SA-SL	3.89 (11.359)	9.59 (17.892)	16.19 (23.658)	25.64 (30.339)	32.84 (34.910)	38.34 (38.217)	10.00
T_3	SA-SPY	3.16 (10.216)	8.71 (16.855)	15.01 (22.718)	24.16 (29.401)	31.06 (33.840)	37.06 (37.468)	13.00
T_4	SA-S+SL	3.91 (11.382)	9.56 (17.862)	16.06 (23.589)	25.41 (30.247)	32.51 (34.742)	37.91 (37.979)	11.01
T_5	SA-S+SL+SPY	3.18 (10.266)	8.68 (16.823)	14.88 (22.673)	23.88 (29.238)	30.68 (33.619)	35.78 (36.719)	16.01
T_6	BABA-S	4.44 (12.149)	10.42 (18.702)	17.57 (24.721)	27.52 (31.606)	35.32 (36.416)	41.32 (39.960)	3.00
T_7	BABA-SL	4.37 (12.050)	10.29 (18.577)	17.34 (24.546)	27.24 (31.386)	34.94 (36.199)	40.89 (39.717)	4.01
T_8	BABA-SPY	4.19 (11.803)	10.04 (18.228)	16.94 (24.240)	26.69 (31.070)	34.24 (35.786)	40.04 (39.226)	6.01
T_9	BABA-S+SL	4.17 (11.762)	10.07 (18.364)	17.07 (24.370)	26.92 (31.232)	34.57 (35.993)	40.47 (39.483)	5.00
T_{10}	BABA-S+SL + SPY	3.79 (11.211)	9.59 (17.776)	16.39 (23.866)	26.04 (30.668)	33.49 (35.344)	39.19 (38.738)	8.00
T_{11}	CHT-S	4.39 (12.067)	10.27 (18.558)	17.22 (24.455)	27.02 (31.266)	34.62 (35.994)	40.47 (39.462)	5.00
T_{12}	CHT-SL	4.00 (11.524)	9.82 (18.120)	16.67 (23.987)	26.37 (30.819)	33.87 (35.552)	39.62 (38.972)	7.00
T_{13}	CHT-SPY	3.61 (10.946)	9.34 (17.519)	15.99 (23.501)	25.49 (30.285)	32.79 (34.905)	38.34 (38.226)	10.00
T_{14}	CHT-S+SL	4.12 (11.697)	9.89 (18.189)	16.64 (24.040)	26.24 (30.791)	33.54 (35.370)	39.19 (38.733)	8.00
T_{15}	CHT-S+SL + SPY	3.53 (10.814)	9.13 (17.301)	15.53 (23.192)	24.78 (29.839)	32.18 (34.545)	37.48 (37.730)	12.02
T_{16}	Control	4.45 (12.174)	10.45 (18.731)	17.60 (24.703)	27.60 (31.658)	35.60 (36.585)	42.60 (40.707)	0.00
	S.E. ±	0.374	0.340	0.424	0.510	0.576	0.503	--
	C.D. at 5 %	1.085	0.988	1.231	1.481	1.671	1.461	--

* : Mean of three replications, Figures in parentheses are angular transformed values

(16.01 %), T_3 i.e. SA-SPY (13.00 %) followed by T_{15} i. e. CHT-S+SL+SPY (12.02 %).

***Kharif* 2015**

The data recorded on percent disease severity of early blight on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 8) followed the same pattern as recorded during previous crop season. All the treatments comparatively reduced the disease severity over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease severity reduction in different treatments varied from 3.42 to 18.24 per cent in comparison with untreated control during crop growth period upto 105 DAT.

Result also indicated that the effect of RIC's treatments on early blight disease severity of tomato was significant up to 105 DAT. The severity of early blight disease was significantly minimum in plots treated with treatment T_5 of SA-S+SL+SPY which was at par with treatment T_3 of SA-SPY or vice versa at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease severity was continuously observed in unsprayed control plot.

The data recorded at 105 DAT showed minimum disease severity i.e. 30.58 per cent in the plots protected with T_5 (SA-S+SL+SPY) treatment which was at par with T_3 (SA-SPY), T_{15} (CHT-S+SL+SPY) and T_4 (SA-S+SL) with disease severity 31.86, 32.28 and 32.71 per cent, respectively. While maximum disease severity 37.40 per cent was recorded in T_{16} i.e. unsprayed control plot. In rest of the treatments the per cent disease severity was ranged between 33.14 (T_{13}) to 36.12 (T_6) per cent and most of them were at par with each other. The percent disease severity reduction was found maximum in treatment T_5 i.e. SA-S+SL+SPY (18.24 %), T_3 i.e. SA-SPY (14.81 %) followed by T_{15} i. e. CHT-S+SL+SPY (13.69 %).

Table 8. Effect of RIC's on Early blight disease severity of tomato *var. Dhanashree* during *Kharif* 2015

Tr. No.	Treatment	Early blight disease severity* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	2.04 (8.197)	6.79 (14.849)	10.49 (18.879)	21.04 (27.290)	28.39 (32.180)	33.99 (35.576)	9.12
T_2	SA-SL	1.69 (7.459)	6.39 (14.360)	9.99 (18.407)	20.44 (26.846)	27.64 (31.700)	33.14 (35.108)	11.39
T_3	SA-SPY	0.96 (5.610)	5.51 (13.212)	8.81 (17.237)	18.96 (27.758)	25.86 (30.551)	31.86 (34.335)	14.81
T_4	SA-S+SL	1.71 (7.502)	6.36 (14.322)	9.86 (18.273)	20.21 (26.697)	27.31 (31.488)	32.71 (34.864)	12.54
T_5	SA-S+SL+SPY	0.98 (5.678)	5.48 (13.171)	8.68 (17.124)	18.68 (25.593)	25.48 (30.302)	30.58 (33.511)	18.24
T_6	BABA-S	2.24 (8.593)	7.22 (15.356)	11.37 (19.676)	22.32 (28.178)	30.12 (33.268)	36.12 (36.867)	3.42
T_7	BABA-SL	2.17 (8.459)	7.09 (15.204)	11.14 (19.480)	22.04 (27.986)	29.74 (33.034)	35.69 (36.650)	4.57
T_8	BABA-SPY	1.99 (8.104)	6.84 (14.908)	10.74 (19.117)	21.49 (27.600)	29.04 (32.591)	34.84 (36.148)	6.84
T_9	BABA-S+SL	1.97 (8.054)	6.87 (14.944)	10.87 (19.222)	21.72 (27.762)	29.37 (32.799)	35.27 (36.413)	5.70
T_{10}	BABA-S+SL + SPY	1.59 (7.234)	6.39 (14.360)	10.19 (18.608)	20.84 (27.151)	28.29 (32.115)	33.99 (35.612)	9.12
T_{11}	CHT-S	2.19 (8.490)	7.07 (15.181)	11.02 (19.375)	21.82 (27.832)	29.42 (32.831)	35.27 (36.354)	5.70
T_{12}	CHT-SL	1.80 (7.698)	6.62 (14.643)	10.47 (18.851)	21.17 (27.378)	28.67 (32.354)	34.42 (35.885)	7.97
T_{13}	CHT-SPY	1.41 (6.807)	6.14 (14.045)	9.79 (18.223)	20.29 (26.754)	27.59 (31.668)	33.14 (35.119)	11.39
T_{14}	CHT-S+SL	1.92 (7.952)	6.69 (14.728)	10.44 (18.822)	21.04 (27.293)	28.34 (32.150)	33.99 (35.642)	9.12
T_{15}	CHT-S+SL + SPY	1.33 (6.614)	5.93 (13.774)	9.33 (17.775)	19.58 (26.246)	26.98 (31.259)	32.28 (34.566)	13.69
T_{16}	Control	2.25 (8.614)	7.25 (15.390)	11.40 (19.719)	22.40 (28.234)	30.40 (33.440)	37.40 (37.677)	0.00
	S.E. $\pm$	0.249	0.101	0.538	0.524	0.502	0.611	--
	C.D. at 5 %	0.721	0.293	1.561	1.520	1.457	1.772	--

* : Mean of three replications, Figures in parentheses are angular transformed values

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on disease severity presented in Table 9 revealed that the data followed the same pattern as recorded during previous crop season. All the treatments comparatively reduced the disease severity over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all treatments the pooled percent disease severity increased with age of the plants *i.e.* from 30 DAT to 105 DAT. However the per cent disease severity were considerably lower in RIC's treated plots. Also, the rate of percent disease severity increase was comparatively low in case of RIC's treated plots over control. The pooled disease severity ranged from 2.08 to 38.72 per cent throughout the crop growth period in comparison with 40.00 per cent recorded in control.

Result also indicated that the pooled early blight disease severity of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 30, 45, 60, 75, 90 and 105 DAT. However the pooled severity of early blight disease was significantly minimum in plots treated with treatment T_5 (SA-S+SL+SPY) which was at par with treatment T_3 (SA-SPY) and treatment T_{15} (CHT-S+SL+SPY) and showed the least disease incidence from 45 DAT to 105 DAT. Whereas maximum disease incidence was continuously observed in unsprayed control plot.

At 30 DAT the percent disease severity was found minimum in treatment T_3 of (SA-SPY) which was statistically at par with treatment T_5 (SA-S+SL+SPY), treatment T_{15} (CHT-S+SL+SPY) and treatment T_{13} (CHT-SPY) in their effectiveness against disease control. The remaining treatments performed comparatively better than control. Maximum disease severity was continuously observed in control plot (Unsprayed plot).

The pooled percent disease severity in different treatments at 45, 60, 75, 90 and 105 days after transplanting (DAT) demonstrated that the treatment T_5 (SA-S+SL+SPY) was significantly superior over rest of

Table 9. Pooled data on effect of RIC's on Early blight disease severity of Tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Early blight disease severity* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	3.14 (10.189)	8.39 (16.655)	13.59 (21.617)	23.64 (29.077)	30.99 (33.802)	36.59 (37.157)	8.52
T_2	SA-SL	2.79 (9.602)	7.99 (16.223)	13.09 (21.175)	23.04 (28.638)	30.24 (33.331)	35.74 (36.675)	10.65
T_3	SA-SPY	2.06 (8.226)	7.11 (15.136)	11.91 (20.174)	21.56 (27.657)	28.46 (32.224)	34.46 (35.915)	13.85
T_4	SA-S+SL	2.81 (9.635)	7.96 (16.190)	12.96 (21.088)	22.81 (28.520)	29.91 (33.139)	35.31 (36.434)	11.73
T_5	SA-S+SL+SPY	2.08 (8.287)	7.08 (15.101)	11.78 (20.064)	21.28 (27.460)	28.08 (31.986)	33.18 (35.138)	17.05
T_6	BABA-S	3.34 (10.513)	8.82 (17.108	14.47 (22.311)	24.92 (29.931)	32.72 (34.869)	38.72 (38.425)	3.20
T_7	BABA-SL	3.27 (10.404)	8.69 (16.973)	14.24 (22.139)	24.64 (29.728)	32.34 (34.638)	38.29 (38.193)	4.27
T_8	BABA-SPY	3.09 (10.117)	8.44 (16.642)	13.84 (21.808)	24.09 (29.370)	31.64 (34.209)	37.44 (37.698)	6.40
T_9	BABA-S+SL	3.07 (10.073)	8.47 (16.740)	13.97 (21.938)	24.32 (29.534)	31.97 (34.419)	37.87 (37.958)	5.32
T_{10}	BABA-S+SL + SPY	2.69 (9.427)	7.99 (16.149)	13.29 (21.374)	23.44 (28.944)	30.89 (33.752)	36.59 (37.192)	8.52
T_{11}	CHT-S	3.29 (10.421)	8.67 (16.952)	14.12 (22.044)	24.42 (29.591)	32.02 (34.439)	37.87 (37.921)	5.33
T_{12}	CHT-SL	2.90 (9.790)	8.22 (16.473)	13.57 (21.587)	23.77 (29.155)	31.27 (33.984)	37.02 (37.439)	7.45
T_{13}	CHT-SPY	2.51 (9.110)	7.74 (15.868)	12.89 (21.017)	22.89 (28.568)	30.19 (33.315)	35.74 (36.685)	10.65
T_{14}	CHT-S+SL	3.02 (9.991)	8.29 (16.548)	13.54 (21.558)	23.64 (29.079)	30.94 (33.778)	36.59 (37.199)	8.52
T_{15}	CHT-S+SL + SPY	2.43 (8.956)	7.53 (15.629)	12.43 (20.636)	22.18 (28.084)	29.58 (32.927)	34.88 (36.168)	12.80
T_{16}	Control	3.35 (10.539)	8.85 (17.139)	14.50 (22.330)	25.00 (29.980)	33.00 (35.031)	40.00 (39.199)	0.00
	S.E. ±	0.316	0.222	0.325	0.383	0.368	0.463	--
	C.D. at 5 %	0.918	0.645	0.944	1.112	1.067	1.342	--

* : Mean of three replications, Figures in parentheses are angular transformed values

the treatment except treatment T_3 (SA-SPY) and T_{15} (CHT-S+SL+SPY). However the remaining treatments performed comparatively better than control.

The data recorded at 105 DAT showed minimum pooled disease severity i.e. 33.18 per cent in the plots protected with T_5 (SA-S+SL+SPY) treatment which was at par with treatment T_3 (SA-SPY), treatment T_{15} (CHT-S+SL+SPY) and treatment T_4 (SA-S+SL) with disease severity 34.46, 34.88 and 35.31 per cent, respectively. While maximum pooled disease severity 40.00 per cent was recorded in T_{16} i.e. unsprayed control plot. In rest of the treatments the per cent disease severity was ranged between 35.74 (T_2) to 38.72 (T_6) per cent. The percent disease severity reduction was found maximum in treatment T_5 i.e. SA-S+SL+SPY (17.05 %), T_3 i.e. SA-SPY (13.85 %) followed by T_{15} i.e. CHT-S+SL+SPY (12.80 %).

A perusal of the data of the effect of RIC's on early blight severity under field conditions indicated that the different treatments has effect on early blight severity and considerable reduction in disease severity was observed when the number of sprays was increased. Critical analysis of the data revealed that the salicylic acid was found effective in reducing the disease severity as compared to other chemicals.

4.1.3 Effect of RIC's on Powdery mildew disease incidence of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatment on powdery mildew disease incidence during *kharif* 2014 and 2015 crop season is presented in Table 10 and Table 11.

The data on percent disease incidence of powdery mildew during *kharif* 2014 and *kharif* 2015 was recorded periodically from 30 to 105 days after transplanting (DAT) with an interval of 15 days (Table 10 and 11). It has been found that in all treatments percent disease incidence increased with age of the plants *i.e.* from 30 DAT to 105 DAT.

However the per cent disease incidence were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence increase was comparatively low in case of RIC's treated plots over control. Data given in Table 10 and 11 revealed that incidence of powdery mildew of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 45, 60, 75, 90 and 105 DAT. Data on disease incidence also showed that among all RIC's tested, treatment T_{10} (BABA-S+SL+SPY) reduced the disease incidence significantly compared to control and showed the least disease incidence at 105 DAT.

***Kharif* 2014**

Data recorded during *Kharif* 2014 indicated that effect of RIC's treatments on incidence of powdery mildew disease of tomato was significant up to 105 DAT. However the incidence of powdery mildew disease was significantly minimum in plots treated with treatment T_{10} (BABA-S+SL+ SPY) at 105 DAT, whereas maximum disease incidence was continuously observed in untreated control plot.

At 45 DAT, significantly minimum disease incidence was recorded in treatment T_4 (SA-S+SL) i.e. 9.05 per cent which was at par with treatment T_{11} (CHT-S) i.e. 9.65 per cent, T_5 (SA-S+SL) i.e. 9.50 per cent and T_{10} (BABA-S+SL+SPY) i.e. 10.23 per cent. Whereas the maximum disease incidence was observed in T_{16} i.e. control (19.83 %). In rest of the treatments the disease incidence was ranged from 12.26 (T_3) to 19.58 (T_8) per cent.

The percent disease incidence in different treatments at 60 and 75 days after transplanting (DAT) demonstrated that the treatment T_5 (SA- S+SL+SPY) was significantly superior over rest of the treatment except treatment T_{11} (CHT-S) and T_{10} (BABA-S+SL+SPY). However the remaining treatments performed comparatively better than control.

At 90 DAT, significantly minimum disease incidence was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 56.58 per cent followed by T_5 (SA-S+SL+SPY) i.e. 57.41 per cent and T_{15} (CHT-S+SL+SPY) i.e.,

Table 10. Effect of RIC's on Powdery mildew disease incidence of tomato *var. Dhanashree* during *Kharif* 2014

Tr. No.	Treatment	Powdery mildew disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	12.84 (20.739)	30.45 (33.430)	47.73 (43.677)	67.83 (55.507)	71.23 (57.649)	4.36
T_2	SA-SL	0.00 (0.00)	16.17 (23.595)	36.46 (37.110)	54.74 (47.707)	68.71 (55.994)	71.07 (57.473)	4.57
T_3	SA-SPY	0.00 (0.00)	12.26 (20.394)	29.42 (32.815)	44.18 (41.637)	67.19 (55.046)	68.31 (55.734)	8.28
T_4	SA-S+SL	0.00 (0.00)	9.05 (17.433)	26.64 (31.051)	39.01 (38.632)	66.32 (54.509)	69.02 (56.164	7.33
T_5	SA-S+SL+SPY	0.00 (0.00)	9.50 (17.929)	24.45 (29.620)	36.72 (37.283)	57.41 (49.242)	64.62 (53.491)	13.24
T_6	BABA-S	0.00 (0.00)	11.29 (19.313)	31.94 (34.356)	48.77 (44.275)	65.64 (54.162)	68.42 (55.874)	8.14
T_7	BABA-SL	0.00 (0.00)	16.97 (24.220)	37.44 (37.694)	47.54 (43.569)	65.51 (54.036)	68.29 (55.734)	8.31
T_8	BABA-SPY	0.00 (0.00)	19.58 (26.210)	32.59 (34.783)	44.94 (42.075)	63.94 (53.074)	67.11 (54.985)	9.90
T_9	BABA-S+SL	0.00 (0.00)	16.39 (23.846)	36.84 (37.350)	47.32 (43.445)	65.07 (53.755)	67.65 (55.315)	9.18
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	10.23 (18.632)	25.76 (30.485)	38.69 (38.447)	56.58 (48.766)	62.71 (52.343)	15.81
T_{11}	CHT-S	0.00 (0.00)	9.65 (17.678)	24.72 (29.726)	37.14 (37.505)	66.37 (54.535)	72.75 (58.571)	2.32
T_{12}	CHT-SL	0.00 (0.00)	18.08 (25.067)	39.90 (39.144)	59.90 (50.703)	70.78 (57.290)	72.38 (58.310)	2.82
T_{13}	CHT-SPY	0.00 (0.00)	15.56 (23.160)	35.35 (36.455)	53.07 (46.743)	67.68 (55.346)	70.20 (56.910)	5.74
T_{14}	CHT-S+SL	0.00 (0.00)	13.42 (21.445)	31.50 (34.121)	47.30 (43.434)	66.42 (54.570)	70.07 (56.818)	5.92
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	12.42 (20.617)	29.71 (33.014)	44.62 (41.894)	58.53 (49.908)	65.39 (53.947)	12.21
T_{16}	Control	0.00 (0.00)	19.83 (26.316)	40.41 (39.436)	66.90 (54.895)	73.86 (59.296)	74.48 (59.705)	0.00
	S.E. $\pm$	0.00	0.758	0.530	0.510	0.689	0.715	--
	C.D. at 5 %	0.00	2.200	1.539	1.479	2.001	2.075	--

* : Mean of three replications, Figures in parentheses are angular transformed values

58.53 per cent. Whereas the maximum disease incidence was continuously observed in T_{16} i.e. control (73.86 %). In rest of the treatments the percent disease incidence was ranged from 63.94 (T_8) to 70.78 per cent (T_{12}) per cent.

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_{10} (BABA- S+SL+SPY) i.e. 62.71 per cent followed by T_5 (SA-S+SL+SPY) i.e. 64.62 per cent and T_{15} (CHT-S+SL+SPY) i.e. 65.39 per cent. Whereas the maximum disease incidence was continuously observed in T_{16} i.e. control (74.48 %). In rest of the treatments the percent disease incidence was ranged from 67.11% (T_8) to 72.75% (T_{11}) per cent. The percent disease reduction was highest recorded in T10 i. e. BABA-S+SL+SPY (15.81 %) followed by T5 (13.24 %) and T15 (12.21 %).

***Kharif* 2015**

The data recorded on percent disease incidence of powdery mildew on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 11) followed the same pattern as recorded during *kharif* 2014. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease incidence in treatments varied from 10.30 to 78.35 per cent in comparison with 80.08 per cent recorded in control.

Result also indicated that the effect of RIC's treatments on powdery mildew disease incidence of tomato was significant up to 105 DAT. However the incidence of powdery mildew disease was significantly minimum in plots treated with treatment T_4 (SA-S+SL) i.e. 10.30 per cent which was at par with treatment T_{11} (CHT-S) i.e. 10.90 per cent, T_5 (SA-S+SL) i.e.10.75 per cent and T_{10} (BABA-S+SL+SPY) i.e. 11.48 per cent and T_6 (BABA-S) i.e. 12.54 per cent at 45 DAT, treatment T_5 found significantly superior over other treatment except treatment T_{11}, T_{10} and T_4 at 60, 75 and 90 DAT. Whereas maximum disease incidence was continuously observed in untreated control plot.

Table 11. Effect of RIC's on Powdery mildew disease incidence of tomato *var. Dhanashree* during *Kharif* 2015

Tr. No.	Treatment	Powdery mildew disease incidence* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	14.09 (21.815)	32.55 (34.773)	50.83 (45.459)	72.03 (58.107)	76.83 (61.288)	4.06
T_2	SA-SL	0.00 (0.00)	17.42 (24.566)	38.56 (38.370)	57.84 (49.501)	72.91 (58.652)	76.67 (61.148)	4.25
T_3	SA-SPY	0.00 (0.00)	13.51 (21.477)	31.52 (34.140)	47.28 (43.421)	71.39 (57.661)	73.91 (59.284)	7.70
T_4	SA-S+SL	0.00 (0.00)	10.30 (18.657)	28.74 (32.396)	42.11 (40.442)	70.52 (57.100)	74.62 (59.737)	6.81
T_5	SA-S+SL+SPY	0.00 (0.00)	10.75 (19.119)	26.55 (30.996)	39.82 (39.110)	61.61 (51.693)	70.22 (56.922)	12.32
T_6	BABA-S	0.00 (0.00)	12.54 (20.471)	34.04 (35.678)	51.87 (46.057)	69.84 (56.716)	74.02 (59.401)	7.57
T_7	BABA-SL	0.00 (0.00)	18.22 (25.172)	39.54 (38.945)	50.64 (45.349)	69.71 (56.617)	73.89 (59.291)	7.73
T_8	BABA-SPY	0.00 (0.00)	20.83 (27.106)	34.69 (36.066)	48.04 (43.858)	68.14 (55.615)	72.71 (58.486)	9.21
T_9	BABA-S+SL	0.00 (0.00)	17.64 (24.802)	38.94 (38.592)	50.42 (45.223)	69.27 (56.319)	73.25 (58.834)	8.53
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	11.48 (19.786)	27.86 (31.836)	41.79 (40.258)	60.78 (51.213)	68.31 (55.720)	14.70
T_{11}	CHT-S	0.00 (0.00)	10.90 (18.938)	26.82 (31.175)	40.24 (39.336)	70.57 (57.125)	78.35 (62.344)	2.16
T_{12}	CHT-SL	0.00 (0.00)	19.33 (25.994)	42.00 (40.366)	63.00 (52.532)	74.98 (60.010)	77.98 (62.050)	2.63
T_{13}	CHT-SPY	0.00 (0.00)	16.81 (24.140)	37.45 (37.713)	56.17 (48.529)	71.88 (57.973)	75.80 (60.536)	5.34
T_{14}	CHT-S+SL	0.00 (0.00)	14.67 (22.481)	33.60 (35.387)	50.40 (45.211)	70.62 (57.163)	75.67 (60.433)	5.50
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	13.67 (21.682)	31.81 (34.300)	47.72 (43.675)	62.73 (52.381)	70.99 (57.396)	11.36
T_{16}	Control	0.00 (0.00)	21.08 (27.216)	42.51 (40.669)	70.00 (56.866)	78.06 (62.237)	80.08 (63.698)	0.00
	S.E. $\pm$	0.00	0.721	0.868	0.610	0.676	0.722	--
	C.D. at 5 %	0.00	2.091	2.520	1.771	1.963	2.094	--

* : Mean of three replications, Figures in parentheses are angular transformed values

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 68.31 per cent which was at par with treatment T_5 of SA-S+SL+SPY (70.22 %) and T_{15} (CHT-S+SL+SPY) i.e. 70.99 per cent. Whereas the maximum disease incidence was continuously observed in T_{16} i.e. control (80.08 %). In rest of the treatments the percent disease incidence was ranged from 72.71 per cent (T_8) to 78.35 (T_{11}) per cent. The percent disease reduction was highest recorded in T_{10} i.e. BABA- S+SL+SPY (14.70 %) followed by T_5 of SA-S+SL+SPY (12.32 %) and T_{15} of BABA-S+SL +SPY (11.36 %).

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on disease incidence presented in Table 12 revealed that the data followed the same pattern as recorded during *kharif* 2014 and *kharif* 2015. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all treatments the pooled percent disease incidence increased with age of the plants *i.e.* from 30 DAT to 105 DAT. However the per cent disease incidence were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence increase was comparatively low in case of RIC's treated plots over control. The pooled disease incidence ranged from 9.68 to 75.55 per cent throughout the crop growth period in comparison with 77.28 per cent recorded in control.

Pooled result also indicated that the effect of RIC's treatments on powdery mildew disease incidence of tomato was significant up to 105 DAT. However the incidence of powdery mildew disease was significantly minimum in plots treated with treatment T_{10} (BABA-S+SL+SPY) which was at par with treatment T_5 (SA-S+SL+SPY) and T_{15} (CHT-S+SL+SPY) at 105 DAT, whereas maximum disease incidence was continuously observed in unsprayed control plot.

Table 12. Pooled data on effect of RIC's on Powdery mildew disease incidence of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Powdery mildew disease incidence* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	13.46 (21.284)	31.50 (34.124)	49.28 (44.568)	69.93 (56.795)	74.03 (59.438)	4.90
T_2	SA-SL	0.00 (0.00)	16.80 (24.088)	37.51 (37.743)	56.29 (48.602)	70.81 (57.308)	73.87 (59.277)	5.10
T_3	SA-SPY	0.00 (0.00)	12.89 (20.946)	30.47 (33.491)	45.73 (42.530)	69.29 (56.341)	71.11 (57.483)	8.68
T_4	SA-S+SL	0.00 (0.00)	9.68 (18.059)	27.69 (31.735)	40.56 (39.540)	68.42 (55.793)	71.82 (57.923)	7.76
T_5	SA-S+SL+SPY	0.00 (0.00)	10.12 (18.528)	25.50 (30.315)	38.27 (38.200)	59.51 (50.463)	67.42 (55.187)	13.46
T_6	BABA-S	0.00 (0.00)	11.92 (19.905)	32.99 (35.029)	50.32 (45.166)	67.74 (55.429)	71.22 (57.613)	8.54
T_7	BABA-SL	0.00 (0.00)	17.60 (24.703)	38.49 (38.326)	49.09 (44.460)	67.61 (55.315)	71.09 (57.486)	8.70
T_8	BABA-SPY	0.00 (0.00)	20.20 (26.657)	33.64 (35.430)	46.49 (42.968)	66.04 (54.335)	69.91 (56.712)	10.24
T_9	BABA-S+SL	0.00 (0.00)	17.01 (24.324)	37.89 (37.978)	48.87 (44.334)	67.17 (55.026)	70.45 (57.050)	9.54
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	10.85 (19.212)	26.81 (31.168)	40.24 (39.355)	58.68 (49.985)	65.51 (54.015)	15.93
T_{11}	CHT-S	0.00 (0.00)	10.28 (18.324)	25.77 (30.488)	38.69 (38.424)	68.47 (55.819)	75.55 (60.419)	2.94
T_{12}	CHT-SL	0.00 (0.00)	18.71 (25.537)	40.95 (39.768)	61.45 (51.614)	72.88 (58.633)	75.18 (60.143)	3.42
T_{13}	CHT-SPY	0.00 (0.00)	16.18 (23.650)	36.40 (37.093)	54.62 (47.635)	69.78 (56.647)	73.00 (58.693)	6.23
T_{14}	CHT-S+SL	0.00 (0.00)	14.04 (21.964)	32.55 (34.758)	48.85 (44.323)	68.52 (55.855)	72.87 (58.596)	6.40
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	13.05 (21.159)	30.76 (33.666)	46.17 (42.786)	60.63 (51.139)	68.19 (55.651)	12.46
T_{16}	Control	0.00 (0.00)	20.46 (26.772)	41.46 (40.058)	68.45 (55.869)	75.96 (60.732)	77.28 (61.639)	0.00
	S.E. $\pm$	0.00	0.738	0.492	0.553	0.659	0.693	--
	C.D. at 5 %	0.00	2.142	1.429	1.604	1.914	2.010	--

* : Mean of three replications, Figures in parentheses are angular transformed values

At 45 DAT, significantly minimum disease incidence was recorded in treatment T_4 (SA-S+SL) i.e.9.68 per cent which was at par with treatment T_{11} (CHT-S) i.e. 10.28 per cent, T_5 (SA-S+SL+SPY) i.e. 10.12 per cent, T_{10} (BABA-S+SL+SPY) i.e. 10.85 per cent and T_6 (BABA-S) i.e. 11.92 per cent. Whereas the maximum disease incidence was observed in T_{16} i.e. control (20.46 %). In rest of the treatments the disease incidence was ranged from 12.89 (T_3) to 20.20 (T_8) per cent.

The percent disease incidence in different treatments at 60 and 75 days after transplanting (DAT) demonstrated that the treatment T_5 (SA-S+SL+SPY) was significantly superior over rest of the treatment except treatment T_{11} (CHT-S), T_{10} (BABA-S+SL+SPY) and T_4 (SA-S+SL). However the remaining treatments performed comparatively better than control.

At 90 DAT, significantly minimum disease incidence was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 58.68 per cent followed by T_5 (SA-S+SL+SPY) i.e. 59.51 per cent and T_{15} (CHT-S+SL+SPY) i.e. 60.63 per cent. Whereas the maximum disease incidence was continuously observed in T_{16} i.e. control (75.96 %). In rest of the treatments the percent disease incidence was ranged from 66.04 per cent (T_8) to 72.88 per cent (T_{12}).

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_{10} (BABA- S+SL+SPY) i.e. 65.51 per cent followed by T_5 (SA-S+SL+SPY) i.e. 67.42 per cent and T_{15} (CHT-S+SL+SPY) i.e. 68.19 per cent. Whereas the maximum disease incidence was continuously observed in T_{16} i.e. control (77.28 %). In rest of the treatments the percent disease incidence was ranged from 69.91 per cent (T_8) to 75.55 per cent (T_{11}). The percent disease reduction was highest recorded in T_{10} i.e. BABA- S+SL+SPY (15.93 %) followed by T_5 i.e. SA-S+SL+SPY (13.46 %) and T_{15} i.e. CHT-S+SL+SPY (12.46 %).

A perusal of the data of the effect of RIC's on powdery mildew incidence under field conditions indicated that the different treatments

has effect on powdery mildew incidence and considerable reduction in disease incidence was observed when the number of sprays was increased. Critical analysis of the data revealed that the β amino butyric acid was found effective in reducing the disease incidence as compared to other chemicals.

4.1.4 Effect of RIC's on Powdery mildew disease severity of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatment on Powdery mildew disease severity during *kharif* 2014 and 2015 crop season is presented in Table 13 and Table 14.

The data on percent disease severity of powdery mildew during *kharif* 2014 and *kharif* 2015 was recorded periodically from 30 to 105 days after transplanting (DAT) with an interval of 15 days (Table 13 and 14). It has been found that in all treatments percent disease severity increased with age of the plants *i.e.* from 45 DAT to 105 DAT. However the per cent disease severity were considerably lower in RIC's treated plots. Also, the rate of percent disease severity increase was comparatively low in case of RIC's treated plots over control. Data given in Table 13 and 14 revealed that severity of Powdery mildew of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 45, 60, 75, 90 and 105 DAT. Data on disease severity also showed that among all RIC's tested, treatment T_{10} (BABA-S+SL+SPY) reduced the disease severity significantly compared to control and showed the least disease severity from 45 DAT to 105 DAT.

***Kharif* 2014**

Data recorded during *Kharif* 2014 indicated that effect of RIC's treatments on severity of powdery mildew disease of tomato was significant up to 105 DAT. However the severity of powdery mildew disease was significantly minimum in plots treated with treatment T_{10}

(BABA-S+ SL+ SPY) from 45 DAT to 105 DAT, whereas maximum disease severity was continuously observed in unsprayed control plot.

At 45 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 5.96 per cent which was at par with treatment T_5 (SA-S+SL+SPY) i.e. 6.33 per cent, T_{15} (CHT-S+SL+SPY) i.e. 6.53 per cent, T_9 (BABA-S+SL) i.e. 6.81 per cent, T_8 (BABA-SPY) i.e. 6.96 per cent, T_7 (BABA-SL) i.e. 7.55 per cent, T_3 (SA-SPY) 7.45 per cent, T6 (BABA-S) i.e. 8.25 per cent, T_2 (SA-SL) 8.26 per cent, T_1 (SA-S) i.e. 8.49 per cent and T_{13} (CHT-SPY) i.e. 8.30 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. control (14.22 %). In rest of the treatments the disease severity was ranged from 8.89 (T_{12}) to 9.02 (T_4) per cent.

At 60 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 9.27 per cent which was at par with treatment T_5 (SA-S+SL+SPY) i.e. 9.78 per cent, T_{15} (CHT-S+SL+SPY) i.e. 10.05 per cent, T_9 (BABA-S+SL) i.e.10.45 per cent, T_8 (BABA-SPY) i.e. 10.65 per cent, T_3 (SA-SPY) i.e. 11.33 per cent and T_7 (BABA-SL) i.e. 11.47 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. control (19.75 %). In rest of the treatments the disease severity was ranged from 12.45 (T_6) to 13.51 (T_4) per cent.

At 75 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 16.61 per cent which was at par with treatment T_5 (SA-S+SL+SPY) i.e. 17.42 per cent, T_{15} (CHT-S+SL+SPY) i.e. 17.87 per cent, T_9 (BABA-S+SL) i.e. 18.51 per cent and T_8 (BABA-SPY) i.e. 18.84 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. Control (31.97 %). In rest of the treatments the disease severity was ranged from 19.94 (T_3) to 23.66 (T_{11}) per cent.

At 90 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 25.71 per cent which was at par with treatment T_5 (SA-S+SL+SPY) i.e. 26.86 per cent and T_{15}

Table 13. Effect of RIC's on Powdery mildew disease severity of tomato *var. Dhanashree* during *Kharif* 2014

Tr. No.	Treatment	Powdery mildew disease severity* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	8.49 (16.412)	12.78 (20.686)	22.29 (28.067)	33.74 (35.460)	40.92 (39.734)	20.28
T_2	SA-SL	0.00 (0.00)	8.26 (16.363)	12.45 (20.488)	21.76 (27.732)	32.99 (35.016)	40.07 (39.244)	21.94
T_3	SA-SPY	0.00 (0.00)	7.45 (15.620)	11.33 (19.555)	19.94 (26.471)	30.42 (33.442)	37.15 (37.529)	27.62
T_4	SA-S+SL	0.00 (0.00)	9.02 (17.403)	13.51 (21.521)	23.47 (28.952)	35.40 (36.491)	42.81 (40.847)	16.60
T_5	SA-S+SL+SPY	0.00 (0.00)	6.33 (14.537)	9.78 (18.202)	17.42 (24.653)	26.86 (31.201)	33.11 (35.113)	35.50
T_6	BABA-S	0.00 (0.00)	8.25 (16.135)	12.45 (20.389)	21.75 (27.690)	32.97 (34.990)	40.05 (39.225)	21.98
T_7	BABA-SL	0.00 (0.00)	7.55 (15.553)	11.47 (19.598)	20.16 (26.597)	30.73 (33.622)	37.50 (37.729)	26.94
T_8	BABA-SPY	0.00 (0.00)	6.96 (15.051)	10.65 (18.921)	18.84 (25.669)	28.87 (32.468)	35.39 (36.479)	31.05
T_9	BABA-S+SL	0.00 (0.00)	6.81 (15.015)	10.45 (18.799)	18.51 (25.451)	28.40 (32.181)	34.86 (36.167)	32.09
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	5.96 (14.093)	9.27 (17.703)	16.61 (24.035)	25.71 (30.453)	31.79 (34.305)	38.07
T_{11}	CHT-S	0.00 (0.00)	9.10 (17.091)	13.63 (21.431)	23.66 (29.010)	35.67 (36.627)	43.12 (41.015)	15.99
T_{12}	CHT-SL	0.00 (0.00)	8.89 (17.048)	13.33 (21.258)	23.18 (28.714)	34.99 (36.229)	42.34 (40.568)	17.51
T_{13}	CHT-SPY	0.00 (0.00)	8.30 (16.559)	12.52 (20.623)	21.86 (27.829)	33.14 (35.119)	40.24 (39.349)	21.61
T_{14}	CHT-S+SL	0.00 (0.00)	9.01 (17.393)	13.50 (21.513)	23.46 (28.945)	35.38 (36.479)	42.79 (40.836)	16.64
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	6.53 (14.772)	10.05 (18.461)	17.87 (24.991)	27.49 (31.607)	33.82 (35.544)	34.11
T_{16}	Control	0.00 (0.00)	14.22 (21.935)	19.75 (26.258)	31.97 (34.375)	45.15 (42.190)	51.33 (45.746)	0.00
	S.E. $\pm$	0.00	0.878	0.713	0.572	0.512	0.498	--
	C.D. at 5 %	0.00	2.547	2.070	1.659	1.484	1.446	--

* : Mean of three replications, Figures in parentheses are angular transformed values

(CHT-S+SL+SPY) i.e. 27.49 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. Control (45.15 %). In rest of the treatments the disease severity was ranged from 28.40 (T_9) to 35.67 (T_{11}) per cent.

At 105 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 31.79 per cent which was at par with treatment T_5 (SA-S+SL+SPY) i.e. 33.11 per cent and T_{15} (CHT-S+SL+SPY) i.e. 33.82 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. Control (51.33 %). In rest of the treatments the disease severity was ranged from 34.86 (T_9) to 43.12 (T_{11}) per cent.

***Kharif* 2015**

The data recorded on percent disease severity of powdery mildew on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 14) followed the same pattern as recorded during *kharif* 2014. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease severity in treatments varied from 7.14 to 49.64 per cent in comparison with 56.93 per cent recorded in control.

Result also indicated that the effect of RIC's treatments on powdery mildew disease severity of tomato was significant up to 105 DAT. However the severity of powdery mildew disease was significantly minimum in plots treated with treatment T_{10} (BABA-S+SL) i.e. 7.14 per cent which was at par with treatment T_5 (SA-S+SL+SPY) i.e. 7.89 per cent, T_{15} (CHT-S+SL+SPY) i.e. 8.21 per cent, T_9 (BABA-S+SL) i.e. 8.59, T_8 (BABA-SPY) 8.87 per cent, T_3 (SA-SPY) i.e.9.06 per cent, T_7 (BABA-SL) i.e. 9.22 per cent at 45 DAT. Whereas the maximum disease severity was observed in T_{16} i.e. Control (16.44 %). In rest of the treatments the disease severity was ranged from 9.90 (T_6) to 11.05 (T_4) per cent.

At 60 DAT, treatment T_{10} of BABA-S+SL+SPY (10.36 %) was found significantly superior over other treatment except treatment T_5 of

Table 14. Effect of RIC's on Powdery mildew disease severity of tomato *var. Dhanashree* during *Kharif* 2015

Tr. No.	Treatment	Powdery mildew disease severity* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	10.15 (18.193)	14.28 (21.986)	25.15 (30.013)	37.86 (37.933)	46.52 (42.980)	18.29
T_2	SA-SL	0.00 (0.00)	9.90 (18.088)	13.96 (21.795)	24.63 (29.694)	37.12 (37.504)	45.67 (42.493)	19.78
T_3	SA-SPY	0.00 (0.00)	9.06 (17.356)	12.86 (20.920)	22.83 (28.499)	34.59 (35.998)	42.75 (40.810)	24.91
T_4	SA-S+SL	0.00 (0.00)	11.05 (19.359)	15.45 (23.108)	27.07 (31.329)	40.56 (39.540)	49.64 (44.776)	12.81
T_5	SA-S+SL+SPY	0.00 (0.00)	7.89 (16.286)	11.34 (19.659)	20.34 (26.793)	31.09 (33.873)	38.71 (38.459)	32.01
T_6	BABA-S	0.00 (0.00)	9.90 (17.938)	13.95 (21.706)	24.62 (29.659)	37.11 (37.488)	45.65 (42.479)	19.81
T_7	BABA-SL	0.00 (0.00)	9.22 (17.395)	13.08 (21.042)	23.18 (28.714)	35.09 (36.290)	43.33 (41.142)	23.89
T_8	BABA-SPY	0.00 (0.00)	8.87 (17.160)	12.62 (20.711)	22.44 (28.231)	34.04 (35.665)	42.12 (40.444)	26.01
T_9	BABA-S+SL	0.00 (0.00)	8.59 (16.963)	12.25 (20.438)	21.83 (27.828)	33.18 (35.151)	41.13 (39.872)	27.75
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	7.14 (15.468)	10.36 (18.755)	18.73 (25.629)	28.83 (32.460)	36.10 (36.914)	36.59
T_{11}	CHT-S	0.00 (0.00)	10.78 (18.821)	15.10 (22.669)	26.51 (30.911)	39.77 (39.060)	48.72 (44.246)	14.42
T_{12}	CHT-SL	0.00 (0.00)	10.56 (18.737)	14.81 (22.501)	26.03 (30.621)	39.09 (38.668)	47.94 (43.799)	15.79
T_{13}	CHT-SPY	0.00 (0.00)	9.95 (18.247)	14.02 (21.905)	24.73 (29.782)	37.27 (37.600)	45.84 (42.594)	19.48
T_{14}	CHT-S+SL	0.00 (0.00)	10.67 (19.006)	14.96 (22.716)	26.27 (30.810)	39.44 (38.885)	48.34 (44.030)	15.09
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	8.21 (16.624)	11.76 (20.036)	21.02 (27.274)	32.05 (34.465)	39.82 (39.110)	30.06
T_{16}	Control	0.00 (0.00)	16.44 (23.748)	21.4 (27.443)	35.07 (36.266)	49.35 (44.608)	56.93 (48.977)	0.00
	S.E. $\pm$	0.00	0.799	0.682	0.552	0.503	0.497	--
	C.D. at 5 %	0.00	2.319	1.979	1.602	1.460	1.441	--

* : Mean of three replications, Figures in parentheses are angular transformed values

SA-S+SL+SPY (11.34 %), T_{15} of CHT-S+SL+SPY (11.76 %), T_9 of BABA-S+SL (12.25 %) and T_8 of BABA-SPY (12.62 %). Whereas the maximum disease severity was observed in T16 i.e. Control (21.40 %). In rest of the treatments the disease severity was ranged from 12.86 (T_3) to 15.45 (T_4) per cent.

At 75 DAT, the treatment T_{10} of BABA-S+SL+SPY showed significantly minimum percent disease severity over other treatments except treatment T_5 of SA-S+SL+SPY and T_{15} of CHT-S+SL+SPY with 18.73, 20.34 and 21.02 per cent disease severity respectively, whereas maximum disease severity (35.07 %) was continuously observed in control plot (Unsprayed plot). In rest of the treatments the disease severity was ranged from 21.83 (T_9) to 27.07 (T_4) per cent.

At 90 DAT, the treatment T_{10} of BABA-S+SL+SPY showed significantly minimum percent disease severity over other treatments except treatment T_5 of SA-S+SL+SPY with 28.83 and 31.09 per cent disease severity respectively, whereas maximum disease severity (49.35 %) was continuously observed in unsprayed control plot. In rest of the treatments the disease severity was ranged from 32.05 (T_{15}) to 40.56 (T_4) per cent.

At 105 DAT, treatment T_{10} of BABA-S+SL+SPY (36.10 %) was found significantly superior over other treatments. Whereas the maximum disease severity was observed in T_{16} i.e. Control (56.93 %). In rest of the treatments the disease severity was ranged from 38.71 (T_5) to 49.64 (T_4) per cent.

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on powdery mildew disease severity presented in Table 15 revealed that the data followed the same pattern as recorded during previous crop season. All the treatments comparatively reduced the pooled disease severity over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all

Table 15. Pooled data on effect of RIC's on Powdery mildew disease severity of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Powdery mildew disease severity* (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	9.32 (17.329)	13.53 (21.345)	23.72 (29.051)	35.80 (36.705)	43.72 (41.363)	19.23
T_2	SA-SL	0.00 (0.00)	9.08 (17.248)	13.21 (21.155)	23.19 (28.721)	35.05 (36.265)	42.87 (40.875)	20.80
T_3	SA-SPY	0.00 (0.00)	8.25 (16.505)	12.10 (20.252)	21.38 (27.494)	32.51 (34.734)	39.95 (39.179)	26.19
T_4	SA-S+SL	0.00 (0.00)	10.03 (18.399)	14.48 (22.326)	25.27 (30.155)	37.98 (38.026)	46.22 (42.814)	14.61
T_5	SA-S+SL+SPY	0.00 (0.00)	7.11 (15.434)	10.56 (18.943)	18.88 (25.739)	28.97 (32.549)	35.91 (36.800)	33.66
T_6	BABA-S	0.00 (0.00)	9.07 (17.059)	13.20 (21.057)	23.18 (28.682)	35.04 (36.247)	42.85 (40.859)	20.84
T_7	BABA-SL	0.00 (0.00)	8.38 (16.496)	12.27 (20.327)	21.67 (27.669)	32.91 (34.967)	40.42 (39.449)	25.33
T_8	BABA-SPY	0.00 (0.00)	7.92 (16.146)	11.64 (19.838)	20.64 (26.972)	31.46 (34.088)	38.75 (38.475)	28.41
T_9	BABA-S+SL	0.00 (0.00)	7.70 (16.017)	11.35 (19.633)	20.17 (26.658)	30.79 (33.682)	37.99 (38.032)	29.82
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	6.55 (14.795)	9.82 (18.240)	17.67 (24.842)	27.27 (31.465)	33.95 (35.623)	37.28
T_{11}	CHT-S	0.00 (0.00)	9.94 (17.979)	14.37 (22.062)	25.08 (29.967)	37.72 (37.850)	45.92 (42.635)	15.17
T_{12}	CHT-SL	0.00 (0.00)	9.72 (17.907)	14.07 (21.887)	24.60 (29.674)	37.04 (37.456)	45.14 (42.188)	16.61
T_{13}	CHT-SPY	0.00 (0.00)	9.13 (17.428)	13.27 (21.272)	23.30 (28.820)	35.20 (36.365)	43.04 (40.978)	20.49
T_{14}	CHT-S+SL	0.00 (0.00)	9.84 (18.215)	14.23 (22.121)	24.86 (29.883)	37.41 (37.689)	45.57 (42.440)	18.78
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	7.37 (15.723)	10.90 (19.258)	19.44 (26.147)	29.77 (33.052)	36.82 (37.342)	31.98
T_{16}	Control	0.00 (0.00)	15.33 (22.857)	20.58 (26.859)	33.52 (35.326)	47.25 (43.401)	54.13 (47.358)	0.00
	S.E. $\pm$	0.00	0.834	0.697	0.561	0.507	0.497	--
	C.D. at 5 %	0.00	2.422	2.022	1.629	1.471	1.441	--

* : Mean of three replications, Figures in parentheses are angular transformed values

treatments the pooled percent disease severity increased with age of the plants *i.e.* from 45 to 105 DAT. However the per cent disease severity were considerably lower in RIC's treated plots. Also, the rate of percent disease severity increase was comparatively low in case of RIC's treated plots over control. The pooled disease severity ranged from 6.55 to 46.38 per cent throughout the crop growth period in comparison with 54.13 per cent recorded in control.

Result also indicated that the effect of RIC's treatments on pooled powdery mildew disease severity of tomato was significant up to 105 DAT. However the severity of powdery mildew disease was significantly minimum in plots treated with treatment T_{10} (BABA-S+SL+SPY) which was at par with treatment T_5 (SA-S+SL+SPY) from 30 DAT to 105 DAT, whereas maximum disease severity was continuously observed in unsprayed control plot.

At 45 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 6.55 per cent, T_5 (SA-S+SL+SPY) i.e. 7.11 per cent, T_{15} (CHT-S+SL+SPY) i.e. 7.37 per cent, T_9 (BABA-S+SL) i.e. 7.70 per cent, T_8 (BABA-SPY) i.e. 7.92 per cent, T_7 (BABA-SL) i.e. 8.38 per cent, T_3 (SA-SPY) i.e. 8.25 per cent, T_6 (BABA-S) i.e. 9.07 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. Control (15.33 %). In rest of the treatments the disease severity was ranged from 9.08 (T_2) to 10.03 (T_4) per cent.

The percent disease severity in different treatments at 60 days after transplanting (DAT) demonstrated that the treatment T_{10} of BABA- S+SL+SPY (9.82 %) was significantly superior over rest of the treatment except treatment T_5 (SA-S+SL+SPY) i.e.10.56, T_{15} (CHT-S+SL+SPY) i.e. 10.90, T_9 (BABA-S+SL) i.e. 11.35 per cent, T_8 (BABA-SPY) i.e. 11.64, T_3 (SA-SPY) i.e. 12.10 per cent. Whereas the maximum disease severity was observed in T_{16} i.e. Control (20.58 %). In rest of the treatments the disease severity was ranged from 12.27 (T_7) to 14.48 (T_4) per cent.

At 75 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA- S+SL+SPY) i.e. 17.67 per cent followed by T_5 (SA-S+SL+SPY) i.e. 18.88 per cent and T_{15} (CHT-S) i.e. 19.44 per cent. Whereas the maximum disease severity was continuously observed in T_{16} i.e. Control (33.52 %). In rest of the treatments the percent disease severity was ranged from 20.17 per cent (T_9) to 25.27 per cent (T_4).

The pooled percent disease severity in different treatments at 90 and 105 days after transplanting (DAT) demonstrated that the treatment T_{10} of BABA-S+SL+SPY was significantly superior over rest of the treatment except treatment T_5 (SA-S+SL+SPY). At 105 DAT, significantly minimum disease severity was recorded in treatment T_{10} (BABA-S+SL+SPY) i.e. 33.95 per cent followed by T_5 (SA-S+SL+SPY) i.e. 35.91 per cent. Whereas the maximum disease severity was continuously observed in T_{16} i.e. Control (54.13 %). In rest of the treatments the percent disease severity was ranged from 36.82 per cent (T_{15}) to 46.22 per cent (T_4). The percent disease reduction was highest recorded in T_{10} i.e. BABA- S+SL+SPY (15.93 %) followed by T_5 i.e. SA-S+SL+SPY (33.66 %) and T15 i.e. CHT-S+SL+SPY (41.81 %).

A perusal of the data of the effect of RIC's on powdery mildew severity under field conditions indicated that the different treatments has effect on powdery mildew severity and considerable reduction in disease severity was observed when the number of sprays was increased. Critical analysis of the data revealed that the β amino butyric acid was found effective in reducing the disease severity as compared to other chemicals.

4.1.5 Effect of RIC's on Tomato leaf curl virus disease incidence of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatments on Tomato leaf curl virus disease incidence during *kharif* 2014 and 2015 crop season is presented in Table 16 and Table 17.

The data on percent disease incidence of tomato leaf curl virus during *kharif* 2014 and *kharif* 2015 was recorded periodically from 30 to 105 days after transplanting (DAT) with an interval of 15 days (Table 16 and 17). It has been found that in all treatments percent disease incidence increased with age of the plants *i.e.* from 30 DAT to 105 DAT. However the per cent disease incidence were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence increase was comparatively low in case of RIC's treated plots over control. Data given in Table 16 and 17 revealed that incidence of leaf curl virus disease of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 30, 45, 60, 75, 90 and 105 DAT. Data on disease incidence also showed that among all RIC's tested, treatment T5 (SA-S+SL+SPY) reduced the disease incidence significantly compared to control and showed the least disease incidence from 30 DAT to 105 DAT.

***Kharif* 2014**

Data recorded during *Kharif* 2014 indicated that effect of RIC's treatments on incidence of leaf curl virus disease of tomato was significant up to 105 DAT. However the incidence of leaf curl virus disease was significantly minimum in plots treated with treatment T5 (SA-S+SL+SPY) at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease incidence was continuously observed in unsprayed control plot.

At 30 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 3.64 per cent. Whereas the maximum disease incidence was observed in T_{16} i.e. Control (5.83 %). In rest of the treatments the percent disease incidence was ranged from 3.85 (T_{15}) to 5.02 (T_6) per cent.

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA- S+SL+SPY) i.e. 19.74 per cent. Whereas the maximum disease incidence was continuously observed in T16 i.e. Control (21.67 %). In rest of the treatments the percent disease incidence was ranged from 20.28 (T_3) to 21.36 (T_6) per cent. The percent disease

Table 16. Effect of RIC's on Leaf curl disease incidence of tomato *var. Dhanashree* during *Kharif* 2014

Tr. No.	Treatment	Leaf curl disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	4.20 (11.587)	6.96 (15.188)	10.11 (18.476)	11.65 (19.905)	14.39 (22.252)	20.78 (27.092)	4.11
T_2	SA-SL	4.18 (11.556)	6.87 (15.084)	10.00 (18.370)	11.52 (19.787)	14.24 (22.129)	20.58 (26.950)	5.03
T_3	SA-SPY	3.94 (11.183)	6.75 (14.945)	9.82 (18.196)	11.33 (19.615)	14.02 (21.947)	20.28 (26.737)	6.41
T_4	SA-S+SL	4.00 (11.277)	6.86 (15.073)	9.97 (18.341)	11.50 (19.769)	14.21 (22.104)	20.54 (26.922)	5.21
T_5	SA-S+SL+SPY	3.64 (10.694)	6.52 (14.674)	9.51 (17.892)	11.00 (19.313)	13.63 (21.622)	19.74 (26.349)	8.91
T_6	BABA-S	5.02 (12.768)	7.20 (15.461)	10.45 (18.799)	12.01 (20.226)	14.81 (22.594)	21.36 (27.500)	1.43
T_7	BABA-SL	4.96 (12.686)	7.20 (15.461)	10.44 (18.790)	12.00 (20.217)	14.80 (22.586)	21.34 (27.486)	1.52
T_8	BABA-SPY	4.22 (11.617)	7.09 (15.337)	10.30 (18.657)	11.85 (20.084)	14.62 (22.440)	21.10 (27.318)	2.63
T_9	BABA-S+SL	4.25 (11.662)	7.15 (15.405)	10.37 (18.724)	11.93 (20.155)	14.72 (22.521)	21.23 (27.409)	2.03
T_{10}	BABA-S+SL + SPY	4.22 (11.617)	7.10 (15.348)	10.31 (18.667)	11.86 (20.093)	14.64 (22.456)	21.12 (27.332)	2.54
T_{11}	CHT-S	4.25 (11.662)	7.14 (15.394)	10.35 (18.705)	11.91 (20.137)	14.69 (22.497)	21.20 (27.388)	2.17
T_{12}	CHT-SL	4.20 (11.587)	6.98 (15.211)	10.15 (18.514)	11.68 (19.932)	14.43 (22.285)	20.84 (27.134)	3.83
T_{13}	CHT-SPY	3.96 (11.214)	6.85 (15.061)	9.96 (18.332)	11.48 (19.751)	14.20 (22.096)	20.52 (26.908)	5.31
T_{14}	CHT-S+SL	4.07 (11.387)	6.96 (15.188)	10.11 (18.476)	11.65 (19.905)	14.39 (22.252)	20.78 (27.092)	4.11
T_{15}	CHT-S+SL + SPY	3.85 (11.039)	6.75 (15.003)	9.82 (18.312)	11.33 (19.733)	14.02 (22.074)	20.28 (26.737)	6.41
T_{16}	Control	5.83 (13.830)	9.17 (17.554)	12.50 (20.657)	13.56 (21.563)	15.83 (23.409)	21.67 (27.717)	0.00
	S.E. ±	0.102	0.044	0.028	0.020	0.013	0.007	---
	C.D. at 5 %	0.297	0.128	0.081	0.057	0.037	0.021	---

* : Mean of three replications, Figures in parentheses are angular transformed values

reduction was highest recorded in T_5 i.e. SA-S+SL+SPY (8.91 %) followed by T_3 i.e. SA-SPY (6.41 %).

***Kharif* 2015**

The data recorded on percent disease incidence of leaf curl virus disease on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 17) followed the same pattern as recorded during *kharif* 2014 crop season. All the treatments significantly reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease incidence in treatments varied from 6.92 to 39.53 per cent in comparison with 39.80 per cent recorded in control.

Result also indicated that the effect of RIC's treatments on leaf curl virus disease incidence of tomato was significant up to 105 DAT. However the incidence of leaf curl virus disease was significantly minimum in plots treated with treatment T_5 (SA- S+ SL+ SPY) at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease incidence was continuously observed in untreated control plot.

At 105 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 36.53 per cent. Whereas the maximum disease incidence was continuously observed in T_{16} i.e. Control (39.80 %). In rest of the treatments the percent disease incidence was ranged from 37.53 (T_3) to 39.53 (T_6) per cent. The percentage disease reduction was highest recorded in T_5 i.e. SA- S+SL+SPY (8.22 %) followed by T_3 (5.70 %).

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on leaf curl virus disease incidence presented in Table 18 revealed that the data followed the same pattern as recorded during previous crop season. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all

Table 17. Effect of RIC's on Leaf curl disease incidence of tomato *var. Dhanashree* during *Kharif* 2015

Tr. No.	Treatment	Leaf curl disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	7.39 (15.675)	16.69 (24.078)	26.27 (30.810)	32.52 (34.748)	38.45 (38.303)	3.39
T_2	SA-SL	0.00 (0.00)	7.30 (15.574)	16.50 (23.932)	25.99 (30.628)	32.19 (34.546)	38.08 (38.085)	4.31
T_3	SA-SPY	0.00 (0.00)	7.16 (15.416)	16.22 (23.714)	25.57 (30.352)	31.45 (34.193)	37.53 (37.760)	5.70
T_4	SA-S+SL	0.00 (0.00)	7.28 (15.552)	16.46 (23.901)	25.93 (30.588)	32.12 (34.503)	38.01 (38.043)	4.50
T_5	SA-S+SL+SPY	0.00 (0.00)	6.92 (15.142)	15.70 (23.306)	24.80 (29.844)	30.82 (33.701)	36.53 (37.166)	8.22
T_6	BABA-S	0.00 (0.00)	7.65 (15.962)	17.24 (24.499)	27.09 (31.342)	33.48 (35.333)	39.53 (38.937)	0.69
T_7	BABA-SL	0.00 (0.00)	7.64 (15.951)	17.22 (24.484)	27.06 (31.323)	33.44 (35.309)	39.49 (38.914)	0.78
T_8	BABA-SPY	0.00 (0.00)	7.53 (15.830)	16.99 (24.309)	26.72 (31.103)	33.05 (35.072)	39.04 (38.650)	1.90
T_9	BABA-S+SL	0.00 (0.00)	7.59 (15.896)	17.12 (24.408)	26.91 (31.226)	33.26 (35.200)	39.29 (38.797)	1.29
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	7.54 (15.841)	17.01 (24.324)	26.75 (31.122)	33.09 (35.096)	39.08 (38.673)	1.81
T_{11}	CHT-S	0.00 (0.00)	7.57 (15.874)	17.09 (24.385)	26.86 (31.193)	33.21 (35.169)	39.23 (38.761)	1.43
T_{12}	CHT-SL	0.00 (0.00)	7.41 (15.697)	16.75 (24.125)	26.35 (30.862)	32.62 (34.809)	38.56 (38.368)	3.11
T_{13}	CHT-SPY	0.00 (0.00)	7.27 (15.540)	16.44 (23.885)	25.90 (30.569)	32.10 (34.491)	37.97 (38.020)	4.60
T_{14}	CHT-S+SL	0.00 (0.00)	7.39 (15.675)	16.69 (24.078)	26.27 (30.810)	32.52 (34.748)	38.45 (38.303)	3.39
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	7.20 (15.461)	16.30 (23.776)	25.60 (30.372)	31.70 (34.142)	37.53 (37.760)	5.70
T_{16}	Control	0.00 (0.00)	9.64 (18.020)	20.45 (26.858)	30.33 (33.396)	35.50 (36.551)	39.80 (39.095)	0.00
	S.E. $\pm$	0.00	0.041	0.019	0.010	0.020	0.003	---
	C.D. at 5 %	0.00	0.120	0.056	0.029	0.057	0.009	---

* : Mean of three replications, Figures in parentheses are angular transformed values

Table 18. Pooled data on effect of RIC's on Leaf curl disease incidence of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Leaf curl disease incidence * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	2.10 (8.161)	7.17 (15.428)	13.40 (21.429)	18.96 (25.783)	23.45 (28.938)	29.62 (32.951)	3.66
T_2	SA-SL	2.09 (8.140)	7.09 (15.337)	13.25 (21.302)	18.76 (25.636)	23.22 (28.782)	29.33 (32.769)	4.58
T_3	SA-SPY	1.97 (7.878)	6.96 (15.188)	13.02 (21.105)	18.45 (25.407)	22.86 (28.537)	28.91 (32.504)	5.97
T_4	SA-S+SL	2.00 (7.945)	7.07 (15.314)	13.22 (21.276)	18.71 (25.599)	23.17 (28.749)	29.27 (32.731)	4.77
T_5	SA-S+SL+SPY	1.82 (7.536)	6.72 (14.910)	12.61 (20.752)	17.90 (24.998)	22.23 (28.105)	28.13 (32.009)	8.48
T_6	BABA-S	2.51 (8.987)	7.43 (15.719)	13.84 (21.798)	19.55 (26.212)	24.15 (29.410)	30.44 (33.464)	0.97
T_7	BABA-SL	2.48 (8.930)	7.42 (15.708)	13.83 (21.789)	19.53 (26.198)	24.12 (29.390)	30.42 (33.452)	1.06
T_8	BABA-SPY	2.11 (8.182)	7.31 (15.585)	13.65 (21.639)	19.28 (26.016)	23.84 (29.202)	30.07 (33.233)	2.17
T_9	BABA-S+SL	2.13 (8.225)	7.37 (15.652)	13.74 (21.714)	19.42 (26.118)	23.99 (29.303)	30.26 (33.352)	1.57
T_{10}	BABA-S+SL + SPY	2.11 (8.182)	7.32 (15.596)	13.66 (21.647)	19.30 (26.031)	23.86 (29.215)	30.10 (33.252)	2.08
T_{11}	CHT-S	2.12 (8.204)	7.36 (15.641)	13.72 (21.698)	19.39 (26.096)	23.95 (29.276)	30.22 (33.327)	1.71
T_{12}	CHT-SL	2.10 (8.161)	7.20 (15.461)	13.45 (21.471)	19.02 (25.826)	23.52 (28.986)	29.70 (33.002)	3.38
T_{13}	CHT-SPY	1.98 (7.900)	7.06 (15.303)	13.20 (21.259)	18.69 (25.584)	23.15 (28.735)	29.24 (32.712)	4.87
T_{14}	CHT-S+SL	2.04 (8.032)	7.17 (15.428)	13.40 (21.429)	18.96 (25.783)	23.45 (28.938)	29.62 (32.951)	3.66
T_{15}	CHT-S+SL + SPY	1.93 (7.789)	7.00 (15.234)	13.02 (21.191)	18.45 (25.466)	22.74 (28.505)	28.91 (32.504)	5.97
T_{16}	Control	2.92 (9.737)	9.41 (17.794)	16.48 (23.916)	21.95 (27.911)	25.67 (30.418)	30.74 (33.651)	0.00
	S.E. ±	0.073	0.043	0.023	0.014	0.009	0.005	---
	C.D. at 5 %	0.212	0.124	0.066	0.040	0.025	0.015	----

* : Mean of three replications, Figures in parentheses are angular transformed values

treatments the pooled percent disease incidence increased with age of the plants *i.e.* from 30 DAT to 105 DAT. However the per cent disease incidence were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence increase was comparatively low in case of RIC's treated plots over control. The pooled disease incidence ranged from 1.82 to 30.44 per cent throughout the crop growth period in comparison with 30.74 per cent recorded in control.

Result also indicated that the effect of RIC's treatments on pooled leaf curl virus disease incidence of tomato was significant up to 105 DAT. However the incidence of leaf curl virus disease was significantly minimum in plots treated with treatment T5 (SA-S+SL+SPY) at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease incidence was continuously observed in untreated control plot.

At 30 DAT, significantly minimum disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e.1.82 per cent. Whereas the maximum disease incidence was observed in T_{16} i.e. Control (2.92 %). In rest of the treatments the disease incidence was ranged from 1.97 (T_3) to 2.51 (T_6) per cent.

The pooled percent disease incidence in different treatments at 30, 45, 60, 75, 90 and 105 days after transplanting (DAT) demonstrated that the treatment T_5 (SA- S+SL+SPY) was significantly superior over rest of the treatments. However the remaining treatments performed comparatively better than control.

At 105 DAT, minimum pooled disease incidence was recorded in treatment T_5 (SA-S+SL+SPY) i.e. 28.13 per cent which was significantly superior over other. Whereas the maximum pooled disease incidence was continuously observed in T_{16} i.e. Control (30.74 %). In rest of the treatments the pooled percent disease incidence was ranged from 28.91 (T_3) to 30.44 (T_6) per cent. The percent disease reduction was highest recorded in T_5 i. e. SA- S+SL+SPY (8.48 %) followed by T_3 (5.97 %).

A perusal of the data of the effect of RIC's on leaf curl virus disease incidence under field conditions indicated that the different treatments has effect on leaf curl virus disease incidence and considerable reduction in disease incidence was observed when the number of sprays was increased. Critical analysis of the data revealed that the salicylic acid was found effective in reducing the disease incidence as compared to other chemicals.

4.1.6 Effect of RIC's on Tomato leaf curl virus disease severity of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different RIC's treatments on leaf curl virus disease severity indices during *kharif* 2014 and 2015 crop season is presented in Table 19 and Table 20.

The data on percent disease severity of leaf curl virus disease during *kharif* 2014 and *kharif* 2015 was recorded periodically from 30 to 105 days after transplanting (DAT) with an interval of 15 days (Table 19 and 20). It has been found that in all treatments percent disease severity increased with age of the plants. It was observed that disease severity was increased during 30 to 105 DAT in all treatments but the rate of percent disease severity increase was comparatively low in case of RIC's treated plots. Data given in Table 19 and 20 revealed that severity of leaf curl virus disease of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 30, 45, 60, 75, 90 and 105 DAT. Data on disease severity also showed that among all RIC's tested, treatment T5 (SA-S+SL+SPY) reduced the disease severity significantly compared to control and showed the least disease severity from 30 DAT to 105 DAT.

***Kharif* 2014**

The data on percent disease severity during *kharif* 2014 (Table 19) demonstrated that severity of leaf curl virus disease of tomato *var. Dhanashree* was significantly differ due to different treatments of

Table 19. Effect of RIC's on Leaf curl disease severity of tomato *var. Dhanashree* during *Kharif* 2014

Tr. No.	Treatment	Leaf curl disease severity * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	8.33 (16.647)	10.83 (19.127)	25.54 (30.326)	39.17 (38.724)	56.44 (48.684)	67.33 (55.128)	4.16
T_2	SA-SL	5.83 (13.748)	9.17 (17.516)	24.17 (29.416)	38.33 (38.230)	54.83 (47.755)	66.52 (54.634)	5.31
T_3	SA-SPY	5.00 (12.632)	5.83 (13.748)	24.48 (29.624)	37.66 (37.834)	53.86 (47.197)	65.96 (54.294)	6.11
T_4	SA-S+SL	5.83 (13.748)	8.33 (16.647)	25.06 (30.009)	37.80 (37.917)	54.65 (47.651)	66.46 (54.597)	5.40
T_5	SA-S+SL+SPY	4.17 (11.387)	5.83 (13.748)	23.44 (28.924)	34.54 (35.971)	50.54 (45.291)	64.54 (53.439)	8.13
T_6	BABA-S	11.98 (20.175)	14.50 (22.325)	32.50 (34.732)	47.66 (43.640)	60.02 (50.765)	68.92 (56.106)	1.89
T_7	BABA-SL	11.67 (19.897)	14.17 (22.053)	30.83 (33.703)	45.00 (42.111)	59.60 (50.519)	68.88 (56.081)	1.95
T_8	BABA-SPY	9.17 (17.516)	12.50 (20.634)	29.17 (32.664)	40.83 (39.695)	58.66 (49.971)	68.40 (55.785)	2.63
T_9	BABA-S+SL	10.83 (19.127)	14.17 (22.053)	30.00 (33.185)	45.00 (42.111)	59.44 (50.426)	68.80 (56.032)	2.06
T_{10}	BABA-S+SL + SPY	8.33 (16.647)	12.50 (20.634)	26.67 (31.064)	40.00 (39.211)	58.33 (49.779)	68.33 (55.742)	2.73
T_{11}	CHT-S	9.17 (17.516)	13.33 (21.349)	30.00 (33.185)	41.67 (40.185)	58.98 (50.158)	68.78 (56.019)	2.09
T_{12}	CHT-SL	9.17 (17.516)	12.50 (20.634)	29.17 (32.664)	40.00 (39.211)	58.13 (49.663)	68.09 (55.594)	3.07
T_{13}	CHT-SPY	7.50 (15.744)	10.00 (18.337)	24.17 (29.416)	38.33 (38.230)	55.06 (47.887)	66.56 (54.658)	5.25
T_{14}	CHT-S+SL	8.33 (16.647)	10.83 (19.127)	25.00 (29.969)	39.17 (38.724)	56.32 (48.614)	67.28 (55.097)	4.23
T_{15}	CHT-S+SL + SPY	5.00 (12.632)	8.33 (16.647)	24.84 (29.863)	36.67 (37.247)	53.94 (47.243)	66.02 (54.331)	6.02
T_{16}	Control	13.33 (21.349)	24.17 (29.416)	37.50 (37.739)	53.33 (46.892)	62.50 (52.224)	70.25 (56.935)	0.00
	S.E. $\pm$	0.284	0.226	0.036	0.012	0.007	0.010	---
	C.D. at 5 %	0.825	0.655	0.106	0.034	0.021	0.030	---

* : Mean of three replications, Figures in parentheses are angular transformed values

RIC's from 30 DAT to 105 DAT. However the severity of leaf curl virus disease was comparatively minimum in plots treated with treatment T5 (SA-S+SL+SPY) at 30, 45, 60, 75, 90 and 105 DAT. The remaining treatments performed significantly better than control. Maximum disease severity was continuously observed in unsprayed control plot.

The data recorded at 105 DAT showed minimum disease severity i.e. 64.54 per cent in the plots protected with T_5 (SA-S+SL+SPY) treatment. While maximum disease severity 70.25 per cent was recorded in T_{16} i.e. unsprayed control plot. In rest of the treatments the per cent disease severity was ranged between 65.96 (T_3) to 68.92 (T_6) per cent and most of them were at par with each other. The percent disease severity reduction was found maximum in treatment T_5 i.e. SA-S+SL+SPY (8.13 %), T_3 i.e. SA-SPY (6.11 %) followed by T_{15} i. e. CHT-S+SL+SPY (6.02 %).

***Kharif* 2015**

The data recorded on percent disease severity of leaf curl virus disease on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 20) followed the same pattern as recorded during *kharif* 2014 crop season. All the treatments comparatively reduced the disease severity over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease severity in treatments varied from 10.40 to 67.05 per cent in comparison with 68.33 per cent recorded in control.

Result also indicated that the effect of RIC's treatments on leaf curl virus disease severity of tomato was significant up to 105 DAT. However the severity of leaf curl virus disease was significantly minimum in plots treated with treatment T_5 (SA- S+ SL+ SPY) at 30, 45, 60, 75, 90 and 105 DAT, whereas maximum disease severity was continuously observed in unsprayed control plot.

The data recorded at 105 DAT showed minimum disease severity i.e. 59.67 per cent in the plots protected with T_5 (SA-S+SL+SPY) treatment. While maximum disease severity 68.33 per cent was recorded

Table 20. Effect of RIC's on Leaf curl disease severity of tomato *var. Dhanashree* during *Kharif* 2015

Tr. No.	Treatment	Leaf curl disease severity * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	0.00 (0.00)	19.67 (26.288)	22.17 (28.055)	36.88 (37.372)	54.15 (47.363)	65.04 (53.739)	4.81
T_2	SA-SL	0.00 (0.00)	17.80 (24.910)	21.14 (27.337)	36.14 (36.931)	52.64 (46.496)	64.33 (53.314)	5.85
T_3	SA-SPY	0.00 (0.00)	14.10 (21.995)	14.93 (22.674)	31.61 (34.185)	48.88 (44.340)	60.96 (51.316)	10.79
T_4	SA-S+SL	0.00 (0.00)	14.33 (22.185)	16.83 (24.172)	33.58 (35.391)	50.41 (45.217)	62.22 (52.059)	8.94
T_5	SA-S+SL+SPY	0.00 (0.00)	10.40 (18.721)	12.06 (20.246)	29.67 (32.978)	45.67 (42.497)	59.67 (50.560)	12.67
T_6	BABA-S	0.00 (0.00)	25.27 (30.148)	27.79 (31.786)	45.79 (42.566)	58.15 (49.675)	67.05 (54.957)	1.87
T_7	BABA-SL	0.00 (0.00)	23.68 (29.086)	26.18 (30.746)	42.84 (40.863)	57.44 (49.263)	66.72 (54.756)	2.36
T_8	BABA-SPY	0.00 (0.00)	16.48 (23.902)	19.81 (26.389)	36.48 (37.134)	54.31 (47.456)	64.05 (53.146)	6.26
T_9	BABA-S+SL	0.00 (0.00)	23.37 (28.877)	26.71 (31.090)	42.54 (40.690)	56.98 (48.996)	66.34 (54.525)	2.91
T_{10}	BABA-S+SL + SPY	0.00 (0.00)	15.37 (23.028)	19.54 (26.194)	33.71 (35.469)	52.04 (46.152)	62.04 (51.952)	9.21
T_{11}	CHT-S	0.00 (0.00)	17.50 (24.684)	21.66 (27.701)	38.33 (38.230)	55.64 (48.222)	65.44 (53.980)	4.23
T_{12}	CHT-SL	0.00 (0.00)	16.95 (24.265)	20.28 (26.727)	36.95 (37.413)	55.08 (47.899)	65.04 (53.739)	4.81
T_{13}	CHT-SPY	0.00 (0.00)	17.64 (24.789)	20.14 (26.627)	34.31 (35.833)	51.04 (45.578)	62.54 (52.248)	8.47
T_{14}	CHT-S+SL	0.00 (0.00)	18.89 (25.720)	21.39 (27.512)	35.56 (36.584)	52.71 (46.536)	63.67 (52.919)	6.82
T_{15}	CHT-S+SL + SPY	0.00 (0.00)	11.77 (19.987)	15.10 (22.812)	33.56 (35.378)	49.78 (44.856)	61.88 (51.858)	9.44
T_{16}	Control	0.00 (0.00)	27.24 (31.433)	38.08 (38.082)	51.41 (45.790)	60.58 (51.093)	68.33 (55.742)	0.00
	S.E. $\pm$	0.00	0.119	0.105	0.022	0.005	0.013	---
	C.D. at 5 %	0.00	0.345	0.303	0.063	0.016	0.037	---

* : Mean of three replications, Figures in parentheses are angular transformed values

in T_{16} i.e. untreated control plot. In rest of the treatments the per cent disease severity was ranged between 60.96 (T_3) to 67.05 (T_6) per cent and most of them were at par with each other. The percent disease severity reduction was found maximum in treatment T_5 i.e. SA-S+SL+SPY (12.67 %), T_3 i.e. SA-SPY (10.79 %) followed by T_{15} i.e. CHT-S+SL+SPY (9.44 %).

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on disease severity presented in Table 21 revealed that the data followed the same pattern as recorded during *kharif* 2014 and *kharif* 2015. All the treatments significantly reduced the disease severity over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all treatments the pooled percent disease severity increased with age of the plants *i.e.* from 30 DAT to 105 DAT. However the per cent disease severity were considerably lower in RIC's treated plots. Also, the rate of percent disease severity increase was comparatively low in case of RIC's treated plots over control. The pooled disease severity ranged from 2.09 to 67.99 per cent throughout the crop growth period in comparison with 69.29 per cent recorded in control.

Result also indicated that the pooled leaf curl virus disease severity of tomato on *var. Dhanashree* was differ significantly due to different treatments of RIC's at 30, 45, 60, 75, 90 and 105 DAT. However the pooled severity of leaf curl virus disease was significantly minimum in plots treated with treatment T_5 (SA-S+SL+SPY) and showed the least disease severity from 30 DAT to 105 DAT. Whereas maximum disease severity was continuously observed in untreated control plot.

The pooled percent disease incidence in different treatments at 30, 45, 60, 75, 90 and 105 days after transplanting (DAT) demonstrated that the treatment T_5 (SA-S+SL+SPY) was significantly superior over rest of the treatment in their effectiveness against disease control. However the remaining treatments performed significantly better than control.

Table 21. Pooled data on effect of RIC's on Leaf curl disease severity of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Leaf curl disease severity * (%)						% Reduction over control
		30 DAT	45 DAT	60 DAT	75 DAT	90 DAT	105 DAT	
T_1	SA-S	4.17 (11.683)	15.25 (22.932)	23.86 (29.205)	38.03 (38.050)	55.30 (48.023)	66.19 (54.431)	4.48
T_2	SA-SL	2.92 (9.669)	13.49 (21.480)	22.66 (28.389)	37.24 (37.582)	53.74 (47.125)	65.43 (53.971)	5.58
T_3	SA-SPY	2.50 (8.891)	9.97 (18.303)	19.71 (26.314)	34.64 (36.029)	51.37 (45.767)	63.46 (52.794)	8.41
T_4	SA-S+SL	2.92 (9.669)	11.33 (19.588)	20.95 (27.199)	35.69 (36.662)	52.53 (46.433)	64.34 (53.320)	7.14
T_5	SA-S+SL+SPY	2.09 (8.019)	8.11 (16.418)	17.75 (24.872)	32.11 (34.490)	48.11 (43.895)	62.11 (51.991)	10.37
T_6	BABA-S	5.99 (14.111)	19.89 (26.444)	30.15 (33.276)	46.73 (43.109)	59.09 (50.219)	67.99 (55.529)	1.88
T_7	BABA-SL	5.84 (13.921)	18.93 (25.746)	28.51 (32.242)	43.92 (41.488)	58.52 (49.890)	67.80 (55.416)	2.15
T_8	BABA-SPY	4.59 (12.283)	14.49 (22.316)	24.49 (29.630)	38.66 (38.421)	56.49 (48.710)	66.23 (54.455)	4.42
T_9	BABA-S+SL	5.42 (13.392)	18.77 (25.632)	28.36 (32.147)	43.77 (41.401)	58.21 (49.709)	67.57 (55.274)	2.48
T_{10}	BABA-S+SL + SPY	4.17 (11.683)	13.94 (21.858)	23.11 (28.697)	36.86 (37.357)	55.19 (47.959)	65.19 (53.827)	5.92
T_{11}	CHT-S	4.59 (12.283)	15.42 (23.064)	25.83 (30.517)	40.00 (39.211)	57.31 (49.187)	67.11 (54.993)	3.15
T_{12}	CHT-SL	4.59 (12.283)	14.73 (22.508)	24.73 (29.787)	38.48 (38.315)	56.61 (48.779)	66.57 (54.661)	3.93
T_{13}	CHT-SPY	3.75 (11.057)	13.82 (21.762)	22.16 (28.045)	36.32 (37.038)	53.05 (46.731)	64.55 (53.445)	6.84
T_{14}	CHT-S+SL	4.17 (11.683)	14.86 (22.618)	23.20 (28.758)	37.37 (37.659)	54.52 (47.573)	65.48 (54.001)	5.51
T_{15}	CHT-S+SL + SPY	2.50 (8.891)	10.05 (18.386)	19.97 (26.505)	35.12 (36.317)	51.86 (46.048)	63.95 (53.086)	7.71
T_{16}	Control	6.67 (14.913)	25.71 (30.435)	37.79 (37.911)	52.37 (46.341)	61.54 (51.657)	69.29 (56.336)	0.00
	S.E. ±	0.205	0.149	0.058	0.016	0.006	0.012	----
	C.D. at 5 %	0.596	0.433	0.169	0.046	0.018	0.034	----

* : Mean of three replications, Figures in parentheses are angular transformed values

The data recorded at 105 DAT showed minimum pooled disease severity i.e. 62.11 per cent in the plots protected with T_5 (SA-S+SL+SPY) treatment. While maximum pooled disease severity 69.29 per cent was recorded in T_{16} i.e. unsprayed control plot. In rest of the treatments the per cent disease severity was ranged between 63.46 (T_3) to 67.99 (T_6) per cent. The percent disease severity reduction was found maximum in treatment T_5 i.e. SA-S+SL+SPY (10.37 %), T_3 i.e. SA-SPY (8.41 %) followed by T_{15} i. e. CHT-S+SL+SPY (7.71 %).

A perusal of the data of the effect of RIC's on leaf curl virus disease severity under field conditions indicated that the different treatments has effect on Leaf curl virus disease severity and considerable reduction in disease severity was observed when the number of sprays was increased. Critical analysis of the data revealed that the salicylic acid was found effective in reducing the disease severity as compared to other chemicals.

4.1.7 Effect of RIC's on Tomato spotted wilt virus disease incidence of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatment on Tomato spotted wilt virus disease incidence during *kharif* 2014 and 2015 crop season is presented in Table 22.

The data on percent disease incidence of Tomato spotted wilt virus disease during *kharif* 2014 and *kharif* 2015 was recorded at the end of season. It has been found that the percent disease incidence was higher during *Kharif* 2015 than *Kharif* 2014. In all treatments percent disease incidence were comparatively lower in RIC's treated plots. Data given in Table 22 revealed that incidence of Tomato spotted wilt virus of tomato on *var. Dhanashree* was differ due to different treatments of RIC's. Data on disease incidence also showed that among all RIC's tested, treatment T_5 (SA-S+SL+SPY) reduced the disease incidence compared to control.

Table 22. Effect of RIC's on Tomato spotted wilt virus disease incidence of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Per cent disease incidence*					
		2014	% reduction over control	2015	% reduction over control	Pooled	% reduction over control
T_1	SA-S	6.67 (14.753)	46.64	38.33 (38.224)	6.12	22.50 (28.288)	15.64
T_2	SA-SL	7.50 (15.744)	40.00	37.50 (37.704)	8.16	22.50 (28.267)	15.64
T_3	SA-SPY	5.83 (13.906)	53.36	35.83 (36.718)	12.25	20.83 (27.119)	21.90
T_4	SA-S+SL	7.50 (15.744)	40.00	38.33 (38.236)	6.12	22.92 (28.588)	14.06
T_5	SA-S+SL+SPY	5.00 (12.916)	60.00	35.00 (36.248)	14.28	20.00 (26.547)	25.01
T_6	BABA-S	10.83 (19.184)	13.36	40.00 (39.184)	2.03	25.42 (30.240)	4.69
T_7	BABA-SL	10.83 (19.184)	13.36	38.33 (38.201)	6.12	24.58 (29.681)	7.84
T_8	BABA-SPY	10.00 (18.337)	20.00	37.50 (37.641)	8.16	23.75 (29.021)	10.95
T_9	BABA-S+SL	9.17 (17.581)	26.67	38.33 (38.114)	6.12	23.75 (29.071)	10.95
T_{10}	BABA-S+SL + SPY	10.00 (18.337)	20.00	36.67 (37.235)	10.19	23.33 (28.851)	12.52
T_{11}	CHT-S	9.17 (17.581)	26.64	40.00 (39.184)	2.03	24.58 (29.674)	7.84
T_{12}	CHT-SL	8.33 (16.590)	33.36	39.17 (38.688)	4.07	23.75 (29.090)	10.95
T_{13}	CHT-SPY	7.50 (15.744)	40.00	36.67 (37.158)	10.19	22.08 (27.908)	17.21
T_{14}	CHT-S+SL	6.67 (14.753)	46.64	36.67 (37.235)	10.19	21.67 (27.703)	18.75
T_{15}	CHT-S+SL + SPY	5.83 (13.906)	53.36	35.83 (36.684)	12.25	20.83 (27.059)	21.90
T_{16}	Control	12.50 (20.634)	0.00	40.83 (39.692)	0.00	26.67 (31.077)	0.00
	S.E. $\pm$	1.323	--	2.231	--	1.411	--
	C.D. at 5 %	3.839	--	N/A	--	N/A	--

* : Mean of three replications, Figures in parentheses are angular transformed values

***Kharif* 2014**

Data recorded during *Kharif* 2014 indicated that effect of RIC's treatments on incidence of Tomato spotted wilt virus disease of tomato was significant. However the incidence of Tomato spotted wilt virus disease was significantly minimum in plots treated with treatment T_5 (SA-S+SL+SPY) i.e. 5.00 per cent which was at par with T_3 (SA-SPY) i.e. 5.83 per cent, T_{15} (CHT-S+SL+SPY) i.e. 5.83 per cent, T_1 (SA-S) i.e. 6.67 per cent, T_{14} (CHT-S+SL) 6.67 per cent, T_2 (SA-SL) i.e. 7.50 per cent, T_4 (SA-S+SL) i.e. 7.50 per cent, T_{13} (CHT-SPY) i.e. 7.50 per cent and T_{12} (CHT-SL) 8.33 per cent, whereas maximum disease incidence was observed in untreated control plot i.e. 12.50 per cent. In rest of the treatments the percent disease incidence was ranged from 9.17 (T_9) to 10.83 (T_6 and T_7) per cent. The percent disease severity reduction was highest recorded in T_5 i.e. SA- S+SL+SPY (60.00 %) followed by T_3 i.e. SA-SPY and T_{15} i.e. CHT-S+SL+SPY (53.36 %).

***Kharif* 2015**

The data recorded on percent disease incidence of Tomato spotted wilt virus on tomato plants *var. Dhanashree* during *kharif* 2015 (Table 22) revealed that all the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. The range of disease incidence in treatments varied from 35.00 to 40.00 per cent in comparison with 40.83 per cent recorded in control. There were no significant differences for any of the treatments on Tomato spotted wilt virus disease incidence during *Kharif* 2015. Comparatively highest reduction in percent disease incidence was observed in treatment T_5 of SA- S+ SL+ SPY (14.28 %) followed by treatment T_3 of SA-SPY and treatment T_{15} of CHT-S+SL+SPY (12.25 %).

Pooled data

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on disease incidence presented in Table 22 revealed that the

data followed the same pattern as recorded during previous crop season. All the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all treatments the pooled percent disease incidence were comparatively lower in RIC's treated plots. The pooled disease incidence ranged from 20.00 to 25.42 per cent throughout the crop growth period in comparison with 26.67 per cent recorded in control. There were no significant differences for any of the treatments on Tomato spotted wilt virus disease incidence during *Kharif* 2015. Comparatively highest reduction in percent disease incidence was observed in treatment T_5 of SA- S+ SL+ SPY (25.01 %) followed by treatment T_3 of SA-SPY and treatment T_{15} of CHT-S+SL+SPY (21.90 %).

4.1.8 Effect of RIC's on Tomato diseases AUDPC during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatment on AUDPC of Tomato diseases during *kharif* 2014 and 2015 crop season is presented in Table 23.

Disease symptoms appeared to start in both treated and untreated plots at about the same time, but subsequent disease progress was rapid in the non-treated plots and on the less frequently applied RIC's as indicated by their higher mean AUDPC values (Table 23). The AUDPC values of all diseases on tomato exhibited significant difference among the treatments. Minimum AUDPC was recorded on plots treated with different RIC's as compared to the untreated plot. Likewise, every week RIC's reduces the AUDPC values significantly as compared to all other treatments as well unsprayed control. The lowest AUDPC values were obtained from RIC's treated plots at every 15 days interval. However, the maximum AUDPC value was recorded on the untreated control (Table 23). Generally, when compared the different treatments among each other SA-S+SL+SPY treatment was found very effective than the other in reducing the AUDPC of Early blight and Leaf curl virus

Table 23. Effect of different RIC's on management of tomato diseases (AUDPC)

Tr. No.	Treatment	Disease Severity (AUDPC)								
		Early blight			Powdery Mildew			Leaf curl		
		2014	2015	Pooled	2014	2015	Pooled	2014	2015	Pooled
T_1	SA-S	1623.38	1270.88	1447.08	1470.53	1663.43	1566.98	2547.18	2298.98	2473.05
T_2	SA-SL	1580.63	1228.13	1404.35	1423.20	1616.10	1519.63	2440.10	2479.35	2509.73
T_3	SA-SPY	1485.75	1133.25	1309.48	1360.67	1553.57	1457.12	2359.70	2094.15	2264.45
T_4	SA-S+SL	1566.75	1214.25	1390.49	1576.38	1769.28	1672.79	2429.85	2447.63	2482.46
T_5	SA-S+SL+SPY	1464.00	1111.50	1287.78	1208.87	1401.77	1305.34	2230.60	2045.25	2162.95
T_6	BABA-S	1705.65	1353.15	1529.43	1351.95	1544.85	1448.43	2926.95	2936.85	3038.14
T_7	BABA-SL	1686.60	1334.10	1510.36	1282.80	1475.70	1379.21	2848.10	2799.53	2892.59
T_8	BABA-SPY	1650.38	1297.88	1474.10	1291.96	1484.86	1388.38	2699.15	2349.45	2568.05
T_9	BABA-S+SL	1664.25	1311.75	1488.03	1307.66	1500.56	1404.12	2826.35	2485.20	2712.04
T_{10}	BABA-S+SL + SPY	1605.00	1252.50	1428.79	1098.63	1291.53	1195.09	2637.43	2373.98	2536.98
T_{11}	CHT-S	1673.40	1320.90	1497.15	1652.40	1845.30	1748.83	2744.30	2630.78	2756.31
T_{12}	CHT-SL	1628.10	1275.60	1451.85	1604.33	1797.23	1700.79	2676.33	2298.98	2537.70
T_{13}	CHT-SPY	1568.78	1216.28	1392.46	1440.58	1633.48	1537.07	2468.90	2115.75	2329.80
T_{14}	CHT-S+SL	1619.48	1266.98	1443.24	1620.28	1813.18	1716.72	2536.90	2510.25	2567.30
T_{15}	CHT-S+SL + SPY	1531.88	1179.38	1355.65	1220.00	1412.90	1316.50	2389.35	2051.03	2245.23
T_{16}	Control	1721.63	1369.13	1545.39	1944.68	2137.58	2041.10	3214.35	3132.83	3286.06
	S.E. $\pm$	35.74	28.10	23.90	92.59	104.80	98.69	168.52	158.31	166.99
	C.D. at 5 %	103.71	81.55	69.36	268.72	304.14	286.42	489.08	459.45	484.63

*AUDPC- Area under disease progress curve

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disease of tomato; S+SL+SPY treatment of all the RIC's were also best in minimizing the AUDPC value of the diseases.

In case of early blight of tomato, treatment T_5 of SA-S+SL+SPY was found significantly superior over other treatments in minimizing the AUDPC value of disease which was at par with treatment T_3 of SA-SPY and treatment T_{15} of CHT-S+SL+SPY for both seasons and pooled too.

The AUDPC value of Powdery mildew disease of Tomato was found minimum in treatment T_{10} of BABA-S+SL+SPY which was at par with treatment T_5 of SA-S+SL+SPY and T_{15} of CHT-S+SL+SPY.

The Tomato leaf curl virus disease AUDPC value was found minimum in treatment T_5 of SA-S+SL+SPY which were significantly superior over other treatment. The highest AUDPC value was observed in untreated control.

4.1.9 Studies on effect of RIC's on yield of Tomato

The results obtained from the effect of different RIC's on yield of tomato *var. Dhanashree* have been presented under the following headings.

Effect of RIC's on yield of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different resistance inducing chemicals (RIC's) treatment on yield of Tomato during *kharif* 2014 and 2015 crop season is presented in Table 24.

The data pertinent to tomato yield (t/ha) as affected by various treatments of RIC's during *Kharif* 2014 and *Kharif* 2015 crop season indicated that the application of RIC's significantly influenced the yield of tomato.

***Kharif* 2014**

During *Kharif* 2014, the yield was ranged from 23.28 to 35.63 t/ha as against least yield 22.35 t/ha in untreated control. The per cent increase in yield with various treatments, over untreated control

Table 24. Effect of RIC's on yield of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Tr. No.	Treatment	Yield (Tonnes/ha)*					
		2014	% increase over control	2015	% increase over control	Pooled	% increase over control
T_1	SA-S	29.26	30.92	27.30	43.38	28.28	36.62
T_2	SA-SL	32.37	44.83	29.22	53.47	30.80	48.77
T_3	SA-SPY	34.39	53.87	31.58	65.86	32.99	59.35
T_4	SA-S+SL	32.19	44.03	29.08	52.73	30.64	48.00
T_5	SA-S+SL+SPY	35.63	59.42	31.78	66.91	33.71	62.83
T_6	BABA-S	23.28	4.16	23.54	23.63	23.41	13.09
T_7	BABA-SL	24.12	7.92	23.92	25.63	24.02	16.04
T_8	BABA-SPY	27.88	24.74	24.62	29.31	26.25	26.81
T_9	BABA-S+SL	24.48	9.53	25.60	34.45	25.04	20.97
T_{10}	BABA-S+SL + SPY	31.48	40.85	28.31	48.69	29.90	44.42
T_{11}	CHT-S	25.35	13.42	24.04	26.26	24.70	19.30
T_{12}	CHT-SL	30.31	35.62	27.68	45.38	29.00	40.07
T_{13}	CHT-SPY	33.07	47.96	29.98	57.46	31.53	52.29
T_{14}	CHT-S+SL	28.88	29.22	26.40	38.66	27.64	33.53
T_{15}	CHT-S+SL + SPY	34.04	52.30	30.28	59.03	32.16	55.36
T_{16}	Control	22.35	0.00	19.04	0.00	20.70	0.00
	S.E. $\pm$	1.85	--	1.73	--	1.46	--
	C.D. at 5 %	5.38	--	5.03	--	4.24	--

* : Mean of three replications

was ranged from 4.16 to 59.42 per cent. Among all the treatments, significantly highest yield (35.63 t/ha) was recorded with the treatment T_5 of SA-S+SL+SPY which was at par with treatment T_3 of SA-SPY (34.39 t/ha), T_{15} of SA-SPY (34.04 t/ha), T_{13} of CHT-SPY (33.07 t/ha), T_2 of SA-SL (32.37 t/ha), T_4 of SA-S+SL (32.19t/ha), T_{10} of BABA-S+SL+SPY (31.48 t/ha) and T_{12} of CHT-SL (30.31t/ha). The highest increase in yield over control was observed in treatment T_5 of SA-S+SL+SPY i.e. (59.42 %) followed by treatment i.e. T_3 of SA-SPY 53.87 per cent and T_{15} of CHT-S+SL+SPY i.e. 52.30 per cent.

***Kharif* 2015**

Similar observations regarding yield were recorded during *Kharif* 2015 crop season. The yield was ranged from 23.54 to 31.78 t/ha. as against least yield 19.04 t/ha in untreated control. The per cent increase in yield with various treatments, over untreated control was ranged from 23.63 to 66.91 per cent. Among all the treatments, significantly highest yield (31.78 t/ha) was recorded with the treatment T_5 of SA-S+SL+SPY which was at par with treatment T_3 of SA-SPY (31.58 t/ha), T_{15} of CHT-S+SL+SPY (30.28 t/ha), T_{13} of CHT-SPY (29.98 t/ha), T_2 of SA-SL (29.22 t/ha), T_4 of SA-S+SL (29.08 t/ha), T_{10} of BABA-S+SL+SPY (28.31 t/ha), T_{12} of CHT-SL (27.68 t/ha) and T_1 of SA-S (27.30 t/ha). The highest increase in yield over control was observed in treatment T_5 of SA-S+SL+SPY i.e. (66.91 %) followed by treatment T_3 of SA-SPY i.e. 65.86 per cent and T_{15} of CHT-S+SL+SPY i.e. 59.03 per cent.

Pooled data

The pooled data showed the similar trend regarding the yield of tomato. The pooled yield was ranged from 23.41 to 33.71 t/ha. as against least yield 20.70t/ha in untreated control. The per cent increase in yield with various treatments, over untreated control was ranged from 13.09 to 62.83 per cent. Among all the treatments, significantly highest yield (33.71 t/ha) was recorded with the treatment T_5 of SA-S+SL+SPY which was at par with treatment T_3 of SA-SPY (32.99 t/ha), T_{15} of CHT-

S+SL+SPY (32.16 t/ha), T_{13} of CHT-SPY (31.53 t/ha), T_2 of SA-SL (30.80 t/ha), T_4 of SA-S+SL (30.64 t/ha), T_{10} of BABA-S+SL+SPY (29.90 t/ha) and T_{12} of CHT-SL (29.00 t/ha). The highest increase in yield over control was observed in treatment T_5 of SA-S+SL+SPY i.e. (62.83 %) followed by treatment T_3 of SA-SPY i.e. 59.35 per cent and T15 of CHT-S+SL+SPY i.e. 55.36 per cent.

Critical analysis of data revealed that, among RIC's tested, the salicylic acid was found most effective in increasing yield of tomato.

4.2 Studies on anatomical changes in cellular leaf structure of tomato due to resistance inducing chemicals

The resistance inducing chemicals particularly salicylic acid, β–aminobutyric acid and chitosan treated plants were used for studies on anatomical changes in cellular structures of tomato leaves.

The microscopic observations revealed that the application of salicylic acid, β–aminobutyric acid and chitosan as seed treatment, seedling treatment, spray treatment, seed + seedling treatment and seed + seedling + spray treatment increased the thickness of epidermis cum cuticle layer and palisade mesophyll cells (Plate 3A, 3B and 3C) of tomato leaves and the increased was measured by using micrometry.

4.2.1 Changes in epidermis cum cuticle layer thickness of tomato leaves due to application of different resistance inducing chemicals

Data presented in Table 25 and Fig. 7 clearly showed that all assigned treatments of RIC's significantly increased epidermis cum cuticle layer thickness in both studied seasons (*kharif* 2014 and *kharif* 2015). The increase in thickness of epidermis cum cuticle due to RIC's treatments was in the range of 12.38 to 77.72 per cent and 12.28 to 76.00 per cent for both studied season. The most effective treatment which induced highest increase in epidermis cum cuticle layer thickness was treatment T_5 (SA-S+SL+SPY).

Table 25. Variations in epidermis-cum-cuticle thickness of tomato leaves due to different resistance inducing chemicals

Tr. No.	Treatment	Thickness of epidermis cum cuticle (µm)			% increase in thickness of epidermis cum cuticle over control		
		2014	2015	Pooled	2014	2015	Pooled
T_1	SA-S	20.56 (3.99)	20.46 (3.91)	20.51 (3.95)	24.10	23.65	23.87
T_2	SA-SL	22.96 (6.39)	22.82 (6.27)	22.89 (6.33)	38.53	37.85	38.19
T_3	SA-SPY	28.16 (11.59)	27.86 (11.31)	28.01 (11.45)	69.95	68.34	69.14
T_4	SA-S+SL	27.34 (10.77)	27.08 (10.53)	27.21 (10.65)	65.02	63.65	64.33
T_5	SA-S+SL+SPY	29.45 (12.88)	29.13 (12.58)	29.29 (12.73)	77.72	76.00	76.86
T_6	BABA-S	18.62 (2.05)	18.58 (2.03)	18.60 (2.04)	12.38	12.28	12.33
T_7	BABA-SL	19.71 (3.14)	19.63 (3.08)	19.67 (3.11)	18.93	18.59	18.76
T_8	BABA-SPY	24.76 (8.19)	24.56 (8.01)	24.66 (8.10)	49.41	48.38	48.89
T_9	BABA-S+SL	25.45 (8.88)	25.23 (8.68)	25.34 (8.78)	53.59	52.45	53.02
T_{10}	BABA-S+SL + SPY	25.76 (9.19)	25.52 (8.97)	25.64 (9.08)	55.44	54.18	54.81
T_{11}	CHT-S	19.63 (3.06)	19.57 (3.02)	19.60 (3.04)	18.45	18.23	18.34
T_{12}	CHT-SL	21.80 (5.23)	21.68 (5.13)	21.74 (5.18)	31.53	30.97	31.25
T_{13}	CHT-SPY	23.12 (6.55)	22.96 (6.41)	23.04 (6.48)	39.51	38.71	39.11
T_{14}	CHT-S+SL	24.63 (8.06)	24.45 (7.90)	24.54 (7.98)	48.62	47.71	48.17
T_{15}	CHT-S+SL + SPY	27.45 (10.88)	27.17 (10.62)	27.31 (10.75)	65.66	64.17	64.92
T_{16}	Control	16.57 (0.00)	16.55 (0.00)	16.56 (0.00)	0.00	0.00	0.00
	S.E. $\pm$	0.14	0.29	0.22	--	--	--
	C.D. at 5 %	0.42	0.84	0.63	--	--	--

*Figures in parenthesis shows the increase in thickness (µm) over control.

The epidermis cum cuticle thickness due to treatment T_5 (SA-S+SL+SPY) was 29.45 µm and 29.13 µm in both studied season respectively, which is followed by treatment T_3 of SA-SPY and treatment T_{15} of CHT-S+SL+SPY. In this respect, the maximum significant increase was detected in treatment T_5 (SA-S+SL+SPY) i.e. 77.72 and 76.00 per cent more than those of the control plant in the first and second season; respectively. Worthy to note that the significantly minimum thickness was observed in control plot.

Similar results were obtained for pooled data. The increase in epidermis cum cuticle thickness due to RIC's treatments was in the range of 12.33 to 76.86 per cent over the control. Maximum increase in the epidermal thickness was observed in treatment T_5 of SA-S+SL+SPY (76.86 %) over the control and it was 29.29 µm in thickness.

4.2.2 Changes in palisade mesophyll layer thickness of tomato leaves due to application of different resistance inducing chemicals

Data presented in Table 26 and Fig. 8 clearly showed that all assigned treatments of RIC's significantly increased palisade mesophyll layer thickness in both studied seasons (*kharif* 2014 and 2015). The increase in thickness of palisade mesophyll due to RIC's treatments was in the range of 4.47 to 28.26 per cent and 3.57 to 27.37 per cent for both studied season. The most effective treatment which induced highest increase in palisade mesophyll thickness was treatment T_5 (SA-S+SL+SPY) which was at par with treatment T_3 (SA-SPY) followed by treatment T_{15} (CHT-S+SL+SPY).

The palisade mesophyll thickness due to treatment T_5 (SA-S+SL+SPY) was 152.67 and 152.83 µm in *kharif* 2014 and *kharif* 2015 season respectively, which is followed by treatment T_3 of SA-SPY and treatment T_{15} of CHT-S+SL+SPY. In this respect, the maximum significant increase was detected in treatment T_5 (SA-S+SL+SPY) i.e. 28.26 and 27.37 per cent more than those of the control plant in the first

Table 26. Changes in thickness of palisade mesophyll of tomato leaves due to different resistance inducing chemicals

Tr. No.	Treatment	Thickness of palisade mesophyll (µm)			% increase in thickness of palisade mesophyll over control		
		2014	2015	Pooled	2014	2015	Pooled
T_1	SA-S	125.26 (6.23)	125.42 (5.43)	125.34 (5.83)	5.23	4.53	4.88
T_2	SA-SL	128.88 (9.85)	129.04 (9.05)	128.96 (9.45)	8.28	7.55	7.91
T_3	SA-SPY	152.37 (33.34)	152.53 (32.54)	152.45 (32.94)	28.01	27.12	27.57
T_4	SA-S+SL	130.29 (11.26)	130.45 (10.46)	130.37 (10.86)	9.46	8.72	9.09
T_5	SA-S+SL+SPY	152.67 (33.64)	152.83 (32.84)	152.75 (33.24)	28.26	27.37	27.81
T_6	BABA-S	125.79 (6.76)	125.95 (5.96)	125.87 (6.36)	5.68	4.96	5.32
T_7	BABA-SL	129.51 (10.48)	129.67 (9.68)	129.59 (10.08)	8.81	8.06	8.43
T_8	BABA-SPY	130.43 (11.40)	130.59 (10.60)	130.51 (11.00)	9.58	8.83	9.20
T_9	BABA-S+SL	134.91 (15.88)	135.07 (15.08)	134.99 (15.48)	13.34	12.57	12.95
T_{10}	BABA-S+SL + SPY	136.42 (17.39)	136.71 (16.72)	136.57 (17.06)	14.61	13.94	14.27
T_{11}	CHT-S	124.35 (5.32)	124.27 (4.28)	124.31 (4.80)	4.47	3.57	4.02
T_{12}	CHT-SL	127.31 (8.28)	126.14 (6.15)	126.72 (7.21)	6.96	5.12	6.04
T_{13}	CHT-SPY	128.59 (9.56)	127.59 (7.60)	128.09 (8.58)	8.03	6.33	7.18
T_{14}	CHT-S+SL	129.48 (10.45)	129.57 (9.58)	129.53 (10.02)	8.78	7.99	8.38
T_{15}	CHT-S+SL + SPY	148.15 (29.12)	148.31 (28.32)	148.23 (28.72)	24.46	23.60	24.03
T_{16}	Control	119.03 (0.00)	119.99 (0.00)	119.51 (0.00)	0.00	0.00	0.00
	S.E. ±	1.33	0.51	0.74	--	--	--
	C.D. at 5 %	3.86	1.48	2.14	--	--	--

*Figures in parenthesis shows the increase in thickness (µm) over control.

and second season; respectively. Worthy to note that the significantly minimum thickness was observed in control plot i.e. 119.03 and 119.99 µm, respectively.

Similar results were obtained for pooled data. The increase in palisade mesophyll thickness due to RIC's treatments was in the range of 4.02 to 27.81 per cent over the control. Maximum increase in the epidermal thickness was observed in treatment T_5 of SA-S+SL+SPY (27.81 %) over the control and it was 152.75 µm in thickness.

4.2.3 Changes in stomatal frequency, stomatal size of tomato leaves due to application of different resistance inducing chemicals

Tomato leaves were critically examined to assess the differences in their stomatal characteristics *viz*. stomatal frequency, stomatal length and width.

From the data obtained (Table 27; Plate 4 and 5), it was evident that irrespective of RIC's treatments the stomatal frequency (No. of stomata/ mm^2) was fairly high on lower (abaxial) leaf surface than the upper (adaxial) leaf surface for both studied season (*kharif* 2014 and *kharif* 2015). The result also showed that different RIC's treatments have no significant effect on stomatal frequency of adaxial and abaxial surface. Relatively higher reduction in stomatal frequency was observed in treatment T_5 of SA-S+SL+SPY as compared to control.

The data presented in Table 28, Fig. 9 and Fig. 10 showed that all RIC's treatments significantly affect the stomatal length and width. When comparison were made between different treatments it was evident that treatment T_{10} (BABA-S+SL+SPY) significantly reduces the stomatal length and width of stomata for both studied season.

The reduction in length of stomata due to RIC's treatments was in the range of 6.06 - 24.71 per cent and 10.44 - 32.70 per cent, whereas the reduction in width was 1.75 - 19.30 per cent and 13.22 - 35.15 per cent for both studied season respectively. The most effective

Table 27. Changes in stomatal frequency of tomato leaves due to different resistance inducing chemicals

Tr. No.	Treatment	Stomatal Frequency (per mm²)*											
		2014				2015				Pooled			
		Adaxial surface	% reduction over control	Adaxial surface	% reduction over control	Adaxial surface	% reduction over control	Adaxial surface	% reduction over control	Adaxial surface	% reduction over control	Adaxial surface	% reduction over control
T_1	SA-S	35.61	5.32	128.11	3.42	38.71	5.22	131.21	2.32	37.16	5.28	129.66	2.86
T_2	SA-SL	34.81	7.44	122.51	7.64	37.29	8.69	124.99	6.95	36.05	8.11	123.75	7.29
T_3	SA-SPY	31.40	16.51	112.24	15.39	33.36	18.32	114.20	14.98	32.38	17.46	113.22	15.18
T_4	SA-S+SL	32.08	14.70	115.28	13.09	33.65	17.61	116.85	13.01	32.87	16.22	116.07	13.05
T_5	SA-S+SL+SPY	30.45	19.05	110.06	17.03	32.48	20.46	111.38	17.08	31.46	19.79	110.72	17.05
T_6	BABA-S	36.44	3.11	117.03	11.78	39.54	3.18	120.13	10.56	37.99	3.16	118.58	11.16
T_7	BABA-SL	35.17	6.49	130.61	1.54	37.65	7.81	133.09	0.92	36.41	7.19	131.85	1.22
T_8	BABA-SPY	32.48	13.64	114.72	13.52	34.44	15.67	116.68	13.13	33.46	14.71	115.70	13.32
T_9	BABA-S+SL	33.64	10.56	120.04	9.51	35.21	13.79	121.61	9.46	34.43	12.25	120.83	9.48
T_{10}	BABA-S+SL + SPY	32.28	14.17	115.10	13.23	33.60	17.73	116.42	13.33	32.94	16.03	115.76	13.28
T_{11}	CHT-S	36.04	4.17	129.08	2.69	39.14	4.16	132.18	1.59	37.59	4.18	130.63	2.14
T_{12}	CHT-SL	34.98	6.99	124.56	6.10	37.46	8.28	127.04	5.42	36.22	7.67	125.80	5.75
T_{13}	CHT-SPY	31.96	15.02	113.78	14.23	33.92	16.94	115.74	13.83	32.94	16.03	114.76	14.02
T_{14}	CHT-S+SL	32.96	12.36	115.78	12.72	34.53	15.45	117.35	12.63	33.75	13.98	116.57	12.67
T_{15}	CHT-S+SL + SPY	30.85	17.98	112.04	15.54	33.30	18.46	113.36	15.60	32.07	18.24	112.70	15.57
T_{16}	Control	37.61	0.00	132.65	0.00	40.84	0.00	134.32	0.00	39.23	0.00	133.48	0.00
	S.E. ±	2.14	---	7.77	---	2.53	---	7.87	---	2.06	---	7.82	---
	C.D. at 5 %	N/A	---	N/A	---	N/A	---	N/A	---	N/A	---	N/A	---

*Mean of ten observations

Table 28. Changes in stomatal size of tomato leaves due to different resistance inducing chemicals

Tr. No.	Treatment	Stomatal size*											
		2014				2015				Pooled			
		Stomatal length	% reduction over control	Stomatal width	% reduction over control	Stomatal length	% reduction over control	Stomatal width	% reduction over control	Stomatal length	% reduction over control	Stomatal width	% reduction over control
T_1	SA-S	20.15	6.06	13.44	1.75	23.25	10.44	16.54	13.22	21.70	8.48	14.99	8.43
T_2	SA-SL	19.38	9.65	12.92	5.56	21.86	15.79	15.40	19.20	20.62	13.03	14.16	13.50
T_3	SA-SPY	18.09	15.66	12.61	7.82	20.05	22.77	14.57	23.56	19.07	19.57	13.59	16.98
T_4	SA-S+SL	18.17	15.29	12.21	10.75	20.13	22.46	14.17	25.66	19.15	19.23	13.19	19.43
T_5	SA-S+SL+SPY	16.95	20.98	11.34	17.11	18.27	29.62	12.66	33.58	17.61	25.73	12.00	26.70
T_6	BABA-S	19.91	7.18	12.91	5.63	23.01	11.36	16.01	16.00	21.46	9.49	14.46	11.67
T_7	BABA-SL	19.26	10.21	12.86	5.99	21.74	16.26	15.34	19.52	20.50	13.54	14.10	13.87
T_8	BABA-SPY	16.56	22.80	11.18	18.27	17.88	31.12	12.50	34.42	17.22	27.37	11.84	27.67
T_9	BABA-S+SL	17.20	19.81	11.76	14.04	18.77	27.70	13.33	30.06	17.99	24.15	12.55	23.37
T_{10}	BABA-S+SL + SPY	16.15	24.71	11.04	19.30	17.47	32.70	12.36	35.15	16.81	29.10	11.70	28.53
T_{11}	CHT-S	20.15	6.06	13.01	4.90	23.25	10.44	16.11	15.48	21.70	8.48	14.56	11.06
T_{12}	CHT-SL	19.72	8.07	12.89	5.77	22.20	14.48	15.37	19.36	20.96	11.60	14.13	13.68
T_{13}	CHT-SPY	17.86	16.74	12.28	10.23	19.82	23.65	14.24	25.29	18.84	20.54	13.26	19.00
T_{14}	CHT-S+SL	18.58	13.38	11.89	13.08	20.15	22.38	13.46	29.38	19.37	18.33	12.68	22.57
T_{15}	CHT-S+SL + SPY	18.01	16.04	12.04	11.99	19.58	24.58	13.61	28.59	18.80	20.73	12.83	21.66
T_{16}	Control	21.45	0.00	13.68	0.00	25.96	0.00	19.06	0.00	23.71	0.00	16.37	0.00
	S.E. $\pm$	0.58	---	0.14	---	0.29	---	0.29	---	0.43	---	0.22	---
	C.D. at 5 %	1.67	---	0.42	---	0.84	---	0.84	---	1.25	---	0.63	---

*Mean of ten observations

treatment which induced highest reduction in stomatal length and width was treatment T10 (BABA-S+SL+SPY) which was at par with treatment T_5 (SA-S+SL+SPY) and treatment T_{15} (CHT-S+SL+SPY).

The stomatal length and width due to treatment T_{10} (BABA-S+SL+SPY) was 16.15 µm×11.04µm and 17.47µm×12.36µm in both studied season (*kharif* 2014 and 2015) respectively, which is followed by treatment T_5 of SA-S+SL+SPY and treatment T_{15} of CHT-S+SL+SPY. In this respect, the maximum significant reduction was detected in treatment T_{10} (BABA-S+SL+SPY) i.e. 24.71 and 32.70 per cent more than those of the control plant in the first and second season; respectively. Worthy to note that the significantly highest length and width were observed in control plots i.e. 21.45 × 13.68 µm and 25.96 ×19.06 µm, respectively.

Similar results were obtained for pooled data. The reduction in stomatal length and width due to RIC's treatment was in the range of 8.48 to 29.10 per cent and 8.43 to 28.53 per cent over control. Maximum reduction in the stomatal length and width was observed in treatment T_{10} of BABA-S+SL+SPY (29.10 %) and (28.53 %) over the control which was at par with treatment T_5 of SA-S+SL+SPY and T_{15} of CHT-S+SL+SPY. In case of pooled stomatal length treatment T_9 of BABA-S+SL was also found at par with treatment T_{10} (BABA-S+SL+SPY). The stomatal size in treatment T_{10} (BABA-S+SL+SPY) was 16.81 × 11.70 µm whereas in control was 23.71 x 16.37 µm.

4.3 Effect of RIC's on Biochemical content of tomato *var. Dhanashree* during *Kharif* 2014 and *Kharif* 2015

Quantitative changes in plant defence related biochemical constituents of tomato leaves due to different resistance inducing chemicals were studied in respect of soluble protein content, reducing sugar content, phenol content, lignin content, phenyl alanine ammonia lyase activity, peroxidase activity, polyphenol oxidase activity, β-1,3 glucanase activity and chitinase activity.

4.3.1 Soluble protein content

In the present investigations it was noticed that there was higher soluble protein content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015 (Table 29 and 30). All assigned treatments of RIC's significantly increased the soluble protein content of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons (*kharif* 2014 and *kharif* 2015), as compare to control. Among the treatments used, treatment T_5 (SA-S+SL+SPY) showed maximum increasing soluble protein content during both season. The change in content of soluble protein due to different RIC's treatments showed increase in trend from 30 DAT to 105 DAT. It indicated that soluble protein content increased with aging.

During both the season, more amount of soluble protein content was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), 15.79 and 16.98 mg gm^{-1} of fresh weight at 105 DAT respectively. There was an increase of 53.90 and 48.95 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. SA-SPY treatment was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of soluble protein content. A slight increase in soluble protein content was seen in untreated control plant. This result showed that RIC's could induce soluble protein content at higher level in infected plant as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in soluble protein content was presented in Table 31 and Fig. 11 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the soluble protein content. The change in soluble protein content due to different RIC's treatments showed increasing trend from 30 DAT to 105 DAT. The higher level of soluble protein content was observed in plants treated with treatment T_5 i.e. (SA-

Table 29. Soluble protein content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Soluble protein content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in soluble protein content over control	60 DAT	Per cent increase in soluble protein content over control	90 DAT	Per cent increase in soluble protein content over control	105 DAT	Per cent increase in soluble protein content over control
T_1	SA-S	6.56 (1.81)	38.11	10.37 (2.99)	40.45	12.09 (2.33)	23.83	12.20 (1.94)	18.91
T_2	SA-SL	7.79 (3.04)	63.89	10.02 (2.64)	35.77	12.11 (2.35)	24.06	12.11 (1.85)	18.03
T_3	SA-SPY	8.82 (4.07)	85.58	11.42 (4.04)	54.77	14.02 (4.26)	43.61	14.20 (3.94)	38.40
T_4	SA-S+SL	8.08 (3.33)	70.02	10.93 (3.55)	48.05	13.08 (3.32)	34.04	13.42 (3.16)	30.80
T_5	SA-S+SL+SPY	9.10 (4.35)	91.58	12.62 (5.24)	71.06	14.15 (4.39)	45.00	15.79 (5.53)	53.90
T_6	BABA-S	6.15 (1.40)	29.56	9.54 (2.16)	29.27	11.94 (2.18)	22.30	11.94 (1.68)	16.37
T_7	BABA-SL	6.86 (2.11)	44.46	9.81 (2.43)	32.98	12.01 (2.25)	23.05	12.42 (2.16)	21.05
T_8	BABA-SPY	7.77 (3.02)	63.54	10.03 (2.65)	35.88	12.87 (3.11)	31.81	13.08 (2.82)	27.49
T_9	BABA-S+SL	7.75 (3.00)	63.05	10.47 (3.09)	41.80	13.01 (3.25)	33.25	13.24 (2.98)	29.04
T_{10}	BABA-S+SL + SPY	8.08 (3.33)	70.00	11.01 (3.63)	49.12	13.29 (3.53)	36.21	14.02 (3.76)	36.65
T_{11}	CHT-S	6.38 (1.63)	34.23	9.81 (2.43)	32.98	12.01 (2.25)	23.05	12.34 (2.08)	20.27
T_{12}	CHT-SL	6.36 (1.61)	33.89	9.78 (2.40)	32.55	12.28 (2.52)	25.80	12.54 (2.28)	22.22
T_{13}	CHT-SPY	7.82 (3.07)	64.63	10.26 (2.88)	39.04	13.07 (3.31)	33.86	13.20 (2.94)	28.65
T_{14}	CHT-S+SL	7.77 (3.02)	63.54	10.66 (3.28)	44.42	13.06 (3.30)	33.79	13.10 (2.84)	27.68
T_{15}	CHT-S+SL + SPY	8.75 (4.00)	84.21	11.07 (3.69)	50.01	13.82 (4.06)	41.56	14.08 (3.82)	37.23
T_{16}	Control	4.75 (0.00)	0.00	7.38 (0.00)	0.00	9.76 (0.00)	0.00	10.26 (0.00)	0.00
	S.E. ±	0.043		0.056		0.063		0.051	
	C.D. at 5 %	0.125		0.163		0.181		0.147	

Figures in parenthesis indicates increase in soluble protein content (mg) over control

Table 30. Soluble protein content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Soluble protein content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in soluble protein content over control	60 DAT	Per cent increase in soluble protein content over control	90 DAT	Per cent increase in soluble protein content over control	105 DAT	Per cent increase in soluble protein content over control
T_1	SA-S	8.76 (2.27)	34.95	12.04 (2.44)	25.31	14.14 (3.14)	28.58	15.02 (3.62)	31.75
T_2	SA-SL	10.00 (3.51)	54.04	12.24 (2.64)	27.37	14.33 (3.33)	30.25	14.38 (2.98)	26.14
T_3	SA-SPY	10.99 (4.50)	69.40	13.74 (4.14)	42.98	16.14 (5.14)	46.69	16.64 (5.24)	45.96
T_4	SA-S+SL	10.04 (3.55)	54.70	12.92 (3.32)	34.42	15.28 (4.28)	38.95	15.87 (4.47)	39.21
T_5	SA-S+SL+SPY	11.10 (4.61)	70.97	14.44 (4.84)	50.24	16.37 (5.37)	48.84	16.98 (5.58)	48.95
T_6	BABA-S	8.18 (1.69)	26.01	11.40 (1.80)	18.67	13.71 (2.71)	24.62	13.74 (2.34)	20.53
T_7	BABA-SL	9.10 (2.61)	40.22	11.66 (2.06)	21.29	12.89 (1.89)	17.18	11.92 (0.52)	4.56
T_8	BABA-SPY	9.98 (3.49)	53.84	12.03 (2.43)	25.22	15.06 (4.06)	36.88	15.52 (4.12)	36.14
T_9	BABA-S+SL	9.98 (3.49)	53.73	12.23 (2.63)	27.21	15.02 (4.02)	36.54	15.65 (4.25)	37.28
T_{10}	BABA-S+SL + SPY	10.09 (3.60)	55.42	13.00 (3.40)	35.32	15.50 (4.50)	40.89	16.20 (4.80)	42.11
T_{11}	CHT-S	8.15 (1.66)	25.64	10.93 (1.33)	13.74	12.02 (1.02)	9.29	11.92 (0.52)	4.56
T_{12}	CHT-SL	8.61 (2.12)	32.67	11.98 (2.38)	24.68	14.03 (3.03)	27.55	14.35 (2.95)	25.88
T_{13}	CHT-SPY	9.42 (2.93)	45.15	12.48 (2.88)	29.89	15.15 (4.15)	37.74	15.84 (4.44)	38.95
T_{14}	CHT-S+SL	9.63 (3.14)	48.35	12.62 (3.02)	31.36	15.01 (4.01)	36.49	15.20 (3.80)	33.33
T_{15}	CHT-S+SL + SPY	10.64 (4.15)	63.90	13.10 (3.50)	36.28	16.03 (5.03)	45.69	16.54 (5.14)	45.09
T_{16}	Control	6.49 (0.00)	0.00	9.61 (0.00)	0.00	11.00 (0.00)	0.00	11.40 (0.00)	0.00
	S.E. ±	0.048		0.054		0.032		0.433	
	C.D. at 5 %	0.140		0.157		0.094		1.253	

Figures in parenthesis indicates increase in soluble protein content (mg) over control

Table 31. Pooled data on soluble protein content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Soluble protein content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in soluble protein content over control	60 DAT	Per cent increase in soluble protein content over control	90 DAT	Per cent increase in soluble protein content over control	105 DAT	Per cent increase in soluble protein content over control
T_1	SA-S	7.66 (2.04)	36.28	11.20 (2.71)	31.96	13.12 (2.74)	26.35	13.61 (2.78)	25.67
T_2	SA-SL	8.89 (3.27)	58.20	11.13 (2.64)	31.10	13.22 (2.84)	27.34	13.25 (2.42)	22.30
T_3	SA-SPY	9.90 (4.28)	76.24	12.58 (4.09)	48.19	15.08 (4.70)	45.24	15.42 (4.59)	42.38
T_4	SA-S+SL	9.06 (3.44)	61.17	11.92 (3.43)	40.42	14.18 (3.80)	36.64	14.65 (3.82)	35.23
T_5	SA-S+SL+SPY	10.10 (4.48)	79.68	13.53 (5.04)	59.38	15.26 (4.88)	47.03	16.39 (5.56)	51.29
T_6	BABA-S	7.17 (1.55)	27.51	10.47 (1.98)	23.35	12.82 (2.44)	23.53	12.84 (2.01)	18.56
T_7	BABA-SL	7.98 (2.36)	42.01	10.74 (2.25)	26.44	11.95 (1.57)	15.13	12.17 (1.34)	12.37
T_8	BABA-SPY	8.88 (3.26)	57.94	11.03 (2.54)	29.93	13.96 (3.58)	34.50	14.30 (3.47)	32.04
T_9	BABA-S+SL	8.86 (3.24)	57.67	11.35 (2.86)	33.63	14.01 (3.63)	34.99	14.45 (3.62)	33.38
T_{10}	BABA-S+SL + SPY	9.08 (3.46)	61.58	12.00 (3.51)	41.40	14.40 (4.02)	38.69	15.11 (4.28)	39.52
T_{11}	CHT-S	7.27 (1.65)	29.27	10.37 (1.88)	22.17	11.52 (1.14)	10.94	12.13 (1.30)	12.00
T_{12}	CHT-SL	7.49 (1.87)	33.19	10.88 (2.39)	28.18	13.15 (2.77)	26.72	13.45 (2.62)	24.15
T_{13}	CHT-SPY	8.62 (3.00)	53.38	11.37 (2.88)	33.94	14.11 (3.73)	35.92	14.52 (3.69)	34.07
T_{14}	CHT-S+SL	8.70 (3.08)	54.77	11.64 (3.15)	37.12	14.04 (3.66)	35.22	14.15 (3.32)	30.66
T_{15}	CHT-S+SL + SPY	9.69 (4.07)	72.48	12.08 (3.59)	42.33	14.92 (4.54)	43.75	15.31 (4.48)	41.37
T_{16}	Control	5.62 (0.00)	0.00	8.49 (0.00)	0.00	10.38 (0.00)	0.00	10.83 (0.00)	0.00
	S.E. ±	0.046		0.055		0.043		0.242	
	C.D. at 5 %	0.132		0.159		0.123		0.699	

Figures in parenthesis indicates increase in soluble protein content (mg) over control

S+SL+SPY), 16.39 mg gm^{-1} of fresh weight. There was an increase of 51.29 per cent as compare to control at 105 DAT. The treatment T_3 i.e. SA-SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Untreated control exhibited least amount of soluble protein content. A slight increase in soluble protein content was seen in untreated control. This result showed that RIC's could induce higher level of soluble protein content in infected plant as compare to untreated control.

4.3.2 Reducing sugar content

In the present investigations it was noticed that there was higher reducing sugar content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015 (Table 32 and 33). All assigned treatments of RIC's significantly increased the reducing sugar content of tomato in all the treatments at 30 DAT, 60 DAT, 90 DAT and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_{10} (BABA-S+SL+SPY) showed maximum increase in reducing sugar content during both season. The change in content of reducing sugar due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity decreased from 30 to 60 DAT but increased from 90 to105 DAT.

During both the season, the great amount of reducing sugar content was observed at 105 DAT in plants treated with treatment T_{10} i.e. (BABA-S+SL+SPY), 4.15 and 4.60 mg gm^{-1} of fresh weight respectively. There was an increase of 97.62 and 91.67 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. The treatment T_5 i.e. SA-S+SL+SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Both treatments were found at par with treatment T_{10} (BABA-S+SL+SPY). Untreated control exhibited least amount of reducing sugar content. The large amount of decrease followed by slight increase in reducing sugar content was seen in untreated control plant from 30 DAT to 105 DAT. This result showed that RIC's could induce

Table 32. Reducing sugar content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Reducing sugar content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in reducing sugar content over control	60 DAT	Per cent increase in reducing sugar content over control	90 DAT	Per cent increase in reducing sugar content over control	105 DAT	Per cent increase in reducing sugar content over control
T_1	SA-S	4.08 (0.34)	9.09	2.07 (0.29)	16.07	2.21 (0.27)	14.12	2.47 (0.37)	17.62
T_2	SA-SL	4.12 (0.38)	10.16	2.14 (0.36)	20.34	2.14 (0.20)	10.41	2.32 (0.22)	10.48
T_3	SA-SPY	4.20 (0.46)	12.30	2.10 (0.32)	17.87	2.15 (0.21)	10.72	2.64 (0.54)	25.71
T_4	SA-S+SL	4.25 (0.51)	13.50	2.48 (0.70)	39.33	2.48 (0.54)	27.63	2.56 (0.46)	21.90
T_5	SA-S+SL+SPY	5.21 (1.47)	39.36	3.42 (1.64)	92.13	3.28 (1.34)	69.07	4.02 (1.92)	91.43
T_6	BABA-S	4.19 (0.45)	12.09	2.42 (0.64)	35.96	2.18 (0.24)	12.58	2.58 (0.48)	22.86
T_7	BABA-SL	4.51 (0.77)	20.48	2.69 (0.91)	50.90	2.56 (0.62)	31.96	2.96 (0.86)	40.95
T_8	BABA-SPY	4.64 (0.90)	24.12	2.59 (0.81)	45.73	2.68 (0.74)	38.25	3.06 (0.96)	45.71
T_9	BABA-S+SL	4.58 (0.84)	22.57	2.60 (0.82)	46.18	2.61 (0.67)	34.43	2.96 (0.86)	40.95
T_{10}	BABA-S+SL + SPY	5.26 (1.52)	40.75	3.48 (1.70)	95.73	3.36 (1.42)	73.40	4.15 (2.05)	97.62
T_{11}	CHT-S	4.96 (1.22)	32.73	2.83 (1.05)	58.99	2.87 (0.93)	47.94	3.76 (1.66)	79.05
T_{12}	CHT-SL	4.65 (0.91)	24.44	2.54 (0.76)	42.81	2.54 (0.60)	30.72	2.54 (0.44)	20.95
T_{13}	CHT-SPY	4.65 (0.91)	24.39	2.70 (0.92)	51.57	2.86 (0.92)	47.32	3.22 (1.12)	53.33
T_{14}	CHT-S+SL	4.25 (0.51)	13.74	2.52 (0.74)	41.57	2.53 (0.59)	30.41	2.84 (0.74)	35.24
T_{15}	CHT-S+SL + SPY	5.18 (1.44)	38.61	3.32 (1.54)	86.74	3.26 (1.32)	67.84	3.92 (1.82)	86.67
T_{16}	Control	3.74 (0.00)	0.00	1.78 (0.00)	0.00	1.94 (0.00)	0.00	2.10 (0.00)	0.00
	S.E. ±	0.144		0.121		0.104		0.081	
	C.D. at 5 %	0.418		0.351		0.301		0.234	

Figures in parenthesis indicates increase in reducing sugar content (mg) over control

Table 33. Reducing sugar content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Reducing sugar content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in reducing sugar content over control	60 DAT	Per cent increase in reducing sugar content over control	90 DAT	Per cent increase in reducing sugar content over control	105 DAT	Per cent increase in reducing sugar content over control
T_1	SA-S	4.65 (0.71)	18.02	2.24 (0.21)	10.34	3.22 (0.86)	36.27	3.36 (0.96)	40.00
T_2	SA-SL	4.82 (0.88)	22.34	2.38 (0.35)	17.29	3.35 (0.99)	42.03	3.36 (0.96)	40.00
T_3	SA-SPY	4.98 (1.04)	26.40	2.52 (0.49)	24.14	3.48 (1.12)	47.63	3.64 (1.24)	51.67
T_4	SA-S+SL	4.90 (0.96)	24.37	2.61 (0.58)	28.62	3.70 (1.34)	56.61	3.52 (1.12)	46.67
T_5	SA-S+SL+SPY	5.21 (1.27)	32.23	3.29 (1.26)	62.17	4.52 (2.16)	91.69	4.55 (2.15)	89.58
T_6	BABA-S	4.62 (0.68)	17.26	2.40 (0.37)	18.13	3.56 (1.20)	50.85	3.28 (0.88)	36.67
T_7	BABA-SL	4.74 (0.80)	20.36	2.73 (0.70)	34.68	3.97 (1.61)	68.22	3.94 (1.54)	64.17
T_8	BABA-SPY	4.80 (0.86)	21.88	2.37 (0.34)	16.55	3.96 (1.60)	67.80	4.02 (1.62)	67.50
T_9	BABA-S+SL	4.81 (0.87)	21.98	2.69 (0.66)	32.61	3.98 (1.62)	68.81	3.87 (1.47)	61.25
T_{10}	BABA-S+SL + SPY	5.31 (1.37)	34.82	3.36 (1.33)	65.52	4.55 (2.19)	92.71	4.60 (2.20)	91.67
T_{11}	CHT-S	4.99 (1.05)	26.55	2.68 (0.65)	32.12	4.09 (1.73)	73.14	4.15 (1.75)	72.92
T_{12}	CHT-SL	4.73 (0.79)	20.15	2.35 (0.32)	15.76	3.84 (1.48)	62.54	3.51 (1.11)	46.25
T_{13}	CHT-SPY	4.98 (1.04)	26.50	2.52 (0.49)	24.14	3.95 (1.59)	67.37	4.02 (1.62)	67.50
T_{14}	CHT-S+SL	4.70 (0.76)	19.39	2.81 (0.78)	38.42	3.89 (1.53)	64.83	3.62 (1.22)	50.83
T_{15}	CHT-S+SL + SPY	5.11 (1.17)	29.64	3.00 (0.97)	47.98	4.48 (2.12)	89.75	4.50 (2.10)	87.50
T_{16}	Control	3.94 (0.00)	0.00	2.03 (0.00)	0.00	2.36 (0.00)	0.00	2.40 (0.00)	0.00
	S.E. $\pm$	0.115		0.087		0.069		0.052	
	C.D. at 5 %	0.334		0.251		0.200		0.150	

Figures in parenthesis indicates increase in reducing sugar content (mg) over control

Table 34. Pooled data on reducing sugar content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Reducing sugar content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in reducing sugar content over control	60 DAT	Per cent increase in reducing sugar content over control	90 DAT	Per cent increase in reducing sugar content over control	105 DAT	Per cent increase in reducing sugar content over control
T_1	SA-S	4.37 (0.53)	13.67	2.15 (0.24)	12.72	2.72 (0.57)	26.28	2.92 (0.67)	29.56
T_2	SA-SL	4.47 (0.63)	16.41	2.26 (0.35)	18.40	2.75 (0.60)	27.77	2.84 (0.59)	26.22
T_3	SA-SPY	4.59 (0.75)	19.53	2.31 (0.40)	20.89	2.82 (0.67)	30.98	3.14 (0.89)	39.56
T_4	SA-S+SL	4.57 (0.73)	19.08	2.55 (0.64)	33.27	3.09 (0.94)	43.53	3.04 (0.79)	35.11
T_5	SA-S+SL+SPY	5.21 (1.37)	35.70	3.36 (1.45)	75.71	3.90 (1.75)	81.49	4.29 (2.04)	90.44
T_6	BABA-S	4.41 (0.57)	14.74	2.41 (0.50)	26.13	2.87 (0.72)	33.58	2.93 (0.68)	30.22
T_7	BABA-SL	4.62 (0.78)	20.42	2.71 (0.80)	41.88	3.27 (1.12)	51.86	3.45 (1.20)	53.33
T_8	BABA-SPY	4.72 (0.88)	22.97	2.48 (0.57)	29.84	3.32 (1.17)	54.47	3.54 (1.29)	57.33
T_9	BABA-S+SL	4.70 (0.86)	22.27	2.65 (0.74)	38.59	3.30 (1.15)	53.30	3.42 (1.17)	51.78
T_{10}	BABA-S+SL + SPY	5.29 (1.45)	37.71	3.42 (1.51)	79.16	3.96 (1.81)	84.00	4.38 (2.13)	94.44
T_{11}	CHT-S	4.98 (1.14)	29.56	2.76 (0.85)	44.29	3.48 (1.33)	61.77	3.96 (1.71)	75.78
T_{12}	CHT-SL	4.69 (0.85)	22.24	2.45 (0.54)	28.06	3.19 (1.04)	48.19	3.03 (0.78)	34.44
T_{13}	CHT-SPY	4.82 (0.98)	25.47	2.61 (0.70)	36.60	3.40 (1.25)	58.33	3.62 (1.37)	60.89
T_{14}	CHT-S+SL	4.48 (0.64)	16.64	2.67 (0.76)	39.53	3.21 (1.06)	49.30	3.23 (0.98)	43.56
T_{15}	CHT-S+SL + SPY	5.15 (1.31)	34.01	3.16 (1.25)	65.65	3.87 (1.72)	79.86	4.21 (1.96)	87.11
T_{16}	Control	3.84 (0.00)	0.00	1.91 (0.00)	0.00	2.15 (0.00)	0.00	2.25 (0.00)	0.00
	S.E. $\pm$	0.13		0.104		0.087		0.066	
	C.D. at 5 %	0.376		0.301		0.251		0.192	

Figures in parenthesis indicates increase in reducing sugar content (mg) over control

reducing sugar content at higher level in infected plant as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in reducing sugar content was presented in Table 34 and Fig. 12 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the reducing sugar content. The change in reducing sugar content due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity decreased from 30 to 60 DAT but increased from 90 to 105 DAT. The higher level of reducing sugar content was observed in plants treated with treatment T_{10} i.e. (BABA-S+SL+SPY), 4.38 mg gm^{-1} of fresh weight at 105 DAT. There was an increase of 94.44 per cent as compare to control at 105 DAT. The treatment T_5 i.e. SA-S+SL+SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Both treatments were found at par with treatment T_{10} (BABA-S+SL+SPY). Untreated control exhibited least amount of reducing sugar content. The large amount of decrease followed by slight increase in reducing sugar content was seen in untreated control plant from 30 to 105 DAT. This result showed that RIC's could induce reducing sugar content at higher level in infected plant as compare to untreated control.

4.3.3 Total phenol content

In the present investigations it was noticed that total phenol content was higher in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015 (Table 35 and 36). All assigned treatments of RIC's significantly increased the total phenol content of tomato leaves in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_5 (SA-S+SL+SPY) showed maximum increase in total phenol content during both season. The change in total

phenol content due to different RIC's treatments showed increasing trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

During both the season, the higher amount of total phenol content was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY) at 105 DAT, 2.08 and 2.10 mg gm^{-1} of fresh weight respectively. There was an increase of 38.67 and 41.89 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. SA-SPY treatment was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of total phenol content. A slight increase in total phenol content was seen in untreated control plant initially up to 90 DAT there after it was observed decreasing. This result showed that RIC's could induce total phenol content at higher level in infected plants as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in total phenol content was presented in Table 37 and Fig. 13 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the total phenol content. The change in total phenol content due to different RIC's treatments showed increasing trend from 30 DAT to 90 DAT, whereas the activity decreased at 105 DAT. The higher level of total phenol content was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), 2.09 mg gm^{-1} of fresh weight. There was an increase of 40.27 per cent as compare to control at 105 DAT. The treatment T_3 i.e. SA-SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Untreated control exhibited least amount of total phenol content. A slight increase in total phenol content was seen in untreated control plant initially up to 90 DAT there after it was observed decreasing. This result showed that RIC's could induce higher level of total phenol content in infected plants as compare to untreated control.

Table 35. Total phenol content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Total phenol content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in total phenol content over control	60 DAT	Per cent increase in total phenol content over control	90 DAT	Per cent increase in total phenol content over control	105 DAT	Per cent increase in total phenol content over control
T_1	SA-S	1.46 (0.08)	5.80	1.60 (0.04)	2.56	1.70 (0.08)	4.94	1.62 (0.12)	8.00
T_2	SA-SL	1.54 (0.16)	11.59	1.75 (0.19)	12.18	2.02 (0.40)	24.69	1.67 (0.17)	11.33
T_3	SA-SPY	1.80 (0.42)	30.43	2.10 (0.54)	34.62	2.68 (1.06)	65.43	1.82 (0.32)	21.33
T_4	SA-S+SL	1.65 (0.27)	19.57	1.85 (0.29)	18.59	2.16 (0.54)	33.33	1.69 (0.19)	12.67
T_5	SA-S+SL+SPY	2.10 (0.72)	52.17	2.34 (0.78)	50.00	2.84 (1.22)	75.31	2.08 (0.58)	38.67
T_6	BABA-S	1.40 (0.02)	1.45	1.65 (0.09)	5.77	1.80 (0.18)	11.11	1.58 (0.08)	5.33
T_7	BABA-SL	1.46 (0.08)	5.80	1.72 (0.16)	10.26	1.81 (0.19)	11.73	1.74 (0.24)	16.00
T_8	BABA-SPY	1.54 (0.16)	11.59	1.80 (0.24)	15.38	1.96 (0.34)	20.99	1.65 (0.15)	10.00
T_9	BABA-S+SL	1.63 (0.25)	18.12	1.77 (0.21)	13.46	2.05 (0.43)	26.54	1.71 (0.21)	14.00
T_{10}	BABA-S+SL + SPY	1.68 (0.30)	21.74	1.87 (0.31)	19.87	2.24 (0.62)	38.27	1.75 (0.25)	16.67
T_{11}	CHT-S	1.43 (0.05)	3.62	1.65 (0.09)	5.77	1.65 (0.03)	1.85	1.60 (0.10)	6.67
T_{12}	CHT-SL	1.40 (0.02)	1.45	1.73 (0.17)	10.90	1.85 (0.23)	14.20	1.65 (0.15)	10.00
T_{13}	CHT-SPY	1.52 (0.14)	10.14	1.81 (0.25)	16.03	2.00 (0.38)	23.46	1.70 (0.20)	13.33
T_{14}	CHT-S+SL	1.56 (0.18)	13.04	1.77 (0.21)	13.46	1.78 (0.16)	9.88	1.74 (0.24)	16.00
T_{15}	CHT-S+SL + SPY	1.72 (0.34)	24.64	2.06 (0.50)	32.05	2.67 (1.05)	64.81	1.78 (0.28)	18.67
T_{16}	Control	1.38 (0.00)	0.00	1.56 (0.00)	0.00	1.62 (0.00)	0.00	1.50 (0.00)	0.00
	S.E. ±	0.017		0.012		0.023		0.029	
	C.D. at 5 %	0.050		0.033		0.067		0.084	

Figures in parenthesis indicates increase in total phenol content (mg) over control

Table 36. Total phenol content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Total phenol content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in total phenol content over control	60 DAT	Per cent increase in total phenol content over control	90 DAT	Per cent increase in total phenol content over control	105 DAT	Per cent increase in total phenol content over control
T_1	SA-S	1.51 (0.09)	6.34	1.65 (0.11)	7.14	1.92 (0.27)	16.36	1.58 (0.10)	6.76
T_2	SA-SL	1.54 (0.12)	8.45	1.74 (0.20)	12.99	2.04 (0.39)	23.64	1.65 (0.17)	11.49
T_3	SA-SPY	1.76 (0.34)	23.94	2.08 (0.54)	35.06	2.65 (1.00)	60.61	1.80 (0.32)	21.62
T_4	SA-S+SL	1.60 (0.18)	12.68	1.84 (0.30)	19.48	2.12 (0.47)	28.48	1.68 (0.20)	13.51
T_5	SA-S+SL+SPY	2.04 (0.62)	43.66	2.36 (0.82)	53.25	2.80 (1.15)	69.70	2.10 (0.62)	41.89
T_6	BABA-S	1.46 (0.04)	2.82	1.64 (0.10)	6.49	1.78 (0.13)	7.88	1.58 (0.10)	6.76
T_7	BABA-SL	1.50 (0.08)	5.63	1.70 (0.16)	10.39	1.86 (0.21)	12.73	1.62 (0.14)	9.46
T_8	BABA-SPY	1.58 (0.16)	11.27	1.78 (0.24)	15.58	1.98 (0.33)	20.00	1.68 (0.20)	13.51
T_9	BABA-S+SL	1.62 (0.20)	14.08	1.80 (0.26)	16.88	2.10 (0.45)	27.27	1.70 (0.22)	14.86
T_{10}	BABA-S+SL + SPY	1.68 (0.26)	18.31	1.85 (0.31)	20.13	2.15 (0.50)	30.30	1.71 (0.23)	15.54
T_{11}	CHT-S	1.45 (0.03)	2.11	1.62 (0.08)	5.19	1.83 (0.18)	10.91	1.58 (0.10)	6.76
T_{12}	CHT-SL	1.48 (0.06)	4.23	1.72 (0.18)	11.69	1.92 (0.27)	16.36	1.64 (0.16)	10.81
T_{13}	CHT-SPY	1.50 (0.08)	5.63	1.79 (0.25)	16.23	1.98 (0.33)	20.00	1.68 (0.20)	13.51
T_{14}	CHT-S+SL	1.52 (0.10)	7.04	1.75 (0.21)	13.64	2.01 (0.36)	21.82	1.70 (0.22)	14.86
T_{15}	CHT-S+SL + SPY	1.69 (0.27)	19.01	2.04 (0.50)	32.47	2.64 (0.99)	60.00	1.72 (0.24)	16.22
T_{16}	Control	1.42 (0.00)	0.00	1.54 (0.00)	0.00	1.65 (0.00)	0.00	1.48 (0.00)	0.00
	S.E. ±	0.012		0.023		0.017		0.029	
	C.D. at 5 %	0.033		0.067		0.050		0.084	

Figures in parenthesis indicates increase in total phenol content (mg) over control

Table 37. Pooled data on total phenol content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Total phenol content (mg gm^{-1} of fresh weight)							
		30 DAT	Per cent increase in total phenol content over control	60 DAT	Per cent increase in total phenol content over control	90 DAT	Per cent increase in total phenol content over control	105 DAT	Per cent increase in total phenol content over control
T_1	SA-S	1.49 (0.09)	6.07	1.63 (0.08)	4.84	1.81 (0.17)	10.37	1.60 (0.11)	7.38
T_2	SA-SL	1.54 (0.14)	10.00	1.75 (0.20)	12.58	2.03 (0.39)	23.78	1.66 (0.17)	11.41
T_3	SA-SPY	1.78 (0.38)	27.14	2.09 (0.54)	34.84	2.67 (1.03)	62.50	1.81 (0.32)	21.48
T_4	SA-S+SL	1.63 (0.23)	16.07	1.85 (0.30)	19.03	2.14 (0.50)	30.49	1.69 (0.20)	13.09
T_5	SA-S+SL+SPY	2.07 (0.67)	47.86	2.35 (0.80)	51.61	2.82 (1.18)	71.95	2.09 (0.60)	40.27
T_6	BABA-S	1.43 (0.03)	2.14	1.65 (0.09)	6.13	1.79 (0.15)	9.15	1.58 (0.09)	6.04
T_7	BABA-SL	1.48 (0.08)	5.71	1.71 (0.16)	10.32	1.84 (0.20)	11.89	1.68 (0.19)	12.75
T_8	BABA-SPY	1.56 (0.16)	11.43	1.79 (0.24)	15.48	1.97 (0.33)	20.12	1.67 (0.18)	11.74
T_9	BABA-S+SL	1.63 (0.23)	16.07	1.79 (0.24)	15.16	2.08 (0.44)	26.52	1.71 (0.22)	14.43
T_{10}	BABA-S+SL + SPY	1.68 (0.28)	20.00	1.86 (0.31)	20.00	2.20 (0.56)	33.84	1.73 (0.24)	16.11
T_{11}	CHT-S	1.44 (0.04)	2.86	1.64 (0.08)	5.48	1.74 (0.10)	6.10	1.59 (0.10)	6.71
T_{12}	CHT-SL	1.44 (0.04)	2.86	1.73 (0.18)	11.29	1.89 (0.25)	14.94	1.65 (0.16)	10.40
T_{13}	CHT-SPY	1.51 (0.11)	7.86	1.80 (0.25)	16.13	1.99 (0.35)	21.34	1.69 (0.20)	13.42
T_{14}	CHT-S+SL	1.54 (0.14)	10.00	1.76 (0.21)	13.55	1.90 (0.26)	15.55	1.72 (0.23)	15.44
T_{15}	CHT-S+SL + SPY	1.71 (0.31)	21.79	2.05 (0.50)	32.26	2.66 (1.02)	61.89	1.75 (0.26)	17.45
T_{16}	Control	1.40 (0.00)	0.00	1.55 (0.00)	0.00	1.64 (0.00)	0.00	1.49 (0.00)	0.00
	S.E. ±	0.015		0.017		0.020		0.029	
	C.D. at 5 %	0.042		0.050		0.059		0.084	

Figures in parenthesis indicates increase in total phenol content (mg) over control

4.3.4 Lignin content

In the present investigations it was noticed that there was higher lignin content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015 (Table 38 and 39). All assigned treatments of RIC's significantly increased the lignin content of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_{10} (BABA-S+SL+SPY) showed maximum increase in lignin content during both season. The change in lignin content due to different RIC's treatments showed increase in trend from 30 DAT to 105 DAT.

During both the season, the higher amount of lignin content was observed in plants treated with treatment T_{10} i.e. (BABA-S+SL+SPY) at 105 DAT, 2.66 and 2.68 per cent, respectively. There was an increase of 83.45 and 79.87 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. SA-S+SL+SPY treatment was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of lignin content. A slight increase in lignin content was seen in untreated control plant up to 105 DAT. This result showed that RIC's could induce lignin content at higher level in infected plants as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in lignin content was presented in Table 40 and Fig. 14 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the lignin content. The change in lignin content due to different RIC's treatments showed increase in trend from 30 DAT to 105 DAT. The higher level of lignin content was observed in plants treated with treatment T_{10} of BABA-S+SL+SPY i.e. 2.67 per cent. There was an increase of 81.63 per cent as compare to control at 105 DAT. The treatment T_5 i.e. SA-S+SL+SPY and treatment T_{15} i.e. CHT-S+SL+SPY was

Table 38. Lignin content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Lignin content (%)							
		30 DAT	Per cent increase in lignin content over control	60 DAT	Per cent increase in lignin content over control	90 DAT	Per cent increase in lignin content over control	105 DAT	Per cent increase in lignin content over control
T_1	SA-S	1.20 (0.22)	22.45	1.32 (0.24)	22.22	1.54 (0.19)	14.07	1.61 (0.16)	11.03
T_2	SA-SL	1.12 (0.14)	14.29	1.28 (0.20)	18.52	1.60 (0.25)	18.52	1.54 (0.09)	6.21
T_3	SA-SPY	1.30 (0.32)	32.65	1.71 (0.63)	58.33	2.04 (0.69)	51.11	2.31 (0.86)	59.31
T_4	SA-S+SL	1.25 (0.27)	27.55	1.64 (0.56)	51.85	2.19 (0.84)	62.22	2.15 (0.70)	48.28
T_5	SA-S+SL+SPY	1.42 (0.44)	44.90	1.83 (0.75)	69.44	2.44 (1.09)	80.74	2.63 (1.18)	81.38
T_6	BABA-S	1.28 (0.30)	30.61	1.35 (0.27)	25.00	1.85 (0.50)	37.04	2.24 (0.79)	54.48
T_7	BABA-SL	1.05 (0.07)	7.14	1.38 (0.30)	27.78	1.50 (0.15)	11.11	1.52 (0.07)	4.83
T_8	BABA-SPY	1.38 (0.40)	40.82	1.68 (0.60)	55.56	2.22 (0.87)	64.44	2.38 (0.93)	64.14
T_9	BABA-S+SL	1.34 (0.36)	36.73	1.58 (0.50)	46.30	2.25 (0.90)	66.67	2.36 (0.91)	62.76
T_{10}	BABA-S+SL + SPY	1.54 (0.56)	57.14	1.87 (0.79)	73.15	2.48 (1.13)	83.70	2.66 (1.21)	83.45
T_{11}	CHT-S	1.13 (0.15)	15.31	1.24 (0.16)	14.81	1.37 (0.02)	-3.70	1.59 (0.14)	9.66
T_{12}	CHT-SL	1.02 (0.04)	4.08	1.18 (0.10)	9.26	1.42 (0.07)	5.19	1.48 (0.03)	2.07
T_{13}	CHT-SPY	1.24 (0.26)	26.53	1.40 (0.32)	29.63	1.94 (0.59)	43.70	1.99 (0.54)	37.24
T_{14}	CHT-S+SL	1.12 (0.14)	14.29	1.33 (0.25)	23.15	1.56 (0.21)	15.56	1.54 (0.09)	6.21
T_{15}	CHT-S+SL + SPY	1.42 (0.44)	44.90	1.71 (0.63)	58.33	2.27 (0.92)	68.15	2.43 (0.98)	67.59
T_{16}	Control	0.98 (0.00)	0.00	1.08 (0.00)	0.00	1.35 (0.00)	0.00	1.45 (0.00)	0.00
	S.E. ±	0.017		0.012		0.023		0.029	
	C.D. at 5 %	0.050		0.033		0.067		0.084	

Figures in parenthesis indicates increase in lignin content (%) over control

Table 39. Lignin content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Lignin content (%)							
		30 DAT	Per cent increase in lignin content over control	60 DAT	Per cent increase in lignin content over control	90 DAT	Per cent increase in lignin content over control	105 DAT	Per cent increase in lignin content over control
T_1	SA-S	1.22 (0.26)	27.08	1.34 (0.24)	21.82	1.56 (0.20)	14.71	1.64 (0.15)	10.07
T_2	SA-SL	1.05 (0.09)	9.38	1.26 (0.16)	14.55	1.48 (0.12)	8.82	1.56 (0.07)	4.70
T_3	SA-SPY	1.35 (0.39)	40.63	1.72 (0.62)	56.36	2.06 (0.70)	51.47	2.30 (0.81)	54.36
T_4	SA-S+SL	1.30 (0.34))	35.42	1.55 (0.45)	40.91	2.20 (0.84)	61.76	2.18 (0.69)	46.31
T_5	SA-S+SL+SPY	1.44 (0.48)	50.00	1.80 (0.70)	63.64	2.42 (1.06)	77.94	2.62 (1.13)	75.84
T_6	BABA-S	1.22 (0.26)	27.08	1.36 (0.26)	23.64	1.87 (0.51)	37.50	2.26 (0.77)	51.68
T_7	BABA-SL	1.20 (0.24)	25.00	1.28 (0.18)	16.36	1.62 (0.26)	19.12	1.52 (0.03)	2.01
T_8	BABA-SPY	1.39 (0.43)	44.79	1.67 (0.57)	51.82	2.25 (0.89)	65.44	2.42 (0.93)	62.42
T_9	BABA-S+SL	1.42 (0.46)	47.92	1.55 (0.45)	40.91	2.28 (0.92)	67.65	2.40 (0.91)	61.07
T_{10}	BABA-S+SL + SPY	1.52 (0.56)	58.33	1.86 (0.76)	69.09	2.46 (1.10)	80.88	2.68 (1.19)	79.87
T_{11}	CHT-S	1.15 (0.19)	19.79	1.26 (0.16)	14.55	1.40 (0.04)	2.94	1.58 (0.09)	6.04
T_{12}	CHT-SL	1.12 (0.16)	16.67	1.20 (0.10)	9.09	1.44 (0.08)	5.88	1.51 (0.02)	1.34
T_{13}	CHT-SPY	1.22 (0.26)	27.08	1.38 (0.28)	25.45	1.95 (0.59)	43.38	2.02 (0.53)	35.57
T_{14}	CHT-S+SL	1.20 (0.24)	25.00	1.45 (0.35)	31.82	1.54 (0.18)	13.24	1.55 (0.06)	4.03
T_{15}	CHT-S+SL + SPY	1.28 (0.32)	33.33	1.73 (0.63)	57.27	2.28 (0.92)	67.65	2.45 (0.96)	64.43
T_{16}	Control	0.96 (0.00)	0.00	1.10 (0.00)	0.00	1.36 (0.00)	0.00	1.49 (0.00)	0.00
	S.E. ±	0.012		0.023		0.017		0.029	
	C.D. at 5 %	0.033		0.067		0.050		0.084	

Figures in parenthesis indicates increase in lignin content (%) over control

Table 40. Pooled data on lignin content in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Lignin content (%)							
		30 DAT	Per cent increase in lignin content over control	60 DAT	Per cent increase in lignin content over control	90 DAT	Per cent increase in lignin content over control	105 DAT	Per cent increase in lignin content over control
T_1	SA-S	1.21 (0.24)	24.74	1.33 (0.24)	22.02	1.55 (0.19)	13.97	1.63 (0.16)	10.54
T_2	SA-SL	1.09 (0.12)	11.86	1.27 (0.18)	16.51	1.54 (0.18)	13.24	1.55 (0.08)	5.44
T_3	SA-SPY	1.33 (0.36)	36.60	1.72 (0.63)	57.34	2.05 (0.69)	50.74	2.31 (0.84)	56.80
T_4	SA-S+SL	1.28 (0.31)	31.44	1.60 (0.51)	46.33	2.20 (0.84)	61.40	2.17 (0.70)	47.28
T_5	SA-S+SL+SPY	1.43 (0.46)	47.42	1.82 (0.73)	66.51	2.43 (1.07)	78.68	2.63 (1.16)	78.57
T_6	BABA-S	1.25 (0.28)	28.87	1.36 (0.27)	24.31	1.86 (0.50)	36.76	2.25 (0.78)	53.06
T_7	BABA-SL	1.13 (0.16)	15.98	1.33 (0.24)	22.02	1.56 (0.20)	14.71	1.52 (0.05)	3.40
T_8	BABA-SPY	1.39 (0.42)	42.78	1.68 (0.59)	53.67	2.24 (0.88)	64.34	2.40 (0.93)	63.27
T_9	BABA-S+SL	1.38 (0.41)	42.27	1.57 (0.48)	43.58	2.27 (0.91)	66.54	2.38 (0.91)	61.90
T_{10}	BABA-S+SL + SPY	1.53 (0.56)	57.73	1.87 (0.78)	71.10	2.47 (1.11)	81.62	2.67 (1.20)	81.63
T_{11}	CHT-S	1.14 (0.17)	17.53	1.25 (0.16)	14.68	1.39 (0.02)	1.84	1.59 (0.12)	7.82
T_{12}	CHT-SL	1.07 (0.10)	10.31	1.19 (0.10)	9.17	1.43 (0.07)	5.15	1.50 (0.02)	1.70
T_{13}	CHT-SPY	1.23 (0.26)	26.80	1.39 (0.30)	27.52	1.95 (0.59)	43.01	2.01 (0.54)	36.39
T_{14}	CHT-S+SL	1.16 (0.19)	19.59	1.39 (0.30)	27.52	1.55 (0.19)	13.97	1.55 (0.08)	5.10
T_{15}	CHT-S+SL + SPY	1.35 (0.38)	39.18	1.72 (0.63)	57.80	2.28 (0.92)	67.28	2.44 (0.97)	65.99
T_{16}	Control	0.97 (0.00)	0.00	1.09 (0.00)	0.00	1.36 (0.00)	0.00	1.47 (0.00)	0.00
	S.E. ±	0.014		0.017		0.02		0.029	
	C.D. at 5 %	0.042		0.050		0.058		0.084	

Figures in parenthesis indicates increase in lignin content (%) over control

proved to be the second and third best treatments. Untreated control exhibited least amount of lignin content (1.47 %). A slight increase in lignin content was seen in untreated control plant up to 105 DAT. This result showed that RIC's could induce higher level of lignin content in infected plants as compare to untreated control.

4.3.5 PAL activity

In the present investigations it was noticed that there was higher PAL activity in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015 (Table 41 and 42). All assigned treatments of RIC's significantly increased the PAL activity of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_5 (SA-S+SL+SPY) showed maximum increase in PAL activity during both season. The change in activity of PAL due to different RIC's treatments showed increasing trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

During both the season, the higher amount of PAL activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), at 105 DAT, 12.822 and 11.35 µ moles of cinnamic acid produced/min./ mg soluble protein respectively. There was an increase of 40.15 and 61.41 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. Treatment SA-SPY was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of PAL activity. A slight increase in PAL activity was seen in untreated control plant initially up to 90 DAT and the PAL activity was further decreased upto 105 DAT. This result showed that RIC's could induce PAL activity at higher level in infected plants as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in PAL activity was presented in Table 43 and Fig. 15 revealed that the data followed the same pattern as recorded

Table 41. Phenyl alanine ammonia lyase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Phenyl alanine ammonia lyase activity (µ moles of cinnamic acid produced min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in PAL activity over control	60 DAT	Per cent increase in PAL activity over control	90 DAT	Per cent increase in PAL activity over control	105 DAT	Per cent increase in PAL activity over control
T_1	SA-S	9.31 (2.63)	39.41	11.03 (3.31)	42.90	11.96 (1.96)	19.56	11.51 (2.36)	25.75
T_2	SA-SL	9.58 (2.90)	43.41	9.62 (1.90)	24.55	11.95 (1.95)	19.48	11.47 (2.32)	25.33
T_3	SA-SPY	10.68 (4.00)	59.93	12.12 (4.40)	56.93	12.54 (2.54)	25.38	12.18 (3.03)	33.09
T_4	SA-S+SL	9.92 (3.24)	48.54	11.42 (3.70)	47.89	12.31 (2.31)	23.10	11.86 (2.71)	29.62
T_5	SA-S+SL+SPY	11.06 (4.38)	65.56	12.90 (5.18)	67.10	13.02 (3.02)	30.24	12.82 (3.67)	40.15
T_6	BABA-S	8.12 (1.44)	21.56	9.76 (2.04)	26.40	10.99 (0.99)	9.90	10.36 (1.21)	13.22
T_7	BABA-SL	9.09 (2.41)	36.14	10.32 (2.60)	33.68	11.00 (1.00)	10.04	10.12 (0.97)	10.61
T_8	BABA-SPY	9.16 (2.48)	37.11	10.14 (2.42)	31.28	10.98 (0.98)	9.80	10.42 (1.27)	13.92
T_9	BABA-S+SL	9.64 (2.96)	44.26	10.98 (3.26)	42.27	11.44 (1.44)	14.36	10.94 (1.79)	19.52
T_{10}	BABA-S+SL + SPY	10.37 (3.69)	55.30	11.93 (4.21)	54.47	12.31 (2.31)	23.12	11.89 (2.74)	29.97
T_{11}	CHT-S	7.98 (1.30)	19.45	10.45 (2.73)	35.31	10.98 (0.98)	9.82	10.30 (1.15)	12.59
T_{12}	CHT-SL	8.05 (1.37)	20.56	10.59 ((2.87)	37.20	11.01 (1.01)	10.08	10.48 (1.33)	14.51
T_{13}	CHT-SPY	8.45 (1.77)	26.45	10.60 (2.88)	37.36	11.00 (1.00)	10.02	10.40 (1.25)	13.68
T_{14}	CHT-S+SL	9.21 (2.53)	37.93	11.02 (3.30)	42.76	11.84 (1.84)	18.40	11.36 (2.21)	24.15
T_{15}	CHT-S+SL + SPY	10.47 (3.79)	56.67	11.82 (4.10)	53.16	12.36 (2.36)	23.60	11.96 (2.81)	30.71
T_{16}	Control	6.68 (0.00)	0.00	7.72 (0.00)	0.00	10.00 (0.00)	0.00	9.15 (0.00)	0.00
	S.E. ±	0.242		0.219		0.196		0.173	
	C.D. at 5 %	0.702		0.635		0.568		0.501	

Figures in parenthesis indicate increase in phenyl ammonia lyase activity (mg) over control.

Table 42. Phenyl alanine ammonia lyase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Phenyl alanine ammonia lyase activity (µ moles of cinnamic acid produced min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in PAL activity over control	60 DAT	Per cent increase in PAL activity over control	90 DAT	Per cent increase in PAL activity over control	105 DAT	Per cent increase in PAL activity over control
T_1	SA-S	7.01 (1.91)	37.45	9.41 (2.89)	44.31	9.75 (1.78)	22.28	9.48 (2.45)	34.85
T_2	SA-SL	6.86 (1.76)	34.43	9.62 (3.10)	47.52	10.11 (2.14)	26.83	9.70 (2.67)	37.98
T_3	SA-SPY	7.97 (2.87)	56.27	10.52 (4.00)	61.30	11.55 (3.58)	44.92	11.03 (4.00)	56.90
T_4	SA-S+SL	6.54 (1.44)	28.16	9.72 (3.20)	49.00	10.13 (2.16)	27.14	9.78 (2.75)	39.12
T_5	SA-S+SL+SPY	8.02 (2.92)	57.25	10.75 (4.23)	64.88	11.83 (3.86)	48.39	11.35 (4.32)	61.41
T_6	BABA-S	6.99 (1.89)	37.00	8.35 (1.83)	27.99	8.98 (1.01)	12.65	8.38 (1.35)	19.17
T_7	BABA-SL	6.60 (1.50)	29.37	8.24 (1.72)	26.35	8.99 (1.02)	12.82	8.41 (1.38)	19.66
T_8	BABA-SPY	6.93 (1.83)	35.84	8.51 (1.99)	30.52	10.00 (2.03)	25.50	9.42 (2.39)	34.03
T_9	BABA-S+SL	6.50 (1.40)	27.53	9.01 (2.49)	38.14	9.22 (1.25)	15.68	9.19 (2.16)	30.73
T_{10}	BABA-S+SL + SPY	7.31 (2.21)	43.37	9.93 (3.41)	52.35	10.32 (2.35)	29.49	9.98 (2.95)	41.96
T_{11}	CHT-S	5.31 (0.21)	4.12	8.78 (2.26)	34.66	9.00 (1.03)	12.90	8.80 (1.77)	25.18
T_{12}	CHT-SL	5.27 (0.17)	3.24	8.40 (1.88)	28.83	9.00 (1.03)	12.97	8.52 (1.49)	21.19
T_{13}	CHT-SPY	6.14 (1.04)	20.39	8.78 (2.26)	34.65	9.01 (1.04)	13.10	8.82 (1.79)	25.46
T_{14}	CHT-S+SL	6.76 (1.66)	32.53	9.02 (2.50)	38.34	9.82 (1.85)	23.21	9.24 (2.21)	31.44
T_{15}	CHT-S+SL + SPY	7.57 (2.47)	48.43	10.12 (3.60)	55.21	11.03 (3.06)	38.44	10.51 (3.48)	49.56
T_{16}	Control	5.10 (0.00)	0.00	6.52 (0.00)	0.00	7.97 (0.00)	0.00	7.03 (0.00)	0.00
	S.E. ±	0.260		0.231		0.242		0.202	
	C.D. at 5 %	0.752		0.668		0.702		0.585	

Figures in parenthesis indicate increase in phenyl ammonia lyase activity (mg) over control.

Table 43. Pooled data on phenyl alanine ammonia lyase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Phenyl alanine ammonia lyase activity (µ moles of cinnamic acid produced min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in PAL activity over control	60 DAT	Per cent increase in PAL activity over control	90 DAT	Per cent increase in PAL activity over control	105 DAT	Per cent increase in PAL activity over control
T_1	SA-S	8.16 (2.71)	49.75	10.22 (3.60)	54.39	10.85 (1.86)	20.70	10.49 (2.40)	29.70
T_2	SA-SL	8.22 (2.77)	50.79	9.62 (3.00)	45.26	11.03 (2.04)	22.67	10.58 (2.49)	30.83
T_3	SA-SPY	9.33 (3.88)	71.13	11.32 (4.70)	70.93	12.04 (3.05)	33.97	11.60 (3.51)	43.44
T_4	SA-S+SL	8.23 (2.78)	50.99	10.57 (3.95)	59.61	11.22 (2.23)	24.82	10.82 (2.73)	33.75
T_5	SA-S+SL+SPY	9.54 (4.09)	75.04	11.83 (5.21)	78.63	12.43 (3.44)	38.21	12.09 (4.00)	49.39
T_6	BABA-S	7.55 (2.10)	38.60	9.05 (2.43)	36.73	9.98 (0.99)	11.06	9.37 (1.28)	15.81
T_7	BABA-SL	7.85 (2.40)	43.97	9.28 (2.66)	40.17	10.00 (1.01)	11.21	9.27 (1.18)	14.54
T_8	BABA-SPY	8.04 (2.59)	47.59	9.32 (2.70)	40.82	10.49 (1.50)	16.70	9.92 (1.83)	22.66
T_9	BABA-S+SL	8.07 (2.62)	48.08	10.00 (3.38)	50.98	10.33 (1.34)	14.88	10.06 (1.97)	24.39
T_{10}	BABA-S+SL + SPY	8.84 (3.39)	62.26	10.93 (4.31)	65.09	11.32 (2.33)	25.87	10.94 (2.85)	35.18
T_{11}	CHT-S	6.64 (1.19)	21.92	9.61 (2.99)	45.21	9.99 (1.00)	11.12	9.55 (1.46)	18.06
T_{12}	CHT-SL	6.66 (1.21)	22.19	9.50 (2.88)	43.44	10.01 (1.02)	11.30	9.50 (1.41)	17.42
T_{13}	CHT-SPY	7.29 (1.84)	33.83	9.69 (3.07)	46.40	10.01 (1.02)	11.32	9.61 (1.52)	18.80
T_{14}	CHT-S+SL	7.99 (2.54)	46.54	10.02 (3.40)	51.37	10.83 (1.84)	20.47	10.30 (2.21)	27.32
T_{15}	CHT-S+SL + SPY	9.02 (3.57)	65.46	10.97 (4.35)	65.74	11.70 (2.71)	30.11	11.24 (3.15)	38.90
T_{16}	Control	5.45 (0.00)	0.00	6.62 (0.00)	0.00	8.99 (0.00)	0.00	8.09 (0.00)	0.00
	S.E. ±	0.251		0.225		0.219		0.188	
	C.D. at 5 %	0.727		0.652		0.635		0.543	

Figures in parenthesis indicate increase in phenyl ammonia lyase activity (mg) over control.

during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the PAL activity. The change in activity of PAL due to different RIC's treatments showed increasing trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT. The higher level of PAL activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), 12.09 µ moles of cinnamic acid produced/min/mg soluble protein at 105 DAT. There was an increase of 49.39 per cent as compare to control at 105 DAT. The treatment T_3 i.e. SA-SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Untreated control exhibited least amount of PAL activity (8.09 µ moles of cinnamic acid produced/min./mg soluble protein). A slight increase in PAL activity was seen in untreated control plant initially up to 90 DAT there after it was observed decreasing. This result showed that RIC's could induce higher level of PAL activity in infected plants as compare to untreated control.

4.3.6 Peroxidase activity

In the present investigations it was noticed that there was higher peroxidase activity in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015 (Table 44 and 45). All assigned treatments of RIC's significantly increased the peroxidase activity of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_5 (SA-S+SL+SPY) showed maximum increasing peroxidase activity during both season. The change in activity of peroxidase due to different RIC's treatments showed increase in trend from 30 DAT to 90 DAT, there after the activity decreased.

During both the season, the higher amount of peroxidase activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), at 105 DAT, 14.32 and 13.05 units mg^{-1} protein respectively. There was an increase of 64.22 and 76.64 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. S-SPY treatment was proved to be

Table 44. Peroxidase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Peroxidase activity (Units min $^{-1}$mg $^{-1}$ protein)							
		30 DAT	Per cent increase in peroxidase activity over control	60 DAT	Per cent increase in peroxidase activity over control	90 DAT	Per cent increase in peroxidase activity over control	105 DAT	Per cent increase in peroxidase activity over control
T_1	SA-S	4.92 (0.84)	20.58	7.56 (1.36)	21.94	11.08 (1.64)	17.37	9.92 (1.20)	13.76
T_2	SA-SL	5.00 (0.92)	22.55	7.60 (1.40)	22.58	11.52 (2.08)	22.03	10.40 (1.68)	19.27
T_3	SA-SPY	6.16 (2.08)	50.98	9.64 (3.44)	55.48	15.12 (5.68)	60.17	13.59 (4.87))	55.87
T_4	SA-S+SL	5.60 (1.52)	37.25	8.84 (2.64)	42.58	13.48 (4.04)	42.80	11.84 (3.12)	35.78
T_5	SA-S+SL+SPY	6.36 (2.28)	55.88	10.08 (3.88)	62.58	16.60 (7.16)	75.85	14.32 (5.60)	64.22
T_6	BABA-S	4.72 (0.64)	15.69	7.16 (0.96)	15.48	10.72 (1.28)	13.56	9.60 (0.88)	10.09
T_7	BABA-SL	4.80 (0.72)	17.65	7.28 (1.08)	17.42	10.92 (1.48)	15.68	9.92 (1.20)	13.76
T_8	BABA-SPY	5.20 (1.12)	27.45	7.92 (1.72)	27.74	12.32 (2.88)	30.51	10.80 (2.08)	23.85
T_9	BABA-S+SL	5.04 (0.96)	23.53	7.60 (1.40)	22.58	11.56 (2.12)	22.46	10.48 (1.76)	20.18
T_{10}	BABA-S+SL + SPY	5.96 (1.88)	46.08	9.04 (2.84)	45.81	13.92 (4.48)	47.46	12.88 (4.16)	47.71
T_{11}	CHT-S	4.63 (0.55)	13.58	7.16 (0.96)	15.48	10.72 (1.28)	13.56	9.60 (0.88)	10.09
T_{12}	CHT-SL	5.00 (0.92)	22.55	7.60 (1.40)	22.58	11.36 (1.92)	20.34	10.32 (1.60)	18.35
T_{13}	CHT-SPY	5.80 (1.72)	42.16	8.92 (2.72)	43.87	13.60 (4.16)	44.07	11.84 (3.12)	35.78
T_{14}	CHT-S+SL	5.60 (1.52)	37.25	8.24 (2.04)	32.90	11.84 (2.40)	25.42	11.56 (2.84)	32.57
T_{15}	CHT-S+SL + SPY	5.96 (1.88)	46.08	9.64 (3.44)	55.48	14.08 (4.64)	49.15	13.00 (4.28)	49.08
T_{16}	Control	4.08 (0.00)	0.00	6.20 (0.00)	0.00	9.44 (0.00)	0.00	8.72 (0.00)	0.00
	S.E. $\pm$	0.115		0.144		0.179		0.101	
	C.D. at 5 %	0.334		0.418		0.518		0.292	

Figures in parenthesis indicate increase in peroxidase activity (mg) over control.

Table 45. Peroxidase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Peroxidase activity (Units min $^{-1}$mg $^{-1}$ protein)							
		30 DAT	Per cent increase in peroxidase activity over control	60 DAT	Per cent increase in peroxidase activity over control	90 DAT	Per cent increase in peroxidase activity over control	105 DAT	Per cent increase in peroxidase activity over control
T_1	SA-S	3.64 (0.82)	29.18	6.38 (0.92)	16.92	9.84 (1.68)	20.59	8.51 (1.12)	15.18
T_2	SA-SL	3.71 (0.89)	31.49	6.43 (0.97)	17.78	10.32 (2.16)	26.47	9.08 (1.69)	22.87
T_3	SA-SPY	4.91 (2.09)	74.18	8.54 (3.08)	56.41	13.85 (5.69)	69.73	12.25 (4.86)	65.70
T_4	SA-S+SL	4.24 (1.42)	50.35	7.68 (2.22)	40.73	12.28 (4.12)	50.49	10.51 (3.12)	42.26
T_5	SA-S+SL+SPY	5.08 (2.26)	80.14	8.94 (3.48)	63.74	15.34 (7.18)	87.99	13.05 (5.66)	76.64
T_6	BABA-S	3.42 (0.60)	21.31	5.94 (0.48)	8.79	9.46 (1.30)	15.93	8.28 (0.89)	12.10
T_7	BABA-SL	3.54 (0.72)	25.53	6.18 (0.72)	13.19	9.84 (1.68)	20.59	8.65 (1.26)	16.98
T_8	BABA-SPY	3.84 (1.02)	36.17	6.84 (1.38)	25.27	11.18 (3.02)	37.01	9.48 (2.09)	28.29
T_9	BABA-S+SL	3.73 (0.91)	32.13	6.45 (0.99)	18.13	10.32 (2.16)	26.47	9.18 (1.79)	24.28
T_{10}	BABA-S+SL + SPY	4.64 (1.82)	64.54	7.84 (2.38)	43.63	12.64 (4.48)	54.90	11.55 (4.16)	56.28
T_{11}	CHT-S	3.39 (0.57)	20.35	5.84 (0.38)	6.96	9.64 (1.48)	18.14	8.25 (0.86)	11.69
T_{12}	CHT-SL	3.78 (0.96)	34.18	6.45 (0.99)	18.13	10.32 (2.16)	26.47	8.86 (1.47)	19.89
T_{13}	CHT-SPY	4.58 (1.76)	62.55	7.68 (2.22)	40.66	12.42 (4.26)	52.21	10.46 (3.07)	41.60
T_{14}	CHT-S+SL	4.32 (1.50)	53.12	7.16 (1.70)	31.14	10.62 (2.46)	30.15	10.24 (2.85)	38.61
T_{15}	CHT-S+SL + SPY	4.72 (1.90)	67.34	8.52 (3.06)	56.04	12.72 (4.56)	55.88	11.67 (4.28)	57.97
T_{16}	Control	2.82 (0.00)	0.00	5.46 (0.00)	0.00	8.16 (0.00)	0.00	7.39 (0.00)	0.00
	S.E. ±	0.09		0.147		0.101		0.115	
	C.D. at 5 %	0.261		0.424		0.292		0.334	

Figures in parenthesis indicate increase in peroxidase activity (mg) over control.

Table 46. Pooled data on peroxidase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Peroxidase activity (Units min $^{-1}$mg $^{-1}$ protein)							
		30 DAT	Per cent increase in peroxidase activity over control	60 DAT	Per cent increase in peroxidase activity over control	90 DAT	Per cent increase in peroxidase activity over control	105 DAT	Per cent increase in peroxidase activity over control
T_1	SA-S	4.28 (0.83)	24.03	6.97 (1.14)	19.59	10.46 (1.66)	18.86	9.22 (1.16)	14.40
T_2	SA-SL	4.35 (0.90)	26.13	7.02 (1.19)	20.33	10.92 (2.12)	24.09	9.74 (1.68)	20.90
T_3	SA-SPY	5.54 (2.08)	60.37	9.09 (3.26)	55.92	14.49 (5.69)	64.60	12.92 (4.86)	60.36
T_4	SA-S+SL	4.92 (1.47)	42.53	8.26 (2.43)	41.72	12.88 (4.08)	46.36	11.18 (3.12)	38.74
T_5	SA-S+SL+SPY	5.72 (2.27)	65.70	9.51 (3.68)	63.12	15.97 (7.17)	81.48	13.69 (5.63)	69.90
T_6	BABA-S	4.07 (0.62)	17.92	6.55 (0.72)	12.35	10.09 (1.29)	14.66	8.94 (0.89)	11.00
T_7	BABA-SL	4.17 (0.72)	20.80	6.73 (0.90)	15.44	10.38 (1.58)	17.95	9.28 (1.23)	15.22
T_8	BABA-SPY	4.52 (1.07)	30.94	7.38 (1.55)	26.59	11.75 (2.95)	33.52	10.14 (2.08)	25.88
T_9	BABA-S+SL	4.38 (0.93)	26.97	7.03 (1.20)	20.50	10.94 (2.14)	24.32	9.83 (1.78)	22.05
T_{10}	BABA-S+SL + SPY	5.30 (1.85)	53.53	8.44 (2.61)	44.79	13.28 (4.48)	50.91	12.21 (4.16)	51.62
T_{11}	CHT-S	4.01 (0.56)	16.28	6.50 (0.67)	11.49	10.18 (1.38)	15.68	8.93 (0.87)	10.81
T_{12}	CHT-SL	4.39 (0.94)	27.23	7.03 (1.20)	20.50	10.84 (2.04)	23.18	9.59 (1.53)	19.04
T_{13}	CHT-SPY	5.19 (1.74)	50.41	8.30 (2.47)	42.37	13.01 (4.21)	47.84	11.15 (3.10)	38.43
T_{14}	CHT-S+SL	4.96 (1.51)	43.66	7.70 (1.87)	32.08	11.23 (2.43)	27.61	10.90 (2.85)	35.32
T_{15}	CHT-S+SL + SPY	5.34 (1.89)	54.68	9.08 (3.25)	55.75	13.40 (4.60)	52.27	12.34 (4.28)	53.14
T_{16}	Control	3.45 (0.00)	0.00	5.83 (0.00)	0.00	8.80 (0.00)	0.00	8.06 (0.00)	0.00
	S.E. $\pm$	0.103		0.145		0.140		0.108	
	C.D. at 5 %	0.297		0.421		0.405		0.313	

Figures in parenthesis indicate increase in peroxidase activity (mg) over control.

the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of peroxidase activity. A slight increase in peroxidase activity was seen in untreated control plant initially up to 90 DAT and there after it was decreased. This result showed that RIC's could induce peroxidase activity at higher level in infected plants as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in peroxidase activity was presented in Table 46 and Fig. 16 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the peroxidase activity. The change in activity of peroxidase due to different RIC's treatments showed increasing trend from 30 DAT to 90 DAT, whereas the activity decreased at 105 DAT. The higher level of peroxidase activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), 13.69 units mg^{-1} protein. There was an increase of 69.90 per cent as compare to control at 105 DAT. The treatment T_3 i.e. SA-SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Untreated control exhibited least amount of peroxidase activity (8.06 units mg^{-1} protein). A slight increase in peroxidase activity was seen in untreated control plant initially up to 90DAT only. This result showed that RIC's could induce higher level of peroxidase activity in infected plants as compare to untreated control.

4.3.7 Polyphenol oxidase activity

In the present investigations it was noticed that there was higher polyphenol oxidase content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015 (Table 47 and 48). All assigned treatments of RIC's significantly increased the polyphenol oxidase content of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_5 (SA-S+SL+SPY) showed maximum increase in polyphenol oxidase activity during both season. The change

in activity of polyphenol oxidase due to different RIC's treatments showed increasing trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

During both the season, the higher amount of polyphenol oxidase activity was observed in plants treated with treatment T5 i.e. (SA-S+SL+SPY), at 105 DAT, 16.99 and 16.87 units mg^{-1} protein respectively. There was an increase of 55.87 and 56.97 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. SA-SPY treatment was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of polyphenol oxidase activity. A slight increase in polyphenol oxidase activity was seen in untreated control plant initially up to 90 DAT thereafter it was decreasing at 105 DAT. This result showed that RIC's could induce chitinase activity at higher level in infected plant as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in polyphenol oxidase activity was presented in Table 49 and Fig. 17 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increase the polyphenol oxidase activity. The change in activity of polyphenol oxidase due to different RIC's treatments showed increasing trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT. The higher level of polyphenol oxidase activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), 16.93 units mg^{-1} protein at 105 DAT. There was an increase of 56.34 per cent as compare to control at 105 DAT. The treatment T_3 i.e. S- SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Untreated control exhibited least amount of polyphenol oxidase activity (10.83 units mg^{-1} protein). A slight increase in polyphenol oxidase activity was seen in untreated control plant initially up to 90 DAT. This result showed that RIC's could induce higher level of polyphenol oxidase activity in infected plant as compare to untreated control.

Table 47. Polyphenol oxidase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Polyphenol oxidase activity (Units min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in polyphenol oxidase activity over control	60 DAT	Per cent increase in polyphenol oxidase activity over control	90 DAT	Per cent increase in polyphenol oxidase activity over control	105 DAT	Per cent increase in polyphenol oxidase activity over control
T_1	SA-S	6.15 (1.05)	20.58	9.45 (1.70)	21.94	13.85 (2.05)	17.37	11.26 (0.36)	3.31
T_2	SA-SL	6.25 (1.15)	22.55	9.50 (1.75)	22.58	14.40 (2.60)	22.03	12.21 (1.31)	11.99
T_3	SA-SPY	7.54 (2.44)	47.84	11.82 (4.07)	52.52	18.72 (6.92)	58.64	16.71 (5.81)	53.34
T_4	SA-S+SL	7.00 (1.90)	37.25	10.45 (2.70)	34.84	16.54 (4.74)	40.17	14.55 (3.65)	33.51
T_5	SA-S+SL+SPY	7.60 (2.50)	49.02	12.36 (4.61)	59.48	19.42 (7.62)	64.58	16.99 (6.09)	55.87
T_6	BABA-S	5.90 (0.80)	15.69	8.95 (1.20)	15.48	13.40 (1.60)	13.56	11.13 (0.23)	2.13
T_7	BABA-SL	6.00 (0.90)	17.65	9.10 (1.35)	17.42	13.65 (1.85)	15.68	11.38 (0.48)	4.44
T_8	BABA-SPY	6.50 (1.40)	27.45	9.90 (2.15)	27.74	15.00 (3.20)	27.12	12.75 (1.85)	17.00
T_9	BABA-S+SL	6.30 (1.20)	23.53	9.50 (1.75)	22.58	14.45 (2.65)	22.46	12.30 (1.40)	12.83
T_{10}	BABA-S+SL + SPY	7.25 (2.15)	42.16	11.05 (3.30)	42.58	17.20 (5.40)	45.76	15.60 (4.70)	43.12
T_{11}	CHT-S	5.79 (0.69)	13.58	8.80 (1.05)	13.55	13.40 (1.60)	13.56	11.34 (0.44)	4.07
T_{12}	CHT-SL	6.25 (1.15)	22.55	9.50 (1.75)	22.58	14.20 (2.40)	20.34	11.84 (0.94)	8.60
T_{13}	CHT-SPY	6.85 (1.75)	34.31	10.82 (3.07)	39.61	17.04 (5.24)	44.41	14.80 (3.90)	35.78
T_{14}	CHT-S+SL	6.64 (1.54)	30.20	9.75 (2.00)	25.81	14.80 (3.00)	25.42	12.59 (1.69)	15.54
T_{15}	CHT-S+SL + SPY	7.30 (2.20)	43.14	11.46 (3.71)	47.87	17.71 (5.91)	50.08	16.61 (5.71)	52.40
T_{16}	Control	5.10 (0.00)	0.00	7.75 (0.00)	0.00	11.80 (0.00)	0.00	10.90 (0.00)	0.00
	S.E. ±	0.017		0.012		0.023		0.029	
	C.D. at 5 %	0.050		0.034		0.067		0.084	

Figures in parenthesis indicate increase in polyphenol oxidase activity (mg) over control.

Table 48. Polyphenol oxidase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Polyphenol oxidase activity (Units min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in polyphenol oxidase activity over control	60 DAT	Per cent increase in polyphenol oxidase activity over control	90 DAT	Per cent increase in polyphenol oxidase activity over control	105 DAT	Per cent increase in polyphenol oxidase activity over control
T_1	SA-S	6.36 (1.11)	21.15	9.63 (1.98)	25.82	13.71 (1.69)	14.04	11.12 (0.37)	3.44
T_2	SA-SL	6.25 (1.00)	19.05	9.45 (1.80)	23.47	14.54 (2.52)	20.99	12.06 (1.31)	12.19
T_3	SA-SPY	7.68 (2.43)	46.29	11.25 (3.60)	47.06	18.96 (6.94)	57.74	16.70 (5.95)	55.33
T_4	SA-S+SL	7.08 (1.83)	34.91	10.42 (2.77)	36.21	16.62 (4.60)	38.25	14.47 (3.72)	34.57
T_5	SA-S+SL+SPY	7.82 (2.57)	48.95	11.78 (4.13)	53.99	19.44 (7.42)	61.76	16.87 (6.12)	56.97
T_6	BABA-S	6.15 (0.90)	17.20	8.90 (1.25)	16.28	13.25 (1.23)	10.24	11.13 (0.38)	3.56
T_7	BABA-SL	6.16 (0.91)	17.42	9.21 (1.56)	20.37	13.72 (1.70)	14.17	11.53 (0.78)	7.22
T_8	BABA-SPY	6.67 (1.42)	27.03	9.79 (2.14)	27.97	15.15 (3.13)	26.07	12.98 (2.23)	20.78
T_9	BABA-S+SL	6.49 (1.24)	23.68	9.40 (1.75)	22.85	14.80 (2.78)	23.15	12.23 (1.48)	13.74
T_{10}	BABA-S+SL + SPY	7.33 (2.08)	39.59	10.82 (3.17)	41.44	17.01 (4.99)	41.49	15.43 (4.68)	43.52
T_{11}	CHT-S	5.87 (0.62)	11.88	8.64 (0.99)	12.94	13.47 (1.45)	12.09	11.20 (0.45)	4.20
T_{12}	CHT-SL	6.21 (0.96)	18.25	9.45 (1.80)	23.47	14.35 (2.33)	19.38	11.77 (1.02)	9.45
T_{13}	CHT-SPY	7.15 (1.90)	36.28	10.54 (2.89)	37.78	16.83 (4.81)	40.02	14.63 (3.88)	36.05
T_{14}	CHT-S+SL	6.82 (1.57)	29.91	9.64 (1.99)	26.01	14.58 (2.56)	21.32	12.30 (1.55)	14.46
T_{15}	CHT-S+SL + SPY	7.42 (2.17)	41.33	11.08 (3.43)	44.84	18.32 (6.30)	52.41	16.33 (5.58)	51.86
T_{16}	Control	5.25 (0.00)	0.00	7.65 (0.00)	0.00	12.02 (0.00)	0.00	10.75 (0.00)	0.00
	S.E. ±	0.012		0.023		0.017		0.035	
	C.D. at 5 %	0.033		0.067		0.050		0.100	

Figures in parenthesis indicate increase in polyphenol oxidase activity (mg) over control.

Table 49. Pooled data on polyphenol oxidase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	Polyphenol oxidase activity (Units min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in polyphenol oxidase activity over control	60 DAT	Per cent increase in polyphenol oxidase activity over control	90 DAT	Per cent increase in polyphenol oxidase activity over control	105 DAT	Per cent increase in polyphenol oxidase activity over control
T_1	SA-S	6.26 (1.08)	20.75	9.54 (1.84)	23.86	13.78 (1.87)	15.69	11.19 (0.36)	3.33
T_2	SA-SL	6.25 (1.07)	20.66	9.47 (1.77)	23.02	14.47 (2.56)	21.51	12.13 (1.30)	12.04
T_3	SA-SPY	7.61 (2.43)	46.91	11.54 (3.84)	49.81	18.84 (6.93)	58.19	16.71 (5.88)	54.26
T_4	SA-S+SL	7.04 (1.86)	35.93	10.44 (2.74)	35.52	16.58 (4.67)	39.20	14.51 (3.68)	33.98
T_5	SA-S+SL+SPY	7.71 (2.53)	48.84	12.07 (4.37)	56.75	19.43 (7.52)	63.16	16.93 (6.10)	56.34
T_6	BABA-S	6.03 (0.85)	16.34	8.92 (1.22)	15.88	13.33 (1.42)	11.89	11.13 (0.30)	2.79
T_7	BABA-SL	6.08 (0.90)	17.42	9.15 (1.45)	18.89	13.69 (1.78)	14.92	11.45 (0.62)	5.77
T_8	BABA-SPY	6.58 (1.40)	27.11	9.85 (2.15)	27.86	15.08 (3.17)	26.59	12.87 (2.04)	18.82
T_9	BABA-S+SL	6.40 (1.22)	23.49	9.45 (1.75)	22.72	14.63 (2.72)	22.81	12.26 (1.43)	13.23
T_{10}	BABA-S+SL + SPY	7.29 (2.11)	40.72	10.94 (3.24)	42.01	17.10 (5.19)	43.61	15.51 (4.68)	43.25
T_{11}	CHT-S	5.83 (0.65)	12.61	8.72 (1.02)	13.25	13.44 (1.53)	12.82	11.27 (0.44)	4.09
T_{12}	CHT-SL	6.23 (1.05)	20.25	9.47 (1.77)	23.02	14.27 (2.36)	19.86	11.80 (0.97)	8.97
T_{13}	CHT-SPY	7.00 (1.82)	35.18	10.68 (2.98)	38.70	16.94 (5.03)	42.19	14.71 (3.88)	35.85
T_{14}	CHT-S+SL	6.73 (1.55)	29.93	9.69 (1.99)	25.91	14.69 (2.78)	23.35	12.45 (1.62)	14.95
T_{15}	CHT-S+SL + SPY	7.36 (2.18)	42.08	11.27 (3.57)	46.36	18.02 (6.11)	51.26	16.47 (5.64)	52.07
T_{16}	Control	5.18 (0.00)	0.00	7.70 (0.00)	0.00	11.91 (0.00)	0.00	10.83 (0.00)	0.00
	S.E. ±	0.015		0.017		0.02		0.032	
	C.D. at 5 %	0.042		0.05		0.059		0.092	

Figures in parenthesis indicate increase in polyphenol oxidase activity (mg) over control.

4.3.8 β 1, 3 glucanase activity

In the present investigations it was noticed that there was higher β 1, 3 glucanase activity in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015 (Table 50 and 51). All assigned treatments of RIC's significantly increased the β 1, 3 glucanase activity of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_{10} (BABA-S+SL+SPY) showed maximum increase in β 1, 3 glucanase activity during both season. The change in activity of β 1, 3 glucanase due to different RIC's treatments showed increasing trend from 30 to 105 DAT.

During both the season, the higher amount of β 1, 3 glucanase activity was observed in plants treated with treatment T_{10} i.e. (BABA-S+SL+SPY), at 105 DAT, 0.242 and 0.248 mg of glucose released min^{-1} mg^{-1} soluble protein respectively. There was an increase of 29.41 and 25.25 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. SA-S+SL+SPY treatment was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of β 1, 3 glucanase activities. A slight increase in β 1, 3 glucanase activity was seen in untreated control plant up to 105 DAT. This result showed that RIC's could induce β 1, 3 glucanase activity at higher level in infected plant as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in β 1, 3 glucanase activity was presented in Table 52 and Fig. 18 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increase the β 1, 3 glucanase activity. The change in activity of β 1, 3 glucanase due to different RIC's treatments showed increasing trend from 30 to 105 DAT. The higher level of β 1, 3 glucanase activity was observed in plants treated with treatment T_{10} i.e. (BABA-S+SL+SPY), 0.245 mg of glucose released min^{-1} mg^{-1} soluble protein at 105 DAT.

Table 50. β, 1, 3 glucanase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	β, 1, 3 glucanase activity (mg of glucose released min $^{-1}$ mg^{-1} soluble protein)							
		30 DAT	Per cent increase in β,1,3 glucanase activity over control	60 DAT	Per cent increase in β,1,3 glucanase activity over control	90 DAT	Per cent increase in β,1,3 glucanase activity over control	105 DAT	Per cent increase in β,1,3 glucanase activity over control
T_1	SA-S	0.148 (0.016)	12.12	0.164 (0.010)	6.49	0.176 (0.008)	4.76	0.194 (0.007)	3.74
T_2	SA-SL	0.152 (0.020)	15.15	0.171 (0.017)	11.04	0.185 (0.017)	10.12	0.204 (0.017)	9.09
T_3	SA-SPY	0.155 (0.023)	17.42	0.187 (0.033)	21.43	0.192 (0.024)	14.29	0.220 (0.033)	17.65
T_4	SA-S+SL	0.162 (0.030)	22.73	0.175 (0.021)	13.64	0.192 (0.024)	14.29	0.214 (0.027)	14.44
T_5	SA-S+SL+SPY	0.182 (0.050)	37.88	0.195 (0.041)	26.62	0.216 (0.048)	28.57	0.227 (0.040)	21.39
T_6	BABA-S	0.164 (0.032)	24.24	0.172 (0.018)	11.69	0.186 (0.018)	10.71	0.198 (0.011)	5.88
T_7	BABA-SL	0.154 (0.022)	16.67	0.169 (0.015)	9.74	0.178 (0.010)	5.95	0.198 (0.011)	5.88
T_8	BABA-SPY	0.160 (0.028)	21.21	0.172 (0.018)	11.69	0.194 (0.026)	15.48	0.206 (0.019)	10.16
T_9	BABA-S+SL	0.169 (0.037)	28.03	0.184 (0.030)	19.48	0.196 (0.028)	16.67	0.215 (0.028)	14.97
T_{10}	BABA-S+SL + SPY	0.182 (0.050)	37.88	0.198 (0.044)	28.57	0.226 (0.058)	34.52	0.242 (0.055)	29.41
T_{11}	CHT-S	0.142 (0.010)	7.58	0.158 (0.004)	2.60	0.174 (0.006)	3.57	0.190 (0.003)	1.60
T_{12}	CHT-SL	0.148 (0.016)	12.12	0.162 (0.008)	5.19	0.176 (0.008)	4.76	0.192 (0.005)	2.67
T_{13}	CHT-SPY	0.153 (0.021)	15.91	0.171 (0.017)	11.04	0.187 (0.019)	11.31	0.198 (0.011)	5.88
T_{14}	CHT-S+SL	0.158 (0.026)	19.70	0.176 (0.022)	14.29	0.182 (0.014)	8.33	0.196 (0.009)	4.81
T_{15}	CHT-S+SL + SPY	0.173 (0.041)	31.06	0.190 (0.036)	23.38	0.210 (0.042)	25.00	0.224 0.037)	19.79
T_{16}	Control	0.132 (0.000)	0.00	0.154 (0.000)	0.00	0.168 (0.000)	0.00	0.187 (0.000)	0.00
	S.E. ±	0.004		0.006		0.005		0.006	
	C.D. at 5 %	0.012		0.017		0.015		0.018	

Figures in parenthesis indicates increase in β, 1, 3 glucanase activity over control

Table 51. β, 1, 3 glucanase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	β, 1, 3 glucanase activity (mg of glucose released min $^{-1}$ mg^{-1} soluble protein)							
		30 DAT	Per cent increase in β,1,3 glucanase activity over control	60 DAT	Per cent increase in β,1,3 glucanase activity over control	90 DAT	Per cent increase in β,1,3 glucanase activity over control	105 DAT	Per cent increase in β,1,3 glucanase activity over control
T_1	SA-S	0.160 (0.004)	2.56	0.181 (0.003)	1.69	0.188 (0.003)	1.62	0.205 (0.007)	3.54
T_2	SA-SL	0.168 (0.012)	7.69	0.184 (0.006)	3.37	0.190 (0.005)	2.70	0.205 (0.007)	3.54
T_3	SA-SPY	0.172 (0.016)	10.26	0.190 (0.012)	6.74	0.202 (0.017)	9.19	0.218 (0.020)	10.10
T_4	SA-S+SL	0.178 (0.022)	14.10	0.190 (0.012)	6.74	0.205 (0.020)	10.81	0.212 (0.014)	7.07
T_5	SA-S+SL+SPY	0.187 (0.031)	19.87	0.196 (0.018)	10.11	0.221 (0.036)	19.46	0.236 (0.038)	19.19
T_6	BABA-S	0.170 (0.014)	8.97	0.187 (0.009)	5.06	0.194 (0.009)	4.86	0.210 (0.012)	6.06
T_7	BABA-SL	0.162 (0.006)	3.85	0.190 (0.012)	6.74	0.192 (0.007)	3.78	0.206 (0.008)	4.04
T_8	BABA-SPY	0.176 (0.020)	12.82	0.195 (0.017)	9.55	0.210 (0.025)	13.51	0.228 (0.030)	15.15
T_9	BABA-S+SL	0.180 (0.024)	15.38	0.187 (0.009)	5.06	0.215 (0.030)	16.22	0.221 (0.023)	11.62
T_{10}	BABA-S+SL + SPY	0.190 (0.034)	21.79	0.198 (0.020)	11.24	0.228 (0.043)	23.24	0.248 (0.050)	25.25
T_{11}	CHT-S	0.160 (0.004)	2.56	0.180 (0.002)	1.12	0.187 (0.002)	1.08	0.201 (0.003)	1.52
T_{12}	CHT-SL	0.163 (0.007)	4.49	0.182 (0.004)	2.25	0.190 (0.005)	2.70	0.204 (0.006)	3.03
T_{13}	CHT-SPY	0.169 (0.013)	8.33	0.181 (0.003)	1.69	0.204 (0.019)	10.27	0.212 (0.014)	7.07
T_{14}	CHT-S+SL	0.172 (0.016)	10.26	0.183 (0.005)	2.81	0.210 (0.025)	13.51	0.220 (0.022)	11.11
T_{15}	CHT-S+SL + SPY	0.184 (0.028)	17.95	0.195 (0.017)	9.55	0.216 (0.031)	16.76	0.230 (0.032)	16.16
T_{16}	Control	0.156 (0.000)	0.00	0.178 (0.000)	0.00	0.185 (0.000)	0.00	0.198 (0.000)	0.00
	S.E. ±	0.004	--	0.001	--	0.006	--	0.005	--
	C.D. at 5 %	0.012	--	0.003	--	0.017	--	0.015	--

Figures in parenthesis indicates increase in β, 1, 3 glucanase activity over control

Table 52. Pooled data on β, 1,3 glucanase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014 and 2015

Tr. No.	Treatment	β, 1, 3 glucanase activity (mg of glucose released min $^{-1}$ mg^{-1} soluble protein)							
		30 DAT	Per cent increase in β,1,3 glucanase activity over control	60 DAT	Per cent increase in β,1,3 glucanase activity over control	90 DAT	Per cent increase in β,1,3 glucanase activity over control	105 DAT	Per cent increase in β,1,3 glucanase activity over control
T_1	SA-S	0.154 (0.010)	6.94	0.173 (0.006)	3.92	0.182 (0.005)	2.82	0.200 (0.007)	3.37
T_2	SA-SL	0.160 (0.016)	11.11	0.178 (0.012)	6.93	0.188 (0.011)	5.93	0.205 (0.012)	5.96
T_3	SA-SPY	0.164 (0.020)	13.54	0.189 (0.023)	13.55	0.197 (0.020)	11.30	0.219 (0.026)	13.47
T_4	SA-S+SL	0.170 (0.026)	18.06	0.183 (0.017)	9.94	0.199 (0.022)	12.15	0.213 (0.020)	10.36
T_5	SA-S+SL+SPY	0.185 (0.041)	28.13	0.196 (0.030)	17.77	0.219 (0.042)	23.45	0.232 (0.039)	19.95
T_6	BABA-S	0.167 (0.023)	15.97	0.180 (0.014)	8.13	0.190 (0.013)	7.34	0.204 (0.011)	5.70
T_7	BABA-SL	0.158 (0.014)	9.72	0.180 (0.014)	8.13	0.185 (0.008)	4.52	0.202 (0.009)	4.66
T_8	BABA-SPY	0.168 (0.024)	16.67	0.184 (0.018)	10.54	0.202 (0.025)	14.12	0.217 (0.024)	12.44
T_9	BABA-S+SL	0.175 (0.031)	21.18	0.186 (0.020)	11.75	0.206 (0.029)	16.10	0.218 (0.025)	12.95
T_{10}	BABA-S+SL + SPY	0.186 (0.042)	29.17	0.198 (0.032)	19.28	0.227 (0.050)	28.25	0.245 (0.052)	26.94
T_{11}	CHT-S	0.151 (0.007)	4.86	0.169 (0.003)	1.81	0.181 (0.004)	1.98	0.196 (0.003)	1.30
T_{12}	CHT-SL	0.156 (0.012)	7.99	0.172 (0.006)	3.61	0.183 (0.006)	3.39	0.198 (0.005)	2.59
T_{13}	CHT-SPY	0.161 (0.017)	11.81	0.176 (0.010)	6.02	0.196 (0.019)	10.73	0.205 (0.012)	6.22
T_{14}	CHT-S+SL	0.165 (0.021)	14.58	0.180 (0.014)	8.13	0.196 (0.019)	10.73	0.208 (0.015)	7.77
T_{15}	CHT-S+SL + SPY	0.179 (0.035)	23.96	0.193 (0.027)	15.96	0.213 (0.036)	20.34	0.227 (0.034)	17.62
T_{16}	Control	0.144 (0.000)	0.00	0.166 (0.000)	0.00	0.177 (0.000)	0.00	0.193 (0.000)	0.00
	S.E. ±	0.004		0.003		0.005		0.006	
	C.D. at 5 %	0.012		0.009		0.015		0.017	

Figures in parenthesis indicates increase in β, 1, 3 glucanase activity over control

There was an increase of 26.94 per cent as compare to control at 105 DAT. The treatment T_5 i.e. SA-S+SL+SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Untreated control exhibited least amount of β 1, 3 glucanase activity. A slight increase in β 1, 3 glucanase activity was seen in untreated control (0.193 mg of glucose released min^{-1} mg^{-1} soluble protein). This result showed that RIC's could induce higher level of β 1, 3 glucanase activity in infected plant as compare to untreated control.

4.3.9 Chitinase activity

In the present investigations it was noticed that there was higher chitinase content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015 (Table 53 and 54). All assigned treatments of RIC's significantly increased the chitinase content of tomato in all the treatments at 30, 60, 90 and 105 DAT in both studied seasons, as compare to control. Among the treatments used, treatment T_5 (SA-S+SL+SPY) showed maximum increase in chitinase activity during both season. The change in activity of chitinase due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity increased at 30 to 60 DAT but decreased at 90 to105 DAT.

During both the season, the great amount of chitinase activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), at 105 DAT, 0.478 and 0.378 mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein respectively. There was an increase of 64.83 and 68.75 per cent as compare to control during *Kharif* 2014 and *Kharif* 2015. SA-SPY treatment was proved to be the second best treatment during *Kharif* 2014 and *Kharif* 2015. Untreated control exhibited least amount of chitinase activity. A slight increase in chitinase activity was seen in untreated control plant initially up to 60 DAT that up to rest of days was decreasing. This result showed that RIC's could

Table 53. Chitinase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014

Tr. No.	Treatment	Chitinase activity in tomato leaves (mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in Chitinase activity over control	60 DAT	Per cent increase in Chitinase activity over control	90 DAT	Per cent increase in Chitinase activity over control	105 DAT	Per cent increase in Chitinase activity over control
T_1	SA-S	0.386 (0.097)	33.56	0.396 (0.070)	21.47	0.348 (0.040)	12.99	0.316 (0.026)	8.97
T_2	SA-SL	0.380 (0.091)	31.49	0.402 (0.076)	23.31	0.392 (0.084)	27.27	0.384 (0.094)	32.41
T_3	SA-SPY	0.402 (0.113)	39.10	0.455 (0.129)	39.57	0.474 (0.166)	53.90	0.452 (0.162)	55.86
T_4	SA-S+SL	0.372 (0.083)	28.72	0.415 (0.089)	27.30	0.386 (0.078)	25.32	0.378 (0.088)	30.34
T_5	SA-S+SL+SPY	0.416 (0.127)	43.94	0.486 (0.160)	49.08	0.482 (0.174)	56.49	0.478 (0.188)	64.83
T_6	BABA-S	0.342 (0.053)	18.34	0.345 (0.019)	5.83	0.320 (0.012)	3.90	0.305 (0.015)	5.17
T_7	BABA-SL	0.354 (0.065)	22.49	0.372 (0.046)	14.11	0.361 (0.053)	17.21	0.356 (0.066)	22.76
T_8	BABA-SPY	0.362 (0.073)	25.26	0.412 (0.086)	26.38	0.432 (0.124)	40.26	0.424 (0.134)	46.21
T_9	BABA-S+SL	0.360 (0.071)	24.57	0.384 (0.058)	17.79	0.372 (0.064)	20.78	0.367 (0.077)	26.55
T_{10}	BABA-S+SL + SPY	0.386 (0.097)	33.56	0.434 (0.108)	33.13	0.430 (0.122)	39.61	0.422 (0.132)	45.52
T_{11}	CHT-S	0.336 (0.047)	16.26	0.342 (0.016)	4.91	0.324 (0.016)	5.19	0.320 (0.030)	10.34
T_{12}	CHT-SL	0.348 (0.059)	20.42	0.350 (0.024)	7.36	0.328 (0.020)	6.49	0.315 (0.025)	8.62
T_{13}	CHT-SPY	0.364 (0.075)	25.95	0.376 (0.050)	15.34	0.370 (0.062)	20.13	0.358 (0.068)	23.45
T_{14}	CHT-S+SL	0.371 (0.082)	28.37	0.392 (0.066)	20.25	0.376 (0.068)	22.08	0.361 (0.071)	24.48
T_{15}	CHT-S+SL + SPY	0.392 (0.103)	35.64	0.442 (0.116)	35.58	0.454 (0.146)	47.40	0.450 (0.160)	55.17
T_{16}	Control	0.289 (0.000)	0.00	0.326 (0.000)	0.00	0.308 (0.000)	0.00	0.290 (0.000)	0.00
	S.E. $\pm$	0.006	--	0.007	--	0.008	--	0.009	--
	C.D. at 5 %	0.018	--	0.021	--	0.023	--	0.025	--

Figures in parenthesis indicates increase in chitinase activity (mg) over control

Table 54. Chitinase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2015

Tr. No.	Treatment	Chitinase activity in tomato leaves (mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in Chitinase activity over control	60 DAT	Per cent increase in Chitinase activity over control	90 DAT	Per cent increase in Chitinase activity over control	105 DAT	Per cent increase in Chitinase activity over control
T_1	SA-S	0.281 (0.067)	31.31	0.292 (0.036)	14.06	0.259 (0.021)	8.82	0.242 (0.018)	8.04
T_2	SA-SL	0.276 (0.062)	28.97	0.309 (0.053)	20.70	0.298 (0.060)	25.21	0.252 (0.028)	12.50
T_3	SA-SPY	0.308 (0.094)	43.93	0.368 (0.112)	43.75	0.370 (0.132)	55.46	0.364 (0.140)	62.50
T_4	SA-S+SL	0.292 (0.078)	36.45	0.316 (0.060)	23.44	0.298 (0.060)	25.21	0.284 (0.060)	26.79
T_5	SA-S+SL+SPY	0.319 (0.105)	49.07	0.390 (0.134)	52.34	0.382 (0.144)	60.50	0.378 (0.154)	68.75
T_6	BABA-S	0.276 (0.062)	28.97	0.284 (0.028)	10.94	0.264 (0.026)	10.92	0.229 (0.005)	2.23
T_7	BABA-SL	0.260 (0.046)	21.50	0.295 (0.039)	15.23	0.274 (0.036)	15.13	0.254 (0.030)	13.39
T_8	BABA-SPY	0.298 (0.084)	39.25	0.335 (0.079)	30.86	0.324 (0.086)	36.13	0.312 (0.088)	39.29
T_9	BABA-S+SL	0.296 (0.082)	38.32	0.332 (0.076)	29.69	0.325 (0.087)	36.55	0.317 (0.093)	41.52
T_{10}	BABA-S+SL + SPY	0.289 (0.075)	35.05	0.312 (0.056)	21.88	0.345 (0.107)	44.96	0.336 (0.112)	50.00
T_{11}	CHT-S	0.248 (0.034)	15.89	0.271 (0.015)	5.86	0.258 (0.020)	8.40	0.252 (0.028)	12.50
T_{12}	CHT-SL	0.259 (0.045)	21.03	0.286 (0.030)	11.72	0.272 (0.034)	14.29	0.258 (0.034)	15.18
T_{13}	CHT-SPY	0.287 (0.073)	34.11	0.302 (0046)	17.97	0.296 (0.058)	24.37	0.290 (0.066)	29.46
T_{14}	CHT-S+SL	0.294 (0.080)	37.38	0.322 (0.066)	25.78	0.319 (0.081)	34.03	0.282 (0.058)	25.89
T_{15}	CHT-S+SL + SPY	0.306 (0.092)	42.99	0.352 (0.096)	37.50	0.364 (0.126)	52.94	0.354 (0.130)	58.04
T_{16}	Control	0.214 (0.000)	0.00	0.256 (0.000)	0.00	0.238 (0.000)	0.00	0.224 (0.000)	0.00
	S.E. ±	0.006	--	0.009	--	0.008	--	0.007	--
	C.D. at 5 %	0.017	--	0.025	--	0.022	--	0.020	--

Figures in parenthesis indicates increase in chitinase activity (mg) over control

Table 55. Pooled data on chitinase activity in tomato leaves as influenced by different resistance inducing chemicals during *Kharif* 2014- *Kharif* 2015

Tr. No.	Treatment	Chitinase activity in tomato leaves (mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein)							
		30 DAT	Per cent increase in Chitinase activity over control	60 DAT	Per cent increase in Chitinase activity over control	90 DAT	Per cent increase in Chitinase activity over control	105 DAT	Per cent increase in Chitinase activity over control
T_1	SA-S	0.334 (0.082)	32.34	0.344 (0.053)	18.21	0.304 (0.031)	11.17	0.279 (0.022)	8.56
T_2	SA-SL	0.328 (0.076)	30.16	0.356 (0.065)	22.16	0.345 (0.072)	26.37	0.318 (0.061)	23.74
T_3	SA-SPY	0.355 (0.103)	40.87	0.412 (0.121)	41.41	0.422 (0.149)	54.58	0.413 (0.156)	60.70
T_4	SA-S+SL	0.332 (0.080)	31.75	0.366 (0.075)	25.60	0.342 (0.069)	25.27	0.331 (0.074)	28.79
T_5	SA-S+SL+SPY	0.368 (0.116)	45.83	0.438 (0.147)	50.52	0.432 (0.159)	58.24	0.428 (0.171)	66.54
T_6	BABA-S	0.309 (0.057)	22.62	0.315 (0.024)	8.08	0.292 (0.019)	6.96	0.267 (0.010)	3.89
T_7	BABA-SL	0.307 (0.055)	21.83	0.334 (0.043)	14.60	0.318 (0.045)	16.30	0.305 (0.048)	18.68
T_8	BABA-SPY	0.330 (0.078)	30.95	0.374 (0.083)	28.35	0.378 (0.105)	38.46	0.368 (0.111)	43.19
T_9	BABA-S+SL	0.328 (0.076)	30.16	0.358 (0.067)	23.02	0.349 (0.076)	27.66	0.342 (0.085)	33.07
T_{10}	BABA-S+SL + SPY	0.338 (0.086)	33.93	0.373 (0.082)	28.18	0.388 (0.115)	41.94	0.379 (0.122)	47.47
T_{11}	CHT-S	0.292 (0.040)	15.87	0.307 (0.016)	5.33	0.291 (0.018)	6.59	0.286 (0.029)	11.28
T_{12}	CHT-SL	0.304 (0.052)	20.44	0.318 (0.027)	9.28	0.300 (0.027)	9.89	0.287 (0.030)	11.48
T_{13}	CHT-SPY	0.326 (0.074)	29.17	0.339 (0.048)	16.49	0.333 (0.060)	21.98	0.324 (0.067)	26.07
T_{14}	CHT-S+SL	0.333 (0.081)	31.94	0.357 (0.066)	22.68	0.348 (0.075)	27.29	0.322 (0.065)	25.10
T_{15}	CHT-S+SL + SPY	0.349 (0.097)	38.49	0.397 (0.106)	36.43	0.409 (0.136)	49.82	0.402 (0.145)	56.42
T_{16}	Control	0.252 (0.000)	0.00	0.291 (0.000)	0.00	0.273 (0.000)	0.00	0.257 (0.000)	0.00
	S.E. $\pm$	0.006	--	0.007	--	0.008	--	0.009	--
	C.D. at 5 %	0.018	--	0.021	--	0.023	--	0.025	--

Figures in parenthesis indicates increase in chitinase activity (mg) over control

induce chitinase activity at higher level in infected plant as compare to untreated control.

The two years pooled analysis of data (*Kharif* 2014 and *Kharif* 2015) on changes in chitinase activity was presented in Table 55 and Fig. 19 revealed that the data followed the same pattern as recorded during *Kharif* 2014 and 2015 crop season. All the treatments significantly increased the chitinase activity. The change in activity of chitinase due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity increased at 30 to 60 DAT but decreased at 90 to105 DAT. The higher level of chitinase activity was observed in plants treated with treatment T_5 i.e. (SA-S+SL+SPY), 0.428 mg of N-acetyl glucosamine released min^{-1} mg $^{-1}$ soluble protein at 105 DAT. There was an increase of 66.54 per cent as compare to control at 105 DAT. The treatment T_3 i.e. SA-SPY and treatment T_{15} i.e. CHT-S+SL+SPY was proved to be the second and third best treatments. Both treatments were found at par with treatment T_5 (SA-S+SL+SPY). Untreated control exhibited least amount of chitinase activity (0.257 mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein). A slight increase in chitinase activity was seen in untreated control plant initially up to 60DAT and thereafter it was decreasing. This result showed that RIC's could induce higher level of chitinase activity in infected plant as compare to untreated control.

5. DISCUSSION

Chemical resistance inducers are largely used as bioactive substances in controlling soil borne as well as foliar plant pathogens. In the present study, different treatment of RIC's *viz.* salicylic acid, BABA and chitosan were used as seed treatment, seedling treatment, spray treatment, seed + seedling treatment, seed + seedling + spray treatments in order to evaluate their efficacy in controlling tomato diseases under open field conditions. Thulke and Conrath (1998) showed that salicylic acid applied either as a seed treatment, spray or soil drench induces systemic resistance in cacao against *Phytophthora palmivora.* Raut and Borkar (2014) employed the seed, seedling and spray treatment to evaluate the effect of salicylic acid, BABA and chitosan against early blight of tomato in green house condition. The results obtained during the investigation on "Field efficacy of resistance inducing chemicals against tomato diseases" are discussed hereunder in context to the references available on following aspects.

5.1 Studies on effect of RIC's on diseases of tomato

The results obtained from the effect of different RIC's on diseases of tomato *var. Dhanashree* have been discussed under the following headings.

5.1.1 Effect of RIC's on Early blight disease incidence and severity of Tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

Early blight disease caused by *Alternaria solani* is considered as the most serious disease attacking tomato plants (Egashira *et al.*, 2000). The data clearly showed that all the resistance inducing chemicals *viz.* salicylic acid, β-amino butyric acid and chitosan enhanced the resistance to early blight disease incidence and severity of tomato when the seeds and seedlings were subjected to different treatments of the resistance inducing chemicals. It is evident from the data that in all

treatments percent disease incidence and severity were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence and severity increase was comparatively low in case of RIC's treated plots over control. The incidence and severity of early blight of tomato on *var. Dhanashree* was differing significantly due to different treatments of RIC's. The lowest records of these two parameters were noted at the seed + seedling + spray treatments of salicylic acid throughout the crop growth period. The most effective treatments were seed + seedling + spray treatment of salicylic acid followed by spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. They reduced both incidence and severity of early blight. These results are in agreement with findings of Jabnoun-Khiareddine *et al.*, (2015), Agamy *et al.*, (2013), Atia and Ahmed (2011), Liao *et al.*, (2003) and Spletzer and Enyedi (1999).

The inhibition effect of tested inducers (salicylic acid and chitosan) on the growth of the causal organism of tomato early blight disease and disease assessment were agreement with the results obtained by Bhaskara *et al.*, (2000) and Atia *et al.*, (2003 and 2005), Awadalla (2008).

Salicylic acid played an important role in plant defense. It is an important endogenous molecule involved in plant defense. Its role in plant disease resistance is well documented for dicotyledonous plants, where it is required for basal resistance against pathogens as well as for the inducible defense mechanism of SAR, which confer resistance against a broad spectrum of pathogens. The link between salicylic acid production and SAR has been well established (Klessig and Malamy 1994). Awadalla (2008) reported marked reduction in the disease index score of early blight of tomato due to soaking of tomato seed in salicylic acid also showed that the post penetration resistance of the host appeared to be increase under the antioxidant treatments at higher concentration. Hammerschmidt (1999) reported that exogeneous

application of salicylic acid or the structural analogue such as benzo (1,2,3) thiadiazole-carbithioic-acid-S-methylester (BTH) and 2,6-dichloroisonicotinic acid induced the systemic acquire resistance. Spletzer and Enyedi (1999) investigated the effect of exogenous application of salicylic acid (SA) against *Alternaria solani* in tomato leaves and found that addition of 200 µM SA to the root system significantly increased the endogenous SA content of leaves. Challenge inoculation of SA-treated tomato plants using conidia of *A. solani* resulted in 83% fewer lesions per leaf and 77 per cent reduction in blighted leaf area as compared with control. Tosun *et al.* (2006) applied the plant disease resistance elicitors i.e. salicylic acid (SA) and $Harpin_{Ea}$ alone and in combination with systemic fungicide phosphorus acid (Agriphos 400) to tomato plants, which were post inoculated with *Phytophthora infestans*. Their results indicated the average disease reduction by 47 per cent after treatment with SA and by 55 per cent with $Harpin_{Ea}$.

The early blight disease resistance inducing effect of chitosan observed in the present study are consistent with the result of many workers who reported that application of chitosan could enhance the disease resistance of many crops. Algam and Elwagia (2015) found that chitosan application reduced disease severity in whatever concentration it was applied as foliar spray alone or in combination with seed treatment or seed treatment alone. Satiyabama *et al* (2014) indicated that chitosan could protect tomato leaves against the early blight disease caused by *Alternaria solani* under glass house condition. The reduction in disease incidence and severity may be due to the mode of action of chitosan. It stimulates natural defence response systems in treated plants. Poly–D-glucosamine binds to fungal receptor sites, mimicking an attack by fungal spores. This in turn results in signals being sent to the nuclei of the plant and triggering signals which elicit multiple genetic and biological responses including the production of phytoalexins (Anti – microbial compounds produced in plants, aimed at inhibiting infections

(Peniston and Johnson, 1980). Liao *et al* (2003) found that resistance in tomato plants to early blight caused by *Alternaria solani* was induced by chitosan (1 mg/ml) at the four leaf stage of seedlings and activate its defense mechanism. El–Mougy *et al* (2012) evaluated the efficacy of different plant resistance inducers treatments against early and late blight foliar diseases incidence as spray treatments on growing tomato plants under commercial plastic houses and resulted in the highest reduction in foliar diseases incidence and severity and increased the yield. Applications of chitosan extract significantly reduced the incidences of *Alternaria solani* and *Xanthomonas vesicatoria* in green house and field trials, therefore chitosan would able to provide broad range protection through induced systemic resistance mechanism.

5.1.2 Effect of RIC's on Powdery mildew disease incidence and severity of tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

Tomato powdery mildew is one of the most aggressive and destructive diseases that affect tomato plants all over the world. Protection of plants from plant pathogens by induction of systemic resistance is a new approach and is of much less harmful to the environment and plant products as compared to deadly agrochemicals applied to control plant diseases.

The obtained results clearly showed that all the resistance inducing chemicals *viz.* salicylic acid, β-amino butyric acid and chitosan enhanced the resistance to powdery mildew disease incidence and severity of tomato when the seeds and seedlings were subjected to different treatments of the resistance inducing chemicals. It is evident from the data that in all treatments percent disease incidence and severity were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence and severity increase was comparatively low in case of RIC's treated plots over control. The incidence and severity of powdery mildew of tomato on *var. Dhanashree* was differing significantly

due to different treatments of RIC's. The lowest records of these two parameters were noted at the seed + seedling + spray treatments of BABA throughout the crop growth period. They reduced both incidence and severity of powdery mildew. The most effective treatments were seed + seedling + spray treatment of BABA followed by seed + seedling + spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. These results are in agreement with previous findings of Papavizas and Davey (1963), Papavizas (1964), Cohen (1994), Cohen *et al.* (1994), Zimmerli *et al.* (2000), Jakab *et al.* (2001), Cohen 2002, Si-Ammour *et al.* (2003), Ton *et al.* (2005), Arici and Dehne (2007), Polyakovskii *et al.* (2008), Eschen-Lippold *et al.*, 2010, Liljeroth *et al.* (2010), Korosi (2011), Justyna and Ewa, 2013, Bengtsson *et al.*, 2014, Balmer *et al.*, 2015, Li *et al.* (2016) and Luna *et al.* (2016).

β-amino butyric acid can prime resistance in many plant species against a range of stresses, including both necrotrophic and biotrophic pathogens (Zimmerli *et al.*, 2000, Ton and Mauch-Mani, 2004, Ton *et al.*, 2005). When applied in tomato, we found that the treatment seed + seedling + spray improved resistance against powdery mildew disease, presumably via effects on SA – dependent resistance, as seen in other biotrophic and hemibiotrophic pathosystems (Zimmerli *et al.*, 2000, Ton *et al.*, 2005, Worrall *et al.*, 2012).

Worrall *et al* (2011) stated that BABA seed treatment provided primed defence against powdery mildew disease caused by biotrophic fungal pathogen, *Oidium neolycopersici* and long term defence priming by seed treatments was not accompanied by reductions in growth. Abada and Ahmed (2014) found that spraying tomato plants with Salicylic acid alone or in different combination with bioagents *B. thuringiensis* and *T. harzianum* resulted in significant reduction to powdery mildew severity during plastic house experiments.

5.1.3 Effect of RIC's on Tomato Leaf curl virus disease incidence and severity on tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

Tomato leaf curl virus is one of the most hazardous pathogen in tomato plants, and it limits the production of tomatoes in many tropical and subtropical areas of the world. Insecticides are commonly applied to suppress whitefly population and to indirectly reduce the spread of tomato leaf curl virus disease. In this study we investigated the effectiveness of the RIC's against tomato leaf curl virus in tomato plants.

The obtained results clearly showed that all the resistance inducing chemicals *viz.* salicylic acid, β-amino butyric acid and chitosan enhanced the resistance to tomato leaf curl virus disease incidence and severity when the seeds and seedlings were subjected to different treatments of the resistance inducing chemicals. It is evident from the data that in all treatments percent disease incidence and severity were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence and severity increase was comparatively low in case of RIC's treated plots over control. The incidence and severity of tomato leaf curl virus disease on *var. Dhanashree* was differing significantly due to different treatments of RIC's. The lowest records of these two parameters were noted at the seed + seedling + spray treatments of SA throughout the crop growth period. They reduced both incidence and severity of tomato leaf curl virus disease. The most effective treatments were seed + seedling + spray treatment of salicylic acid followed by spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. These results are in agreement with previous findings of Madhusudhan *et al.*, 2011, Ong and Cruz, 2016.

Several studies have demonstrated the efficacy of exogenous application of salicylic acid or its functional analogue (BTH) for controlling fungal and bacterial diseases (Siegrist *et al.*, 1997; Cole, 1999) the effect of which is in the form of induced resistance. However

induced resistance to viruses through exogenous application of salicylic acid or its functional analogue has been demonstrated in only few studies. The resistance of tomato to subsequent infection of tomato leaf curl virus is found to be enhanced by treating plants with 50 µM salicylic acid regardless of induction time and consistently delayed and reduced the severity of leaf curl disease, amount of disease (AUDPC values), lower symptom severity score and lower disease index than the untreated control (Ong and Cruz, 2016). Application of salicylic acid as seed and spray treatment, 1 hr. before inoculation with tobamovirus, protected plants against virus by reducing the number of local lesions formed by the challenge inoculation of tobamoviruses, (Madhusudhan *et al.*, 2005 and 2011). Likewise, the resistance of tobacco to infection of TMV is found to be enhanced by pre-treatment of plant with Aspirin or salicylic acid (White, 1979). Resistance to PepGMV infection is induced in pepper plant by BTH treatment and is through the activation of salicylic acid pathway (Trejo-Saavedra *et al.*, 2013). Treatment of pepper plants with 150-300 mg L^{-1} BTH reduce the symptom severity and percent of infected plants.

5.1.4 Effect of RIC's on Tomato spotted wilt virus disease incidence on tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The obtained result showed that all the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. It has been found that in all treatments the percent disease incidence were comparatively lower in RIC's treated plots. There were no significant differences for any of the treatments on Tomato spotted wilt virus disease incidence during *Kharif* 2015. Pooled analysis also showed same results as *Kharif* 2015. Hence the differences in the results between the two growing seasons may be due the variation in the environmental conditions. Comparatively highest reduction in percent disease incidence

was observed in seed + seedling + spray treatment of salicylic acid followed by spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. The findings obtained in this study were not in agreement with Madhusudhan *et al.*, 2011, Ong and Cruz, 2016.

According to Walters and Boyle (2005), the differences in the above mentioned results may reflect the differences between species as well as environmental conditions. The environmental conditions play an important role in the apparently contradictory results, which in fact represent different outcomes of a complex interplay of factors.

5.1.5 Effect of RIC's on yield of tomato *var. Dhanashree* during *kharif* 2014 and *kharif* 2015

The effect of different RIC's on yield of tomato *var. Dhanashree* has been discussed below.

The investigation confirms that the application of RIC's significantly influenced the yield of tomato during both season (*kharif* 2014 and *kharif* 2015) under field condition. All RIC's promoted yield of tomato compared to control treatment. Among RIC's tested, the salicylic acid was found most effective in increasing yield of tomato. Among all the treatments, significantly highest yield was recorded with the seed + seedling + spray treatment of salicylic acid followed by spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. However the minor differences in the results between the two growing seasons may be due the variation in the environmental conditions.

The yield promoting effect of the RIC's observed in the present study are consistent with the result of Javaheri *et al.* (2012), El-Mohamedy *et al.* (2014), Kazemi (2014), Farouk *et al.* (2008).

Application of salicylic acid 100mM was most efficient for increase of yield and quality of tomato fruits along with reduction in root rot severity under field condition (El-Mohamedy *et al.,* 2014). The fruit yield in cucumber and tomato, enhanced significantly when the plants were sprayed with lower concentration of salicylic acid (Ayala-Zavala *et*

al., 2004). Kumar *et al.* (1999) who found that foliar application of salicylic acid to soybean enhanced the flowering, pod formation and consequently yield of soybean. According to Shakirova (2007) the positive effect of salicylic acid on growth and yield can be due to its influence on other plant hormones. Salicylic acid altered the auxin, cytokinin and ABA balances in wheat and increased the growth and yield under both normal and saline conditions. Increasing of yield under foliar application of salicylic acid could be ascribed to the well known roles of salicylic acid on photosynthetic parameters and plant water relations. Fariduddin *et al.* (2003) reported that exogenous application of salicylic acid enhanced the net photosynthetic rate, internal CO_2 concentration and water use efficiency in *Brassica juncea*. Abdel-Monaim *et al.* (2012) suggested that application of salicylic acid to treat seedling by soaking increased both quality and quantity of tomato along with controlling tomato root rot and wilt diseases. The increase in growth, yield quantity and quality may attributed to elicitors effect on physiological processes in plant such as ion uptake, cell elongation, cell division, enzymatic activation and protein synthesis (Amin *et al.*, 2007, Gharib and Hegazi, 2010). Sharma *et al.* (1986) stated that Salicylic acid play role in stimulation of physiological processes which reflect on improving vegetative growth that followed by active translocation of the photoassimilates from source to sink in plant due to increasing leaf blade thickness as well as dimensions of vascular bundles which may lead to the increase in yield of cucumber plant. In addition, application of SA inhibits ethylene production leading to increasing fruit number and consequently increasing fruit yield per plant (Leslie and Romani, 1986).

5.2 Studies on anatomical changes in cellular leaf structure due to resistance inducing chemicals

The results showed an important effect of RIC's on alterations in the leaf tissue organization in the seedlings of tomato; the anatomy of the foliar tissue was markedly affected, showing

morphological changes compared to control plants. The increase in epidermal layer thickness played an important role in the pathogenesis of *Alternaria* blight infection. Due to increase in epidermal layer thickness, the germ tube/infection peg was unable to penetrate the epidermal cell and further the palisade mesophyll cells to cause the infection. Therefore the first barrier to the infection of *Alternaria* disease was at the anatomical level itself (Raut, 2012).

The evidences clearly showed that all assigned treatments of RIC's significantly increased epidermis cum cuticle layer thickness in both studied seasons. The increase in thickness of epidermis cum cuticle due to RIC's treatments was in the range of 12.38 to 77.72 per cent and 12.28 to 76.00 per cent for both studied season. The most effective treatment which induced highest increase in epidermis cum cuticle layer thickness was in seed + seedling + spray treatment of salicylic acid which is followed spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan.

Also indicated that all assigned treatments of RIC's significantly increased palisade mesophyll layer thickness in both studied seasons (*kharif* 2014 and 2015). The increase in thickness of palisade mesophyll due to RIC's treatments was in the range of 4.47 to 28.26 per cent and 3.57 to 27.37 per cent for both studied season. The most effective treatment which induced highest increase in palisade mesophyll thickness was seed + seedling + spray treatment of salicylic acid which is followed spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan.

In this connection, Raut (2012) reported the application of salicylic acid and β–aminobutyric acid as seed treatment, seedling treatment and seed+seedling treatment increased the thickness of epidermal layer and palisade mesophyll cells of tomato leaves. Yang *et al* (1992) reported the cuticle as effective barrier to *Rhizoctonia solani*. They demonstrated that *Brassica napus* plant did not show an obvious cuticle

layer at one week stage but it was present at three week stage and therefore the plants were resistant at three week stage rather than at one week stage and thus they confirmed the cuticle plays an important role in the resistance to *R. solani*. Our reslts also confirmed the findings of Farouk *et al*. (2008). They reported that the application of chitosan @ 0.05 per cent and salicylic acid @ 100 mg/lit increased the thickness of leaf blade, palisade and spongy parenchyma as well as midrib region which they presume to confirm resistance against downy mildew pathogen.

Gelelcha and Hanchinal (2003) also reported that rust resistant wheat genotypes had thicker epidermis, more number of epidermal cells and thicker leaf lamina than the corresponding susceptible genotypes and these morphophysiological traits had significant contribution in rust resistance mechanism. Azevedo *et al*. (2011) also reported that thick epidermis of sugarcane cultivars prevents the penetration of fungi. Kaur and Dhillon (1985) also reported that structural characteristic of wheat leaf like thickness of epidermal cell wall, number of sclerenchyma layers and their cell wall thickness was significantly higher in wheat plants which confirmed resistance to leaf pathogens.

Gomma *et al*. (2015) found that the treatment of foliar spray of 75 ppm SA conc. induced prominent increase in stem diameter mainly due to the prominent increases induced in thickness of stem wall and in diameter of hollow pith. The increase in stem wall thickness could be attributed to the induced increase in thickness of epidermis, cortex, fiber strands, phloem and xylem tissues as well as in thickness of parenchymatous area of the pith and in vessel diameter. Likewise, such treatment increased thickness of both midvein and lamina of leaflets due to increase induced in thickness of both palisade and spongy tissues and to increase induced in size of midvein bundle. Nour *et al*. (2012) found that foliar application with 50 ppm salicylic acid on bean plants resulted

in thicker leaflets due mainly to the prominent increase induced in thickness of both midvein and lamina. The thicker lamina could be attributed to the increase in thickness of both palisade and spongy tissues. Likewise, midvein bundle was increased in size as a result of spraying salicylic acid. These results are in harmony with the present findings.

The critical analysis of stomatal characteristics of tomato leaves indicated that irrespective of RIC's treatments the stomatal frequency (No. of stomata/mm^2) was fairly high on lower (abaxial) leaf surface than the upper (adaxial) leaf surface for both studied season. The result also showed that different RIC's treatments have no significant effect on stomatal frequency of adaxial and abaxial surface. Relatively higher reduction in stomatal frequency was observed in seed + seedling + spray treatment of salicylic acid as compared to control. These results are quite agreed with Obsuwan *et al.* (2010). They studied the effect of chitosan on the morphological characteristics of the adaxial leaf side and showed that chitosan treatments had a lower stomata number, but a larger stomata size compared to control. On the abaxial leaf side, the concentration of chitosan did not affect stomata number and size. These results are contradictory with Cárcamo *et al.* (2012) who found that exogenous application of SA increased the number of stomata per unit area of lluteño maize under Nacl stress.

Though different RIC's have no effect on stomatal frequency, the result showed that it has a marked effect on stomatal size. The treatment seed + seedling + spray treatment of β-aminobutyric acid significantly reduces the stomatal length and width of stomata for both studied season which was followed by seed + seedling + spray treatment of salicylic acid and seed + seedling + spray treatment of chitosan. This coincides with Lee *et al.* (1999) who showed that these elicitors reduced the size of the stomatal aperture. Chitosan inhibited light-induced stomatal opening in tomato epidermis.

5.3 Studies on biochemical changes induced in tomato plants due to resistance inducing chemicals

5.3.1 Changes in soluble protein and reducing sugar content

In the present investigations it was noticed that there was higher soluble protein content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the soluble protein content of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of salicylic acid showed maximum increase in soluble protein content during both season followed by salicylic acid spray treatment. The change in content of soluble protein due to different RIC's treatments showed increasing trend from 30 to 105 DAT.

In case of reducing sugar content it was noticed that there was higher reducing sugar content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the reducing sugar content of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of β-aminobutyric acid showed maximum increase in reducing sugar content during both season followed by seed + seedling + spray treatment of salicylic acid. The change in content of reducing sugar due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity decreased from 30 to 60 DAT but increased from 90 to 105 DAT.

Ashry and Mohammad (2012) studied the changes in various physiological defenses like secondary metabolites, proline, total soluble protein in leaves and stems of 18 flax lines either resistant or susceptible to powdery mildew. Results showed that total soluble protein content in flax leaves increased significantly in resistant lines when compared with susceptible parent but decreased significantly in susceptible lines when

compared with resistant parent. Samia and Khallal (2007) treated the *Fusarium oxysporum* infected tomato plants with arbuscular mycorrhizal (AM) fungi and/or sprayed three times with inducer (JA and SA) and showed that total soluble sugars, free amino acids and total soluble proteins increased in both leaves and roots of AM and/or JA and SA-treated plants as compared with infected control.

5.3.2 Changes in phenol content

The evidences noticed that there was higher total phenol content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the total phenol content of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of salicylic acid showed maximum increase in total phenol content during both season followed by spray treatment of salicylic acid. The change in total phenol content due to different RIC's treatments showed increase in trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

Barilli *et al.* (2009) reported the production of phenolic compounds such as scopoletin and pisatin by use of resistance inducing chemical benzothiadiazole, was inhibitory to the fungal pathogen of pea rust at early stage. These compounds showed a similar inhibitory effect when exogenously applied *in vitro* bioassay. Ashry and Mohammad (2012) reported the higher content of total phenol in resistant flax line than susceptible one and this higher level of phenol was inhibitory to the pathogen.

5.3.3 Changes in lignin content

In the present investigations it was noticed that there was higher lignin content in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the lignin content of tomato in all the treatments in both studied seasons, as compare to control. Among

the treatments used, seed + seedling + spray treatment of β-aminobutyric acid showed maximum increase in lignin content during both season followed by seed + seedling + spray treatment of salicylic acid. The change in lignin content due to different RIC's treatments showed increase in trend from 30 DAT to 105 DAT.

Tiuterev *et al.* (1996) stated that the mechanism of action of natural disease inducer chitosan was based on increase in lignin biosynthesis and plant cell wall lignification. Vance *et al.* (1980) stated that lignin, a major component of cell walls of vascular plants, accumulated in cell wall mediated defense and was considered a first line defense against successful penetration of invasive pathogens. Lignification renders the cell wall more resistant to mechanical pressure applied during penetration by fungal appressoria as well as more water resistant and thus less accessible to cell wall-degrading enzymes. Freeman and Beattie (2008) stated that many cell walls contain lignin, a heterogeneous polymer of phenolic compounds that gives the cell rigidity. Lignin is the primary component of wood and cell walls that become "lignified" and were highly impermeable to pathogens and difficult for small insects to chew.

5.3.4 Changes in phenyl alanine ammonia lyase activity, peroxidise activity and polyphenol oxidase activity due to resistance inducing chemicals

In the present investigations it was noticed that there was higher PAL activity in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the PAL activity of tomato in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of salicylic acid showed maximum increase in PAL activity during both season which is followed by spray treatment of salicylic acid. The change in activity of PAL due to

different RIC's treatments showed increase in trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

The higher peroxidase activity was observed in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the peroxidase activity of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of salicylic acid showed maximum increase in peroxidase activity during both season followed by spray treatment of salicylic acid. The change in activity of peroxidase due to different RIC's treatments showed increase in trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

Higher polyphenol oxidase content was observed in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the polyphenol oxidase content of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of salicylic acid showed maximum increase in polyphenol oxidase activity during both season followed by spray treatment of salicylic acid. The change in activity of polyphenol oxidase due to different RIC's treatments showed increase in trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

Mur *et al*. (1996) proposed a dual role of salicylic acid in induced resistance, a direct one leading to the expression of some PR-proteins and an indirect one regulating augmented expression of defense related gene such as phenyl alanine ammonia lyase upon ilucidation of defense. Conrath *et al*. (2002) demonstrated that pretreatment of salicylic acid or benzothiadiazole leads direct activation of certain defence related gene including those encoding phenyl alanine ammonia lyase. Ju *et al*. (2002) studied the effect of seedling treatment of salicylic acid on the activity of peroxidase, polyphenol oxidase and phenyl alanine ammonia

lyase at two leaves stage in cucumber and reported the change in peroxidase activity at 24 hr after treatment which increased steadily till 144 hr with 193 per cent increment as compared to control. Similarly the activity of polyphenol oxidase increased by 61.7 per cent at 144 hr after treatment. The phenyl alanine ammonia lyase activity reached a peak 72 hr after treatment. Feng *et al.* (2004) reported the increased activity of phenyl alanine ammonia lyase, peroxidase and polyphenol oxidase within 7 days of salicylic acid treatment as compared to control and the increased activity of these enzymes was involved in the biochemical mechanism of salicylic acid induced resistance to *Verticillium* wilt. Korosi (2011) reported that the activator treatment of INA, BABA and BTH increased the defence related polyphenol oxidase and peroxidase enzyme activity both in susceptible and resistance sunflower against downy mildew pathogen. Akinwunmi *et al.* (2001) also reported that increase in the activities of two key enzymes of phenyl propanoid pathway and phenyl alanine ammoia lyase was responsible for resistance against *Colletotrichum destructivum* and the increase in enzyme activity was obtained with the use of BTH. Hassni *et al.* (2004) reported the increased activity of peroxidase and polyphenol oxidase with the application of chitosan. Sujeeth *et al.* (2010) reported the increased activity of peroxidase with application of chitosan. Our results of increased polyphenoloxidase, peroxidase and phenyl alanine ammonia lyase are in conformity with those of above reports.

Thus it is proved beyond doubt that the resistance inducers particularly salicylic acid, β–aminobutyric acid and chitosan elicits the increased activity of polyphenol oxidase, peroxidase and phenyl alanine ammonia lyase of the phenolic synthesis pathway and increased level of phenol content in the RIC's treated plants, inhibited the pathogen and process of pathogenesis and are the basics of the resistance induction mechanism.

5.3.5 Changes in β 1, 3 glucanase activity and chitinase activity

The obtained results confirmed that there was higher β 1, 3 glucanase activity in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the β 1, 3 glucanase activity of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment of β-aminobutyric acid showed maximum increase in β 1, 3 glucanase activity during both season followed by seed + seedling + spray treatment of salicylic acid. The change in activity of β 1, 3 glucanase due to different RIC's treatments showed increasing trend from 30 DAT to 105 DAT.

Higher chitinase content was observed in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the chitinase content of tomato in all the treatments in both studied seasons, as compare to control. Among the treatments used, seed + seedling + spray treatment spray treatment of salicylic acid showed maximum increase in chitinase activity during both season followed by spray treatment of salicylic acid. The change in activity of chitinase due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity increased at 30 to 60 DAT but decreased at 90 to105 DAT.

Tosun *et al*. (2006) reported salicylic acid induced accumulation of PR-proteins i.e. chitinase and β-1,3 glucanase in tomato plants which confers resistance against *Phytophthora infestans*. Feng *et al*. (2004) also reported increase in glucanase activity in cotton plant with application of salicylic acid to confirm resistance against *Verticillium* wilt. Barilli *et al* (2009) reported the enhancement of PR-proteins such as β-1,3 glucanase and chitinase by application of benzothiadiazole and β–aminobutyric acid in pea plants to confer the reisitance against pea rust

pathogen. Iriti *et al*. (2009) and Hadrami *et al*. (2010) also reported the increase in PR-proteins with application of chitosan. Raut and Borkar (2014) studied the effect of application of resistance inducing chemicals on PR-proteins accumulation in tomato plant during period of induced resistance against *alternaria* leaf blight and reported that the application of RIC SA and BABA elicits the increased activity of PR-proteins *viz*. chitinase and β 1,3-glucanase which inhibit the process of pathogenesis in susceptible tomato cultivars to exhibit the resistance. Thus our results also confirms that resistance inducing chemicals accumulate the PR-proteins i.e β-1,3 glucanase and chitinase to confer resistance against pathogen in tomato.

6. SUMMARY AND CONCLUSION

The results obtained during the investigation on "Field efficacy of resistance inducing chemicals against tomato diseases" are summarized as below.

The resistance inducing chemicals *viz.* salicylic acid @1.5 mM conc., β–aminobutyric acid @ 15 mM conc. and chitosan @15 mM conc. were tested for their field efficacy in induction of resistance in tomato crop against naturally occurring diseases when exposed to open field condition.

The results obtained showed that all the RIC's tested were effective with significant reductions in the disease incidence, disease severity and AUDPC value of disease.

All the resistance inducing chemicals *viz.* salicylic acid, β-amino butyric acid and chitosan enhanced the resistance to early blight disease incidence and severity of tomato when the seeds and seedlings were subjected to different treatments of the resistance inducing chemicals. In all the treatments percent disease incidence and severity were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence and severity increase was comparatively low in case of RIC's treated plots over control. The incidence and severity of early blight of tomato on *var. Dhanashree* was differing significantly due to different treatments of RIC's. The lowest records of these two parameters were noted at the seed + seedling + spray treatments of salicylic acid throughout the crop growth period. They reduced both incidence and severity of early blight. The most effective treatments were seed + seedling + spray treatment of salicylic acid (Pooled PDI: 67.40 %, Pooled PDS: 33.18 % and Pooled PDC : 17.05 %) followed by spray treatment of salicylic acid (Pooled PDI: 69.98 %, Pooled PDS: 34.46 % and Pooled PDC : 13.85 %) and seed + seedling + spray treatment of chitosan (Pooled

PDI : 71.85 %, Pooled PDS: 34.88 % and Pooled PDC : 12.80 %) against untreated control. The pooled percent reduction in early blight disease incidence and severity were in the range of 7.32 to 20.81 per cent and 3.20 to 17.05 per cent, respectively.

In case of early blight of tomato, treatment T_5 of SA-S+SL+SPY was found significantly superior over other treatments in minimizing the pooled AUDPC value of disease (1287.78) which was at par with treatment T_3 of SA-SPY (1309.48) and treatment T_{15} of CHT-S+SL+SPY (1355.65).

The obtained results clearly showed that all the resistance inducing chemicals *viz.* salicylic acid, β-amino butyric acid and chitosan enhanced the resistance to powdery mildew disease incidence and severity of tomato during *kharif* 2014 and *kharif* 2015 when the seeds and seedlings were subjected to different treatments of the resistance inducing chemicals. In all treatments percent disease incidence and severity were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence and severity increase was comparatively low in case of RIC's treated plots over control. The incidence and severity of powdery mildew of tomato on *var. Dhanashree* was differing significantly due to different treatments of RIC's. The lowest records of these two parameters were noted at the seed + seedling + spray treatments of BABA throughout the crop growth period. They reduced both incidence and severity of powdery mildew. The most effective treatments were seed + seedling + spray treatment of BABA (pooled PDI: 65.51 %, pooled PDS: 33.95 % and pooled PDC: 37.28 %) followed by seed + seedling + spray treatment of salicylic acid (pooled PDI: 67.42 %, pooled PDS: 35.91 %, and pooled PDC : 33.66) and seed + seedling + spray treatment of chitosan (pooled PDI: 68.19 %, pooled PDS: 36.82 %, and pooled PDC: 31.98 %) against untreated control (pooled PDI: 77.28 %, pooled PDS: 54.13 %). The pooled percent reduction in powdery mildew disease

incidence and severity were in the range of 2.94 to 15.93 per cent and 14.61 to 37.28 per cent, respectively.

The pooled AUDPC value of powdery mildew disease of tomato was found minimum in treatment T_{10} of BABA-S+SL+SPY (1195.09) which was at par with treatment T_5 of SA-S+SL+SPY (1305.34) and T_{15} of CHT-S+SL+SPY (1316.50).

The RIC's tested *viz.* salicylic acid, β-amino butyric acid and chitosan enhanced the resistance to tomato leaf curl virus disease incidence and severity when the seeds and seedlings were subjected to different treatments of the resistance inducing chemicals. It is evident from the data that in all treatments percent disease incidence and severity were considerably lower in RIC's treated plots. Also, the rate of percent disease incidence and severity increase was comparatively low in case of RIC's treated plots over control. The incidence and severity of tomato leaf curl virus disease on *var. Dhanashree* was differing significantly due to different treatments of RIC's. The lowest records of these two parameters were noted at the seed + seedling + spray treatments of SA throughout the crop growth period. They reduced both incidence and severity of tomato leaf curl virus disease. The most effective treatment was seed + seedling + spray treatment of salicylic acid (pooled PDI: 28.13%, pooled PDS: 62.11 % and pooled PDC: 10.37 %) followed by spray treatment of salicylic acid (pooled PDI: 28.91 %, pooled PDS: 63.46 % and pooled PDC: 8.41 %) and seed + seedling + spray treatment of chitosan (pooled PDI: 28.91 %, pooled PDS: 63.95 % and pooled PDC: 7.71 %) against untreated control (pooled PDI: 30.74% and pooled PDS: 69.29 %). The pooled percent reduction in tomato leaf curl virus disease incidence and severity were in the range of 0.97 to 8.48 per cent and 1.88 to 10.37 per cent, respectively.

The tomato leaf curl virus disease AUDPC value was found minimum in treatment T_5 of SA-S+SL+SPY (2162.95) which were

significantly superior over other treatment. The highest AUDPC value was observed in untreated control (3286.06).

In case of tomato spotted wilt virus disease, the obtained result showed that all the treatments comparatively reduced the disease incidence over control. However, the magnitude of reduction varied from treatment to treatment. In all treatments the percent disease incidence was comparatively lower in RIC's treated plots. There were no significant differences for any of the treatments on Tomato spotted wilt virus disease incidence during *Kharif* 2015. Pooled analysis also showed same results as *Kharif* 2015. Hence the differences in the results between the two growing seasons may be due the variation in the environmental conditions. Comparatively highest reduction in percent disease incidence was observed in treatment T_5 of SA- S+ SL+ SPY (25.01 %) followed by treatment T_3 of SA-SPY (21.90 %) and treatment T_{15} of CHT-S+SL+SPY (21.90 %).

The investigation confirms that the application of RIC's significantly influenced the yield of tomato during both seasons (*kharif* 2014 and *kharif* 2015) under field condition. All RIC's promoted yield of tomato as compared to control treatment. Among RIC's tested, the salicylic acid was found most effective in increasing yield of tomato. Among all the treatments, significantly highest yield was recorded with the treatment T_5 of SA-S+SL+SPY (*kharif* 2014: 35.63 t/ha, *kharif* 2015 : 31.78 t/ha and pooled 33.71 t/ha) followed by treatment T_3 of SA-SPY (*kharif* 2014 : 34.04 t/ha, *kharif* 2015 : 30.28 t/ha and pooled : 32.16 t/ha) and T_{15} of CHT-S+SL+SPY (*kharif* 2014 : 34.39 t/ha, *kharif* 2015 : 31.58 t/ha and pooled : 32.99 t/ha). However the minor differences in the results between the two growing seasons may be due the variation in the environmental conditions.

The evidences on effect of RIC's on anatomical changes in tomato showed that all assigned treatments of RIC's significantly increased epidermis cum cuticle layer thickness in both studied seasons

(*kharif* 2014 and 2015). The increase in thickness of epidermis cum cuticle due to RIC's treatments was in the range of 12.38 to 77.72 per cent and 12.28 to 76.00 per cent for both studied season and 12.33 to 76.86 per cent in pooled data. The most effective treatment which induced highest increase in epidermis cum cuticle layer thickness was treatment T_5 of SA-S+SL+SPY (*kharif* 2014 : 29.45 µm, *kharif* 2015 : 29.13 µm and pooled : 29.29 µm) which is followed by treatment T_3 of SA-SPY (*kharif* 2014 : 28.16 µm, *kharif* 2015 : 27.86 µm and pooled : 28.01µm) and treatment T15 of CHT-S+SL+SPY (*kharif* 2014 : 27.45µm, *kharif* 2015 : 27.17 µm and pooled : 27.31 µm).

Also indicated that all assigned treatments of RIC's significantly increased palisade mesophyll layer thickness in both studied seasons (*kharif* 2014 and 2015). The increase in thickness of palisade mesophyll due to RIC's treatments was in the range of 4.47 to 28.26 per cent and 3.57 to 27.37 per cent for both studied season. The most effective treatment which induced highest increase in palisade mesophyll thickness was treatment T_5 of SA-S+SL+SPY (*kharif* 2014 : 152.67 µm, *kharif* 2015 : 152.83 µm and pooled : 152.75 µm) which was at par with treatment T_3 of SA-SPY (*kharif* 2014 : 152.37 µm, *kharif* 2015 : 152.53 µm and pooled : 152.45 µm) followed by treatment T_{15} of CHT-S+SL+SPY (*kharif* 2014 : 148.15 µm, *kharif* 2015 : 148.31 µm and pooled : 148.23 µm).

The critical analysis of stomatal characteristics of tomato leaves indicated that irrespective of RIC's treatments the stomatal frequency (No. of stomata/mm^2) was fairly high on lower (abaxial) leaf surface than the upper (adaxial) leaf surface for both studied season. The result also showed that different RIC's treatments have no significant effect on stomatal frequency of adaxial and abaxial surface. Relatively higher reduction in stomatal frequency was observed in treatment T_5 of SA-S+SL+SPY as compared to control.

Though different RIC's have no effect on stomatal frequency, the result showed that it has a marked effect on stomatal size. The treatment T_{10} of (BABA-S+SL+SPY) significantly reduces the stomatal length and width of stomata for both studied season (*kharif* 2014 : 16.15 × 11.04 µm, *kharif* 2015 : 17.47 × 12.36 µm and pooled : 16.81 × 11.70 µm) as compared to control (*kharif* 2014 : 21.45 × 13.68 µm, *kharif* 2015 : 25.96 × 19.06 µm and pooled : 23.71 × 16.37µm) which was followed by treatment T_5 of SA-S+SL+SPY (*kharif* 2014 : 16.56 × 11.18 µm, *kharif* 2015 : 17.88 × 12.50 µm and pooled : 17.22 × 11.84 µm) and treatment T_{15} of CHT-S+SL+SPY (*kharif* 2014 : 16.95 × 11.34 µm, *kharif* 2015 : 18.27 × 12.66 µm and pooled : 17.61 × 12.00 µm). The reduction in the size of stomata due to RIC's treatments was in the range of 6.06 - 24.71 per cent (stomata length) to 4.90 - 19.30 per cent (stomata width) and 10.44 - 32.70 per cent (stomata length) to 13.22 – 35.15 per cent (stomata width) for both studied season and 8.48 - 29.10 per cent (stomata length) to 8.43 - 28.53 per cent (stomata width) for pooled data.

The investigation on effect of RIC's on biochemical aspect reflects increase in biochemical constituents *viz.* protein content, sugar content, phenol content, lignin content and increase in enzyme activities *viz.* phenyl alanine ammonia lyase activity, peroxidise activity, polyphenol oxidase activity and pathogenesis related proteins *viz.* β-1,3-glucanase activity and chitinase activity in treated plant as compare to untreated control.

The increase in biochemical constituents was inhibitory to fungal pathogen, increased the thickness of epidermal layer as primary barrier to fungal penetration and PR-proteins triggered the resistance response in plants.

Higher soluble protein content was observed in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the soluble protein content of tomato leaves in the range of 16.37 to 53.90 per cent

during *kharif* 2014, 4.56 to 48.95 per cent during *kharif* 2015 and 12.00 to 51.29 per cent in pooled analysis, as compare to control. Among the treatments used, treatment T_5 of SA-S+SL+SPY showed maximum increase in soluble protein content during both season (15.79 mg gm^{-1} of fresh weight during *kharif* 2014, 16.98 mg gm^{-1} of fresh weight during *kharif* 2015 and 16.39 mg gm^{-1} of fresh weight in pooled analysis) followed by treatment T3 of SA-SPY (14.20 mg gm^{-1} of fresh weight during *kharif* 2014, 16.64 mg gm^{-1} of fresh weight during *kharif* 2015 and 15.42 mg gm^{-1} of fresh weight in pooled analysis). The change in content of soluble protein due to different RIC's treatments showed increasing trend throughout the crop growth period. The increase in protein content in tomato leaves did not have direct effect on leaf blight pathogen. However, increase in PR-proteins triggered the resistance response of tomato leaves to pathogen.

Increase in reducing sugar content in tomato leaves was observed in plants treated with different RIC's as compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the reducing sugar content of tomato leaves in the range of 10.48 to 97.62 per cent during *kharif* 2014, 36.67 to 91.67 per cent during *kharif* 2015 and 26.22 to 94.44 per cent in pooled analysis, in all the treatments, as compare to control. Among the treatments used, treatment T_{10} of BABA-S+SL+SPY showed maximum increase in reducing sugar content during both season (4.15 mg gm^{-1} of fresh weight during *kharif* 2014, 4.60 mg gm^{-1} of fresh weight during *kharif* 2015 and 4.38 mg gm^{-1} of fresh weight in pooled analysis) followed by treatment T5 of SA-S+SL+SPY (4.02 mg gm^{-1} of fresh weight during *kharif* 2014, 4.55 mg gm^{-1} of fresh weight during *kharif* 2015 and 4.29 mg gm^{-1} of fresh weight in pooled analysis). The change in content of reducing sugar due to different RIC's treatments showed fluctuation in trend from 30 DAT to 105 DAT, whereas in most cases the activity decreased from 30 to 60 DAT but increased from 90DAT to105 DAT. Thus the increase level of

reducing sugar content in leaves might inhibit the pathogen multiplication, its spread and pathogenesis.

Higher total phenol content was observed in tomato leaves treated with different RIC's as compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the total phenol content of tomato in all the treatments in both studied seasons, as compare to control. The range of increase in total phenol content was 5.33 to 38.67 per cent during *kharif* 2014, 6.76 to 41.89 per cent during *kharif* 2015 and 6.04 to 40.27 per cent in pooled analysis. Among the treatments used, treatment T_5 of SA-S+SL+SPY showed maximum increase in total phenol content during both season (2.08 mg gm^{-1} of fresh weight during *kharif* 2014, 2.10 mg gm^{-1} of fresh weight during *kharif* 2015 and 2.09 mg gm^{-1} of fresh weight in pooled analysis) followed by T_3 of SA-SPY (1.82 mg gm^{-1} of fresh weight during *kharif* 2014, 1.80 mg gm^{-1} of fresh weight during *kharif* 2015 and 1.81 mg gm^{-1} of fresh weight in pooled analysis). The change in total phenol content due to different RIC's treatments showed increase in trend from 30 DAT to 90 DAT, whereas the activity decreased at 105 DAT. Thus the increase level of phenol content in leaves might inhibit the pathogen multiplication, its spread and pathogenesis.

Higher lignin content was observed in tomato leaves treated with different RIC's as compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the lignin content of tomato in all the treatments in both studied seasons, as compare to control. The range of increase in lignin content was 4.83 to 83.45 per cent during *kharif* 2014, 2.01 to 79.87 per cent during *kharif* 2015 and 3.40 to 81.63 per cent in pooled analysis. Among the treatments used, treatment T_{10} of BABA-S+SL+SPY showed maximum increase in lignin content during both season (2.66 % during *kharif* 2014, 2.68 % during *kharif* 2015 and 2.67 % in pooled analysis) followed by treatment T_5 of SA-S+SL+SPY (2.63 % during *kharif* 2014, 2.62 %

during *kharif* 2015 and 2.63 % in pooled analysis). The change in lignin content due to different RIC's treatments showed increase in trend throughout the crop growth period. Increase in lignin biosynthesis causes cell wall lignification which makes cell wall highly impermeable to pathogen for initiation of infection and is first line defense against invasion of pathogen.

Higher phyenyl alanine ammonia lyase (PAL) activity was observed in tomato leaves treated with different RIC's as compared to control during *kharif* 2014 and *kharif* 2015. All assigned treatments of RIC's significantly increased the PAL activity of tomato in both studied seasons, as compare to control. The range of increase in PAL activity was 10.61to 40.15 per cent during *kharif* 2014, 19.17 to 61.41 per cent during *kharif* 2015 and 14.54 to 49.39 per cent in pooled analysis. Among the treatments used treatment T_5 of SA-S+SL+SPY showed maximum increase in PAL activity during both seasons (12.82 µ moles of cinnamic acid produced min^{-1} mg^{-1} soluble protein) during *kharif* 2014, 11.35 µ moles of cinnamic acid produced min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 12.09 µ moles of cinnamic acid produced min^{-1} mg^{-1} soluble protein in pooled analysis) which is followed by T_3 of SA-SPY (12.18 µmoles of cinnamic acid produced min^{-1} mg^{-1} soluble protein during *kharif* 2014, 11.03 µmoles of cinnamic acid produced min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 11.60 µmoles of cinnamic acid produced min^{-1} mg^{-1} soluble protein in pooled analysis). The change in activity of PAL due to different RIC's treatments showed increase in trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT. PAL is an enzyme of phenolic synthesis pathway which is responsible for increase level of phenols in tomato leaf tissues due to resistance inducing chemicals and the increase level of phenolic was inhibitory to plant pathogens.

The higher peroxidase activity was observed in tomato leaves treated with different RIC's as compared to control during *kharif* 2014

and *kharif* 2015. All assigned treatments of RIC's significantly increased the peroxidase activity of tomato leaves in both studied seasons, as compare to control. The range of increase in peroxidase activity was 10.09 to 64.22 per cent during *kharif* 2014, 11.69 to 76.64 per cent during *kharif* 2015 and 11.00 to 69.90 per cent in pooled analysis. Among the treatments used, treatment T_5 of SA-S+SL+SPY showed maximum increase in peroxidase activity during both season (64.22 units min^{-1} mg^{-1} protein during *kharif* 2014, 76.64 units min^{-1} mg^{-1} protein during *kharif* 2015 and 69.90 units min^{-1} mg^{-1} protein in pooled analysis) followed by treatment T3 of SA-SPY (13.59 units min^{-1} mg^{-1} protein during *kharif* 2014, 12.25 units min^{-1} mg^{-1} protein during *kharif* 2015 and 12.92 units min^{-1} mg^{-1} protein in pooled analysis). The change in activity of peroxidase due to different RIC's treatments showed increase in trend from 30 to 90 DAT, whereas the activity decreased at 105 DAT.

Higher polyphenol oxidase content was observed in tomato leaves treated with different RIC's compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the polyphenol oxidase content of tomato leaves in both studied seasons, as compare to control. The range of increase in polyphenol oxidase content was 2.13 to 55.87 per cent during *kharif* 2014, 3.44 to 56.97 per cent during *kharif* 2015 and 2.79 to 56.34 per cent in pooled analysis. Among the treatments used, treatment T5 of SA-S+SL+SPY showed maximum increase in polyphenol oxidase activity during both season (16.99 units min^{-1} mg^{-1} soluble protein during *kharif* 2014, 16.87 units min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 16.93 units min^{-1} mg^{-1} soluble protein in pooled analysis) followed by T_3 of SA-SPY (16.71 units min^{-1} mg^{-1} soluble protein during *kharif* 2014, 16.70 units min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 16.71 units min^{-1} mg^{-1} soluble protein in pooled analysis). The change in activity of polyphenol oxidase due to different RIC's treatments showed increase in trend from 30 to 90

DAT, whereas the activity decreased at 105 DAT. The increase in defense related polyphenol oxidase and peroxidase inhibit the process of pathogenesis and are the basic of resistance induction mechanism.

Higher β 1, 3 glucanase activity was observed in tomato leaves treated with different RIC's as compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the β 1, 3 glucanase activity of tomato in both studied seasons, as compare to control. The range of increase in β 1, 3 glucanase activity was 1.60 to 29.41 per cent during *kharif* 2014, 1.52 to 25.25 per cent during *kharif* 2015 and 1.30 to 19.95 per cent in pooled analysis. Among the treatments used treatment T_{10} of BABA-S+SL+SPY showed maximum increase in β 1, 3 glucanase activity during both season (0.242 mg of glucose released min^{-1} mg^{-1} soluble protein during *kharif* 2014, 0.248 mg of glucose released min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 0.245 mg of glucose released min^{-1} mg^{-1} soluble protein in pooled analysis) followed by treatment T_5 of SA-S+SL+SPY (0.227 mg of glucose released min^{-1} mg^{-1} soluble protein during *kharif* 2014, 0.236 mg of glucose released min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 0.232 mg of glucose released min^{-1} mg^{-1} soluble protein in pooled analysis). The change in activity of β 1, 3 glucanase due to different RIC's treatments showed increasing trend from 30 to 105 DAT.

Higher chitinase content was observed in tomato leaves treated with different RIC's as compared to control during *kharif* 2014 and 2015. All assigned treatments of RIC's significantly increased the chitinase content of tomato leaves in both studied seasons, as compare to control. The range of increase in chitinase activity was 5.17 to 64.83 per cent during *kharif* 2014, 2.23 to 68.75 per cent during *kharif* 2015 and 3.89 to 66.54 per cent in pooled analysis. Among the treatments used treatment T_5 of SA-S+SL+SPY showed maximum increase in chitinase activity during both season (0.478 mg of N-acetyl glucosamine released min-1 mg-1 soluble protein during *kharif* 2014, 0.378 mg of N-

acetyl glucosamine released min^{-1} mg^{-1} soluble protein during *kharif* 2015 and 0.428 mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein in pooled analysis) followed by treatment T3 of SA-S+SL+SPY (0.452 mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein during *kharif* 2014, 0.364 mg of N-acetyl glucosamine released $min^{-1}mg^{-1}$ soluble protein during *kharif* 2015 and 0.413 mg of N-acetyl glucosamine released min^{-1} mg^{-1} soluble protein in pooled analysis). The change in activity of chitinase due to different RIC's treatments showed fluctuation in trend from 30 to 105 DAT, whereas in most cases the activity increased at 30 to 60 DAT but decreased at 90 to 105 DAT.

Thus the resistance inducers particularly salicylic acid and β–aminobutyric acid elicits the increased activity of pathogenesis related proteins (PR-proteins) β-1, 3 glucanase and chitinase of the phenolic synthesis pathway which inhibit the pathogen and process of pathogenesis to induce resistance in susceptible cultivars.

Conclusion:

1. The field study of efficacy of RIC'S against tomato diseases revealed that seed + seedling + spray (15 days interval) treatment of salicylic acid @ 1.5 mM conc. were merely effective in reducing the incidence and severity of early blight, leaf curl disease of tomato, whereas Powdery mildew of tomato was controlled by the seed + seedling+ spray treatment of BABA.
2. Increased in tomato yield was observed in the treatment T_5 of Seed + seedling + spray of salicylic acid @1.5mM conc. compared to non-treated plants.
3. The seed + seedling + spray treatment of SA @ 1.5 mM conc. increased the thickness of epidermis cum cuticle layer and palisade mesophyll cells followed by Chitosan and BABA. Increase in thickness of epidermis cum cuticle layer.
4. Resistance inducing chemicals under study were less effective in increasing the stomatal frequency but found effective in reducing

the stomatal size. The reduction in length and breadth of stomata was found maximum in seed + seedling + spray treatment of BABA.

5. RIC's increased soluble protein content, total phenol content, phenylalanine ammonia lyase activity, peroxidase activity, polyphenol oxidase activity, chitinase activity and these were maximum for seed + seedling + spray treatment of SA @ 1.5mM concentration.
6. The reducing sugar content, lignin content, β 1, 3 glucanase activities and these were maximum in seed + seedling + spray treatment of BABA @ 15mM concentration.
7. RIC's were effective up to early growth stage of tomato in complex disease situation while these were observed to be less effective in advance growth stage of the plant.
8. Thus resistance inducing chemicals *viz.* salicylic acid, β–aminobutyric acid and chitosan does reduce susceptibility of tomato plant to disease causing pathogen, due to the anatomical and biochemical alteration and that are able to induced broad disease resistance in tomato hence offer an additional option for farmers to complement genetic disease resistance and the use of fungicides.

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8. VITA

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of

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