International Advance Journal of Engineering, Science and Management (IAJESM)
ISSN -2393-8048, January-June 2021, Submitted in June 2021, iajesm2014@gmail.com

Literature Study on the effect of phytotoxic compounds of Alternaria alternata on physiology of *Solanum lycopersicum*

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Abstract:

Plants undergo continuous exposure to various biotic and abiotic stresses in their natural environment. In the course of evolution, they have evolved specific mechanisms allowing them to adapt and survive stressfully. Exposure of plants to biotic and abiotic stresses induces a disruption in plant metabolism implying physiological costs (Heil and Bostock, 2002; Swarbrick et al., 2006; Bolton, 2009; Massad et al., 2012; Stotz et al., 2014; Prasch and Sonnewald, 2015) leading to a reduction in fitness and ultimately loss in productivity (Shao et al., 2008). Biotic stress is an additional challenge inducing a strong pressure on plants adding to the damage through pathogen or herbivore attack (Brown and Hovmoller, 2002; Strauss and Zangerl, 2002; Maron and Crone, 2006; Mordecai, 2011). Moreover, abiotic stress has a huge impact on growth. Consequently, they are responsible for severe losses in the yield. The resulting growth reductions can reach >50% in most plant species (Wang et al. 2003).

Keywords: Literature, Phytotoxic, Physiology, Solanum

Introduction: A cell death pathway is thought to be activated during the HR of plants or against invading pathogens. Mostly cell death inducers in plants are toxins from a number of pathogens such as the hairpins from Pseudomonas syringae and Erwinia amylovora (Samuel et al., 2005), the fungal toxin victorin from Helminthosporium victoriae (Yao et al., 2001), xylanase from Trichoderma viridae (Grant and Mansfield, 1999), AAL-toxin from Alternaria alternata f. sp. lycopersici (Spassieva et al., 2002) and fumonisin B1 from Fusarium moniliforme (Wang et al., 1996). Also, plant viruses such as tobacco mosaic virus (TMV) have been reported to elicit PCD (Del Pozo and Lam, 2003). The recognition of an invading pathogen triggers a signal transduction pathway that results in the coordinated activation of many plant defense mechanisms, including the accumulation of SA, the synthesis of pathogenesis-related proteins, the thickening of cell walls and the increased production of antimicrobial compounds (phytoalexins) as well as ROS leading to cell death (Durrant and Dong, 2004). Plants activate genetic programs for cellular suicide under a variety of circumstances. There is not always a requirement for a living pathogen to trigger the HR. Purified elicitors, such as fungal toxin fumonisin B1 or hairpins can induce physiological changes associated with disease resistance (Numberger et al., 1994). It has been shown that spraying plants with hairpins coordinately induces systemic resistance to pathogens and micro-HR (Peng et al., 2003). The spontaneous activation of HR cell death in absence of pathogens has also been reported in transgenic plants that express foreign genes.

Literature Survey

Being one of the most popular vegetable throughout the world, the importance of its cultivation is threatened by a wide array of pathogens (fungi, bacteria, viruses and nematodes). This diversity of the pathogens emphasizes the importance of the tomato pathosystem as a favourable model for studying plant-pathogen interactions. Moreover, tomato carries several specific resistance (R) genes against a variety of pathogens, which make this plant suitable for genetic studies of plant host-specific resistance based on the gene for gene theory. The famous models are the interactions with the fungal mold Cladosporium fulvum (Joosten and de Wit, 1999), the bacterial speck Pseduomonas syringae pv. Tomato (Ronald et al., 1992) and the fungal wilt Verticillium dahliae (Fradin and Thomma 2006). There has also been substantial investigation of interactions between tomato and other pathogens such as Fusarium oxysporum, Alternaria alternata, cucumber mosaic virus (CMV) (Di Pietro et al., 2003). Tomato expresses a large number of defense compounds and is also used as a model plant to test whether an elicitor or a particular pathogen is able to induce basal resistance or to activate forms of induced resistance through SA or JA/ET signaling pathways. In the last twenty years, this plant has been successfully used as a model plant to investigate the induction of defense pathways after exposure to fungal, bacterial and abiotic

molecules, showing triggering of different mechanisms of resistance. Understanding these mechanisms in order to improve crop production is a main goal of this study.

TeA, iso-tenuazonic acid and its salts exhibit herbicidal activity with broad spectrum properties, quick killing and high efficiency detrimental effects on plants (Devi et al., 2010a). TeA has been found in Canadian lentils and is reported from beer and cereal foods (Ostry et al., 2004; Siegel et al., 2010; Asam et al., 2012). Recently, many in vitro studies have reported that AOH causes DNA damage by inducing cell cycle arrest (Pfeiffer et al., 2007; Fehr et al., 2009) which leads to mutations in living beings (Brugger et al., 2006; Schreck et al., 2012; Solhaug et al., 2013). Furthermore, AOH also exhibits cytotoxic, foetotoxic, mutagenic and teratogenic effects that are responsible for the etiology of oesophageal cancer (Liu et al., 1992). It has been shown that both AME and AOH have potential carcinogenic, genotoxic and cytotoxic activity in both microbial and mammalian cell systems (Liu et al., 1992). According to Graf et al. (2012), in case of Alternaria alternata, an external addition of alternariol restored the pathogenicity. Many other fungal genera such as Stagonospora nodorum (Tan et al., 2009) and Phomopsis isolates (Abreu et al., 2012) have also been found to produce AOH and AME. TeA is also produced by other species of fungi including Pyricularia oryzae and Phoma sorghina (Iwasaki et al., 1972; Steyn and Rabie, 1976; Bottalico and Logrieco, 1998; Ostry, 2008).

ROS are generally produced during aerobic phase of photosynthesis and photorespiration (Asada and Takahashi, 1987; Mittler, 2002; Kotchoni et al., 2006). Accumulation of these molecules can also be detected in peroxisomes under abiotic (Ramanjulu and Bartels, 2002; Mittler, 2002) and biotic stresses (Mittler, 2002). During cellular metabolism, oxygen molecules are often converted into several intermediates such as superoxide ($O2^{\cdot-}$), hydrogen peroxide (H2O2), hydroxyl radical ($OH^{\cdot}$), which often leak out from electron transport system (Banerjee et al., 2003) and can therefore, be detected as traces in various cell compartments. New sources of ROS production include cell-wall-bound peroxidases, chloroplasts and mitochondria (Mittler, 2002; Davletova et al., 2005) (Fig. 2.3). The chloroplast is considered to be a focal point of ROS metabolism. It is a major producer of $O2^{-}$ and H2O2 and contains a large array of ROS-scavenging mechanisms that have been extensively studied (Davletova et al., 2005).

Acclimation of plants to changes in their environment requires a new state of cellular homeostasis achieved by a delicate balance between multiple pathways that reside in different cellular compartments. Despite the fact that ROS has been considered as toxic molecules during stress conditions, recent studies have shown that ROS play a key role in plants as signal transduction molecules acting as signaling molecules that regulate stress responses as well as growth and development of plants (Foyer and Noctor, 2005; Miller et al., 2010; Barba-Espín et al., 2011). A direct result of stress-induced cellular changes is overproduction of ROS in plants are produced in such a way that they are confined to a small area and also in specific pattern in biological responses. The production of ROS is an inevitable consequence of aerobic metabolism during stressful conditions (Bhattacharjee, 2012). ROS are highly reactive and toxic, affecting various cellular functions in plant cells through damage to nucleic acids, protein oxidation and lipid peroxidation, eventually resulting in cell death (Fig. 2.4) (Bhattacharjee, 2005; Amirsadeghi et al., 2006; Suzuki et al., 2012; Tuteja et al., 2012). ROS toxicity due to stresses is considered to be one of the major causes of low crop productivity worldwide (Vadez et al., 2012).

ROS system consists of both free radicals including superoxide ($O2^{\cdot-}$), hydroxyl radicals ($OH^{\cdot}$), alkoxyl radicals and non-radicals like hydrogen peroxide (H2O2) and singlet oxygen (1O2) (Gill and Tuteja, 2010). During stress conditions, these species are always formed by the leakage of electrons from the electron transport activities of chloroplasts, mitochondria and plasma membranes or also as a by-product of various metabolic pathways localized in different cellular compartments (Del Rio et al., 2006; Gill and Tuteja, 2010; Sharma et al., 2012; Fig. 2.4). Depending upon their concentrations, ROS play dual role as both deleterious and beneficial species in plants (Kotchoni and Gachomo, 2006). At low/moderate concentrations, ROS act as second messengers in various intercellular signaling pathways

that mediate many responses in plants, thus regulating cellular redox state whereas at higher concentrations they have detrimental effects on plant growth (Mittler, 2002; Torres et al., 2002; Yan et al., 2007; Miller et al., 2008; Sharma et al., 2012). Plants have various metabolic and developmental processes which are regulated by cross-talk between ROS and hormones (Kocsy et al., 2013). ROS can activate the synthesis of many plant hormones such as brassinosteroids, ethylene, jasmonate and salicylic acid (Ahmad et al., 2010). The phytotoxic metabolites of A. alternata pathogen causes significant damages to cell by disrupting its structural integrity maintained by cellular membranes either by targeting the proteins relevant to it directly or indirectly by inducing the production of key ROS molecules generated by ROS system which disrupt and change the overall chemical composition of its structure. This change in membrane structure is contributed by modification of the chemical groups including addition of peroxide moiety resulting into lipid peroxidation (Heiser et al., 1998). The increased production of ROS is specific attribute of the defense responses to pathogens in plants (Shereefa and Kumaraswamy, 2014) or involved directly in the expression of resistance genes (Bandyopadhyay et al., 1999).

The first two enzymes involved in nitrate assimilation are nitrate reductase (NR) and nitrite reductase (NiR). Of these two enzymes, NR is considered to catalyze the rate-limiting step in $NO3^-$ assimilation because it initiates the reaction when $NO3^-$ is available (Fonseca et al., 1997). Nitrate, taken up by $NO3^-$ transporters, is reduced to ammonium by the sequential reaction of NR in the cytosol and NiR in the plastids/chloroplasts. NR is the enzyme, which is involved in nitrogen metabolism, play important role in amino acid biosynthesis and regulates the protein synthesis (Nair and Abrol, 1977; Harris et al., 2000). Nitrate is first reduced to nitrite, which is subsequently reduced to ammonia and then incorporated into the amino acids glutamine/glutamate using the C-skeletons produced via other metabolic pathways such as respiration and photosynthesis (Huber et al., 1996). NiR requires reduced ferredoxin to reduce nitrite to ammonia, which is subsequently assimilated via glutamine synthetase (GS) and glutamate synthase (GOGAT) (Andrews et al., 2013). In turn, oxidized ferredoxin accepts new electrons from PSII and PSI. Hence, nitrate assimilation is an alternative sink for the photosynthetic electrons. Ammonium can be directly incorporated into glutamate by the aminating reaction of glutamate dehydrogenase (NADH GDH). Since GDH reversibly deaminates glutamate to NH4 + and 2-oxoglutarate, the physiological role of GDH in vivo remains controversial. The ammonium assimilation into glutamine and glutamate is vital for plant growth as these two amino acids serve as the precursors for the synthesis of the other amino acids as well as almost all nitrogenous compounds. Salt stress inhibits the ammonium assimilation (Sahu et al., 2001; Khadri et al., 2001) and induces changes in the pool of amino acids (Lacerda et al., 2001; Ashraf and Bashir, 2003). NR is also able to synthesize NO from nitrite in tobacco (Lu et al., 2014). Indirectly, nitrate affects plant-pathogen interactions via the production of nitric oxide, which is catalysed by nitrate NR under certain conditions (Kaiser and Huber, 2001).

Lipid peroxidation, a well-established mechanism of cellular injury in plants, is used as an indicator of oxidative stress in cells and tissues. Peroxidation of lipids (primarily the phospholipids of cell membranes) is mechanistically important from free radical production perspective as it is one of the few examples of carbon centred radical production in plant cells (Winston, 1990). Peroxidation of lipids in plant cells appears to be initiated by a number of ROS. Essentially membrane lipid peroxidation involves three distinct stages which include initiation, progression and termination. Initiation event involves transition metal complexes, especially those of Fe and Cu. The role of these metal complexes lies in the fact that either they form an activated oxygen complex that can abstract allylic hydrogens or act as a catalyst in the decomposition of existing lipid hydroperoxides. Although $O2^{\cdot -}$ and H2O2 are capable of initiating the reactions but as $OH^{\cdot}$ is sufficiently reactive, the initiation of lipid peroxidation is mainly mediated by $OH^{\cdot}$. Loosely bound Fe is also able to catalyze the decomposition of lipid peroxides resulting in the formation of alkoxy and peroxy radicals, which further stimulate lipid peroxidation. Lipid peroxidation in plant cells can also be

initiated by the enzyme lipoxygenase. The enzyme is able to initiate the formation of fatty acid hydroperoxides resulting in peroxidation (Winston, 1990).

SA is a key signaling molecule in pathogen-induced disease resistance and plays a direct role in cell death regulation during the HR. It is a major phenylpropanoid compound whose biosynthesis is triggered by various biotic and abiotic stresses. It was originally thought that SA was synthesized only from phenylalanine via cinnamic acid and benzoic acid in tobacco, potato and Arabidopsis (Leon et al., 1993; Yalpani et al., 1999; Mauch-Mani and Slusarenko, 1996; Coquoz et al., 1998; Ribnicky et al., 1998). Phenylalanine ammonia lyase (PAL), which catalyzes the transformation of phenylalanine into cinnamic acid, is a rate-limiting enzyme in the production of phenylpropanoid compounds in tobacco (Bate et al., 1994; Howles et al., 1996). SA accumulation decreases in pathogen-challenged or elicitor-treated plants when endogenous PAL expression is suppressed by means of genetic manipulation or treatment with the PAL inhibitor 2-aminoindan-2-phosphonic acid (Mauch-Mani and Slusarenko, 1996; Coquoz et al., 1998; Pallas et al., 1996). These results suggest that PAL is an important enzyme in the pathway of SA synthesis. SA is a regulator of plant resistance to biotrophic and hemibiotrophic pathogens such as Hyaloperonosporaara bidopsidis and P. syringae and it also regulates SAR (systemic acquired resistance), a well studied type of induced resistance (Glazebrook, 2005). In addition, SA is a central regulator of immunity. It interacts with other signaling pathways (e.g., ethylene and JA pathways), as a strategy to induce the proper resistance responses and to reduce the associated fitness costs (Vlot et al., 2009; Thaler et al., 2012). Identification of SA binding proteins has suggested as how SA could lead to enhanced ROS production. Strikingly, two SA-binding proteins are also antioxidant proteins (Durner and Klessig, 1995, 1996). Both CAT and APX are inhibited by SA concentrations that can be attained locally in pathogen infected tissue (Durner et al., 1997). Inhibition of these ROS detoxification proteins could result in enhanced H2O2 accumulation.

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