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Molecular Mechanisms of Interactions of *Trichoderma* with other Fungal Species

Birinchi K. Sarma[1,*], Sudheer K. Yadav[2], Jai S. Patel[2] and Harikesh B. Singh[1]

[1]*Department of Mycology and Plant Pathology, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi 221005, India*

[2]*Department of Botany, Banaras Hindu University, Varanasi 221005, India*

Abstract: *Trichoderma* species are known globally mostly for the production of industrially useful enzymes as well as their biocontrol ability against plant pathogens. One of the major strategies of biological control is mycoparasitism against fungal pathogens of crop plants. However, till recently the mechanisms of mycoparasitism by biocontrol potential *Trichoderma* species at molecular level were not clearly understood. The biochemical signaling and the involvement of secondary metabolites that lead to mycoparasitic activities of *Trichoderma*, in particular, were not very clearly known earlier. Recent findings in this regard revealed that there are a number of signaling cascades activated during the process of mycoparasitism by *Trichoderma* species against phytopathogenic fungal pathogens. In addition *Trichoderma* also interacts with beneficial root inhabiting fungi like mycorrhizae. The interaction of *Trichoderma* species with mycorrhizal fungi is different as during interaction with mycorrhizal fungi different signaling cascades are activated that lead to a synergistic action. In the current review, we gathered updated evidences regarding the signaling cascades that are generated during interactions between *Trichoderma* species with fungal pathogens resulting mycoparasitism as well as interactions of *Trichoderma* species with mycorrhizal fungi resulting synergism at molecular level. We also highlighted the role of secondary metabolites that are reported to be associated in the signaling processes.

Keywords: cAMP signaling, G-protein, MAP Kinase, mycoparasitism, signal transduction, *Trichoderma*.

INTRODUCTION

The fungal species *Trichoderma* has worldwide occurrence and can be easily isolated from a variety of soils, decomposing woods and other sporocarps. The potentiality of different *Trichoderma* species as effective biocontrol agents against plant diseases especially those caused by soil borne pathogens have been demonstrated long ago. They directly influence the growth and development of mycelia or other surviving propagules of pathogenic fungi through mycoparasitism commonly by releasing antimicrobial secondary metabolites, secreting enzymes that degrade fungal cell wall and forming structures aimed to restrict pathogen growth [1]. Among the fungal biocontrol agents the significance of *Trichoderma* is very high due to its mycoparasitic potentiality against a wide range of fungal pathogens such as *Botrytis cinerea, Fusarium* spp., *Pythium* spp., *Rhizoctonia solani, Sclerotium rolfsii,* and *Sclerotinia sclerotiorum*. *Trichoderma* strains are being used as an alternative to chemical pesticides to manage various plant pathogens mostly attributed to their mycolytic and antibiosis activities as well as plant host mediated physiological changes [2, 3]. They produce a number of antimicrobial secondary metabolites such as peptaibols, gliovirin, and gliotoxin known to inhibit a wide range of plant pathogens [4]. Secondary metabolites produced by fungi are normally not utilized for achieving growth but they play other important roles while interacting with other organisms through facilitating signal transduction [5, 6]. Mycoparasitism by *Trichoderma* [7, 8] could be explained through the presence of an unusually diverse reservoir of secondary metabolite biosynthetic genes evident from the whole genome sequences of *T. virens* and *T. atroviride*, two proven biocontrol species. Products from most of these genes responsible for production of secondary metabolites are associated with mycoparasitism by *Trichoderma* species against other microbes. Some of the highly characterized secondary metabolites belong to the groups of pyrones, terpenoids, steroids and polyketides which are non-polar compounds with low molecular mass. *Trichoderma* spp. also produces nonribosomal peptides, as most other ascomycetes do, such as epipolythiodioxopiperazines (ETPs) and siderophores and many of them are antimicrobial in nature. The precise roles of these compounds in biocontrol by *Trichoderma* species while interacting with other plant pathogens are yet to be clearly understood. However, many of them are reported to act in concert in a synergistic manner with the cell wall degrading enzymes for enhancing host cell wall lysis [9]. Recently, Malmierca *et al.* [10] showed that *Trichoderma* species produce *Fusarium* orthologues trichothecenes such as trichodermin and harzianum A (HA). Disruption of *tri4* gene that hampers biosynthesis of trichothecenes results in lowering down their biocontrol activity against the pathogens *Rhizoctonia solani* and *Botrytis cinerea*. The strain due to silenced *tri4* gene also reduced expression of some defense genes of the salicylic

*Address correspondence to this author at the Department of Mycology and Plant Pathology, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi 221005, India; Tel: 91-542-6702578;
Fax: 91-542-2368993; E-mail: birinchi_ks@yahoo.com

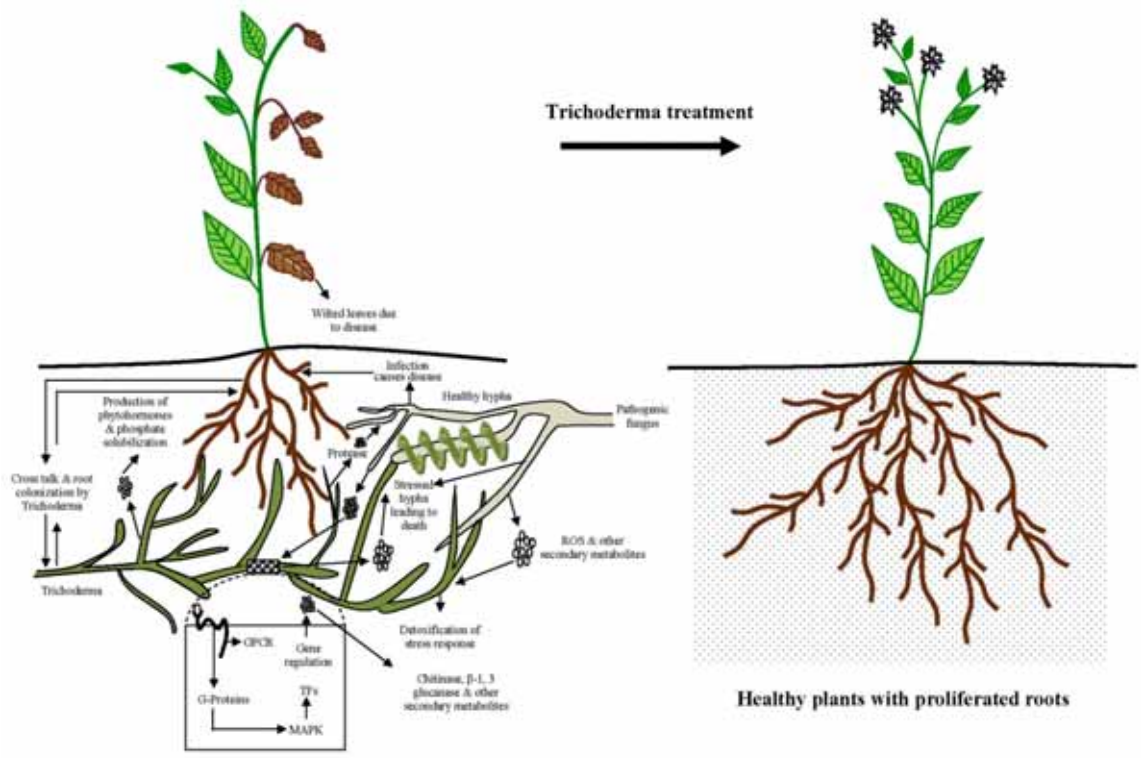

Fig. (1). Molecular mechanisms of *Trichoderma* interacting with other fungal pathogens.

acid (SA) and jasmonate (JA) pathways in tomato against *B. cinerea* compared to the wild-type strain. Such results indicate that *Trichoderma* produced HA sensitize the tomato plants pre-treated with *Trichoderma* and increases the expression of defense genes belonging to SA and JA pathways when challenge against *B. cinerea*. The role of secondary metabolites in fungus-fungus interactions between *Trichoderma* and other fungal species has recently been reviewed by Mukherjee *et al.* [11] and therefore not included in this review. Numerous strains of *Trichoderma* have been shown to interact with other fungi and plants resulting positive impact on the hosts. *Trichoderma* species not only suppress the growth and development of fungal pathogens but also promote growth of the treated plants and stimulate expression of defence genes. Antagonism, parasitism or even killing other fungi are special and widespread mechanisms prevailed in most of the *Trichoderma* spp. Biocontrol efficient strains of *Trichoderma* spp. also establish successfully in the rhizosphere of treated plants where they promote growth of plants and stimulate defence responses when challenged by pathogens [12, 13].

HOST RECOGNITION MECHANISMS OF *TRICHODERMA* FOR MYCOPARASITISM

Trichoderma spp. can use dead fungal biomass as a food and therefore, their nutritional strategies comprise of both saprotrophic and biotrophic nature and it will be appropriate to coin them as mycotrophic rather than mycoparasitic. It is observed that highly conserved signaling components are involved when two fungi interact [14]. *Trichoderma* generally penetrates the host fungus by degrading the cell wall and utilizing their cellular contents. It is achieved with the help of some lytic enzymes including chitinases, glucanases, and proteases that are induced in *Trichoderma* before directly coming in contact with the host and play major roles in biocontrol [15]. During interaction of *Trichoderma* with *R. solani*, a special diffusible factor is identified that is released from the host which is responsible for induced transcription of *ech42* (endochitinase 42-encoding) gene in *Trichoderma* before getting any physical contact [16]. Similarly, during direct contact between *Trichoderma* and its host, coiling by *Trichoderma* mycelia around the hyphae of host is induced by the lectins present in the cell wall of hosts [17]. Production of lytic enzymes and formation of infection structure are triggered by chemical components of host fungus which may be either structural or extracellular and is categorized as induced response [16]. Recent advances in genome sequencing and annotation of *Trichoderma* genomes have made understanding of these phenomena much simpler. Knowledge regarding molecular physiology of mycotrophic life style of *Trichoderma* had also been gained through genomic and transcriptomic studies (Fig. **1**). However, several genes are also expressed in *Trichoderma* spp. either away or on contact with the pathogenic fungi [18, 19]. Such genes are mostly oligopeptide transporter and proteases encoding genes. Many of the proteases encoding genes are from the subtilisin-like serine proteases and are found to be expressed significantly in *T. harzianum* strain CECT 2413 when grown under conditions mimicking biological control [19]. The expression of subtilisin-like proteases encoding genes is also abundant at the contact point between *T. atroviride* and its fungal host species *R. solani* and *S. sclerotiorum*. Over-expression of the proteases gene *prb1* from *T. atroviride* showed its direct involvement in biocontrol activity through enhanced mycoparasitism. The released oligopeptides bind to the receptors present on the surface of *Trichoderma* which

sense nitrogen starvation of *T. atrovirde* [18], a host trapping mechanism very similar to nematophagous fungi, triggered by the nematode released oligopeptides [20]. In *Trichoderma*, the released oligopeptides are sensed by the class IV G protein-coupled receptors (GPCRs) [18]. Two paralogous from the class IV GPCRs are present each in *T. atroviride, T. virens* and *T. jecorina*. Some more GPCRs that are associated with sensing the signals from the host were also reported. Gpr1, a GPCR gene from the cyclic AMP pathway, is also found essential in mycoparasitism by *T. atroviride* [21]. Further, downstream signal transduction from such receptors occur through a conserved G protein signaling cascade which is composed of three subunits, *viz*., Gα, Gβ and Gγ subunits. Mutants that are defective in the Gα subunit cause loss of function in Tga1 and the strain looses mycoparasitic overgrowth on three hosts. The effect is coupled with significant reduction in the activities of chitinase followed by reduced accumulation of the antifungal compound 6-pentyl pyrone [22, 23]. On the other hand the deletion of a *tga1* homologue *tgaA* in *T. virens* also reduced mycoparasitism by the strain on the phytopathogen *S. rolfsii* [24].

SIGNAL TRANSDUCTION IN *TRICHODERMA* DURING MYCOPARASITISM

Mycotrophy and mycoparasitism both are signal dependent mechanisms and as mentioned earlier recognition of fungal host released lectins is the most important initial step. However, plant lectins also induce coiling in *Trichoderma* spp. but lectins released from the plant host do not determine the specificity in attachment of *Trichoderma* spp. to their hosts [22]. Coiling and mycoparasitism cannot be correlated always as self coiling (coiling around own hyphae) in some *Trichoderma* spp. is also observed. It needs further investigation to reveal the exact mechanism(s) of induction of coiling. Hyphal growth of *Trichoderma* spp. takes place along with the host followed by formation of papilla-like structures especially at the point where cell wall degradation and lumen penetration occur prior to mycoparasitism. *Trichoderma* spp. at this stage behaves like a true fungal pathogen that attacks a plant host. The papilla-like structures are very similar to appressorium produced by fungal plant pathogens on the surface of their hosts. Glycerol is produced in these structures that cause increase in turgor and is believed to be essential for exerting mechanical pressure on the host surface that facilitates invasion in to the host cell. At the time of contact during mycoparasitism by *T. atroviride*, upregulation in lipid catabolism and osmoregulation genes takes place which is very similar to pathogenicity by the fungus *Magnaporthe grisea* on its plant host rice [18]. Finally, a concerted effort of released antifungal secondary metabolites and lytic enzymes *Trichoderma* kills the host. *Trichoderma* genome decoded the presence of a large number of enzymes encoding genes essential for biosynthesis of such antifungal compounds. Among the *Trichoderma* species, abundance of nonribosomal-peptide synthetases are observed in *T. virens* [24] compared to the other known fungi whereas *T. atroviride* and *T. virens* have many genes that encode several chitinases [25]. Increased chitinase activity is directly correlated with enhanced mycoparasitic ability in *T. harzianum* and the cellulose-binding ability facilitates tight binding of chitinases with insoluble substrates of chitin. However, deletion of chitinases genes in different *Trichoderma* strains did not result in loss of mycoparasitic as well as biocontrol activities suggesting that production of lesser chitinases due to gene redundancy is one of the several mechanisms adopted by *Trichoderma* spp. for biological control. *Trichoderma* spp. also contains a large number of chitosanases belonging to the GH family. β-1,3-glucan is the second most abundantly present fungal cell wall polymer [26] and β-1,3-glucanases hydrolyses it. β-1,6-glucanase homologue Bgn16.3 overexpression in *T. harzianum* CECT 2413 converted it to a better biocontrol strain with enhanced biocontrol efficacy against *B. cinerea, R. solani* and *Phytophthora citrophthora*. β-1,6-glucanases overproducing strains of *T. harzianum* and *T. virens* also showed enhanced biocontrol against *B. cinerea, R. solani* and *P. ultimum* [1, 27]. However, it is still difficult to narrow down one single mechanism that is essentially required for the mycotrophic life style of *Trichoderma* spp.

MECHANISMS OF SIGNAL TRANSDUCTION IN *TRICHODERMA*

Downstream transduction of signals, generated at the site of receptors, is essential for further expression of genes in the host. Three important signal transduction pathways are identified in *Trichoderma* spp. that enhance the expression of biocontrol and mycoparasitism associated genes. The overexpressed genes belong to the heterotrimeric G-protein signaling, MAPK (mitogen-activated protein kinase) cascades and the cAMP pathways [14]. Most importantly the *T. virens* MAP-kinase TVK1 and its orthologs TmkA in *T. asperellum* and TMK1 in *T. atroviride* [28], are vital in regulating signaling mechanisms for enhanced biocontrol efficiency. Transcript levels of the respective genes also increase in *T. virens* and *T. asperellum* while interacting with plant roots. Detailed studies of the two genes of the heterotrimeric G protein signaling pathway *viz*., the class I (adenylate cyclase inhibiting) G-α subunits TGA1 of *T. atroviride* and TgaA of *T. virens* as well as the class III (adenylate cyclase activating) G-α subunits TGA3 of *T. atroviride* and GNA3 of *T. reesei* have revealed that functions of these two genes are associated with biocontrol. TGA1 was identified vital in the production of antifungal metabolites and regulating coiling around the host hyphae. Loss of function of TGA1 reduces the growth inhibition effect on the fungal host [22]. Similarly, TgaA was shown to have a host specific involvement associated with the MAP-kinases activities whereas TGA3 was found to be important for biocontrol activities as the corresponding gene deletion showed loss of virulence in the strains [29]. Activation of the constitutive GNA3 in *T. reesei* indicates a positive effect on mycoparasitism [30]. The results thus suggest a positive role of MAP-kinases activities and cAMP in biocontrol by *Trichoderma* species [31].

HETEROTRIMERIC G PROTEIN MEDIATED SIGNALING

Mycoparasitism is very essential for a successful interaction between a host and a parasite. In such interactions

host recognition or mycoparasite enzyme-mediated release of host molecules is the initial step. Such signals could be perceived by the release of fungal cell wall degradation products due to the enzymatic action of the mycoparasite either during contact with or while approaching towards the host [16, 32]. The perception of the molecules by the signal transduction pathways is because of the chemical properties of the signaling compounds. The heterotrimeric G proteins play very crucial roles during antagonism of fungal pathogens in *T. atroviride* and *T. virens*. Significant differences were observed among *T. atroviride* and *T. virens* G-protein signaling despite both being closely-related biocontrol agents. In plant pathogenic as well as in saprophytic fungi the α-subunits of G-proteins are involved in different activities such as signal transduction during growth, pathogenicity and production of secondary metabolites. Antagonistic interactions between two fungi provide novel mode of regulation of these conserved signaling elements. When expression of *tga1* was increased in *T. atroviride* by knocking down the G-α subunit homologue by antisense expression it resulted in reduced extension of hyphae. Interestingly, profuse conidiation was observed in the mutant colonies. Recently, it was confirmed that host-mediated signaling and expression of G-α subunit *tga1* in *T. atroviride* is very important for the mycoparasitism on the hyphae of *R. solani*. Loss in the signaling function was also found to impair virulence of pathogens in plant-pathogen interactions [33]. The reduced expression of a MAPK (*bmp1*) and a Gαi protein (*bcg1*) encoding genes have a negative impact on virulence against host plants [34]. The MAPK loss-of function mutants of *Trichoderma* are less effective in parasitism on *R. solani* sclerotia and totally unable to parasitize the *S. rolfsii* sclerotia. It is now confirmed that the *tgaA* mediated pathway is involved in the sclerotial parasitism of *S. rolfsii* but not of *R. solani*. Different functions have been designated to the *T. atroviride* gene *tgaA* and its homologue. However, *T. virens* employs both the MAPK pathway [35] and the G-protein pathway for biocontrol activities in a host-specific manner. The sclerotia of *S. rolfsii* contain high amount of melanin and thereby makes it difficult to penetrate. Therefore, it requires assistance of some specific enzymes for the biocontrol agents to degrade the outer layer and subsequent penetration. A gene for such degrading enzyme could be the downstream target of the *tmkA* and *tgaA* pathways and thereby these two pathways of *T. virens* are considered to be very important for biocontrol [24].

The signaling via heterotrimeric G protein needs basically three components such as a G protein-coupled receptor (GPCR), a heterotrimeric G protein (α, β, γ subunits), and an effector [36]. GCPR proteins have seven common transmembrane domains where the N-terminus reside outside and the C-terminus inside the cytoplasm. When ligands bind to these receptors a conformational change occurs and they release the G-α subunit from the G protein for exchanging GDP with GTP. Subsequently the GTP bound G-α subunit dissociates from their G-ß and -γ subunits and these two signaling units then regulate the downstream activities of the effectors. Receptors are classified into nine groups [37] and class IX comprises of fungal opsins that are quite similar to rhodopsin, the bacterial retinal-binding proteins. Preliminary investigations of the *T. reesei* and *T. atroviride* genomes also revealed that at least 16 putative proteins with 7-transmembrane domains are distributed all over these nine receptor classes. G-proteins were found essential in fungi during sexual multiplication and pathogenic development on a prey. They were also found to be vital in secondary metabolism leading to different developmental as well as morphogenetic processes associated with virulence of mycoparasitic fungi and fungal pathogens of plants. G-protein α subunits are also classified into three major subgroups [38]: subgroup I, subgroup II¸ and subgroup III. Over-expression of subgroup I Gα subunit Tga1 in *T. atroviride* showed its involvement in both coiling and conidiation [22] but *tga1* mutant G-protein α subunit affects processes like vegetative growth, production antifungal metabolites and chitinases [39] that are involved partially in biocontrol by *Trichoderma* spp. The *tga1* mutant was also unable to overgrow and lyse host fungi such as *B. cinerea*, *R. solani*, and *S. sclerotiorum*. However, formation of infection structure was not affected and overgrowing ability is reduced possibly due to growth inhibition of the host fungi at an enhanced level because of its over-production and release of low molecular weight secondary metabolites. Further characterization of mutants of *T. atroviride* bearing a gpr1-silencing construct of a GPCR revealed that Gpr1 is important for growth, conidial production and germination [40].

MAP KINASE MEDIATED SIGNALING

Trichoderma spp. are not only restricted to suppression of pathogens but also are opportunistic plant symbionts and triggers systemic resistance in treated plants [41]. Functional MAPK is also essential in signal perception by plants as well as by *Trichoderma* spp. [42] and the MAPK signaling play important role during *Trichoderma*-plant interaction and induces plant systemic resistance. However, some contrasting reports are also available regarding the role of MAP Kinases in parasitism by *Trichoderma* species. When spore germination of *Trichoderma* took place near the cucumber roots, the *tmkA* mutants also colonized the cucumber roots as effectively as the wild type strains. The fungal MAPKs play important role in several developmental processes such as hyphal growth, sporulation, mating and pathogenicity [43]. Signal transduction in phytopathogenic fungi takes place via MAPK cascades during parasitic interactions [44]. There are three common MAPKs reported in fungi [45]. MAPK pathways are conserved evolutionarily in all eukaryotic organisms and the three kinases, *viz.*, a MAPK, a MAPK activator and a MEK activator (MEK kinase = MEKK or MAPK kinase kinase) are essentially required for functioning of the MAP kinase pathways. In the MAP Kinase encoding gene mutant strains of *Trichoderma*, the expression level of mycoparasitism-related genes (MGRs) increases during mycoparasitism when they come in direct conflict with the plant pathogenic fungus *R. solani*. The disease control was more in the null mutants compared to their wild-type strain or a recommended chemical fungicide. It is also found that in *T. virens* the MAP kinase

Tvk1 serve as negative modulator during the process of host sensing and sporulation. MAPK pathway is involved in transduction of a large variety of signals, amongst which one is associated with pathogenesis. Kss1 from *Saccharomyces cerevisiae*, a homologue of MAPK have been implicated in the expression of cell degrading enzyme both in phytopathogenic as well as non-phytopathogenic fungi. The transcription of a proteinase-encoding gene (*prb1*) is blocked by limiting nitrogen through addition of a MAP kinase inhibitor in two closely related species *T. atroviride* and *T. virens* [46]. The best studied MAPKs of *Trichoderma* are from the yeast family and the fungal extracellular-related kinases (YERK1). The *T. virens* MAPK homologue was described in two different groups as *tmkA* and *tvk1* that have been described encoding the same protein in different *T. virens* strains. Contrasting results about the role of this MAP kinase is that it produced mycoparasitism-related enzymes [47]. Enzyme activities of chitinases and proteases is high in *tvk1* mutants, but *tmkA* mutant strains take time compared to *tvk1* mutants in clearing a chitin containing medium. The protein sequence of *tvk1* shares a high degree of similarity with other MAPKs such as Pmk1 from *M. grisea* [48]. Even the three introns reported in *tvk1* gene are also reported for other fungal MAPK genes. Tvk1 belong to the family of kinases and extracellular-regulated kinases (ERKs) under the umbrella of the MAPK superfamily. The conserved sequence points out its relation with the YERK1 family reported from both yeast and fungi [49]. MGRs regulation in *T. virens* is very complex, however, they share common elements including Tvk1 like other fungi [50]. The *tmk1* in *T. atroviride* play a very crucial role in MAPK signaling during its growth and interaction with other fungi. Deletion of *tmk1* alters the radial growth, conidiation and also spoils the regulation of infection structure formation in the absence of a host-derived signal. In other words, presence of host sensing mechanism will involve a number of signal transduction pathways in *Trichoderma* spp. that leads to successful expression of MGRs. Molecules either released from the fungal host or located on its surface (e.g. lectins) induce both production of enzyme and formation of infection structure. Production of antimicrobial metabolites such as 6-pentyl-a-pyrone is enhanced in *Trichoderma* in the presence of a host such as *R. solani* [51]. Characterization of MAPK genes in the rice pathogen *M. grisea* revealed that some of them such as pmk1 and mps1 are essential for pathogenesis including formation of appressoria, colonization on host tissue, and penetration of the host cuticle [52].

CAMP MEDIATED SIGNALING

cAMP signaling is an important pathway in fungi in controlling of differentiation, sexual development, virulence, monitoring of the nutritional status, stress, transcription and cell cycle progression [53]. The cascade of cAMP signaling is regulated by a membrane-associated adenylate cyclase. Adenylate cyclase synthesizes the intracellular messenger cAMP, which is regulated by α-subunits of heterotrimeric G-proteins in most fungi. The cAMP works in eukaryotic organism through the activation of cAMP-dependent protein kinases (PKA) and it consists of two regulatory and two catalytic subunits [54]. cAMP act as a positive effector of endoglucanase induction in *T. reesei*, a species which is able to antagonize and overgrow on *P. ultimum* and provide in planta protection of zucchini plants against *P. ultimum* blight [55]. The exogenous cAMP increased coiling around nylon fibers in the biomimetic system in *T. harzianum* and substances (e.g. dinitrophenol, caffeine, aluminium tetra fluoride) which increased the intracellular levels of cAMP and repressed the synthesis of N-acetyl-β-D- glucosam-inidase [56]. cAMP signaling plays important role during the conidiation in *T. viride* and *T. atroviride* [57]. The mechanism of *Trichoderma* to survive and disperse in the environment is production of conidia which is induced by environmental factors such as blue light and nutrient stresses in these mycoparasites. In *T. viride*, the photoinduction increases the intracellular level of cAMP due to which conidiation occur [58] and the use of exogenous cAMP stimulate the formation of conidia in both colonies that were kept in the dark and light as well [59]. PKA plays a crucial role in regulation of light responses in this fungus [57]. Deletion of tac1, an adenylate-cyclase-encoding gene, brought intracellular cAMP levels below the detection limit and the mutants showed only 5-6 % of the wild-type growth rate. In mutants, sporulation did not occur in darkness, germination of spores failed in water, did not overgrow the test plant pathogens *Pythium* sp., *R. solani* and *S. rolfsii* and showed reduced secondary metabolite production. It was concluded that cAMP signaling in *T. virens* positively regulates secondary metabolism and Tac1 is the first regulatory protein involved in growth, germination, mycoparasitism and secondary metabolism [60].

INTERACTIONS BETWEEN *TRICHODERMA* AND MYCORRHIZAL FUNGI

Arbuscular mycorrhizal fungi (AMF) and biocontrol agent *Trichoderma* both are helpful in promoting plant growth and improving health when they colonize roots. Fungal biocontrol agents do not cause hindrance on AMF establishment rather facilitates them in a synergistic manner [61]. It is also evident that *T. harzianum* induces the symbiotic association of AMF with vascular plants. A recent study conducted by Al-Asbahi [62] showed that volatile biomolecules released by *T. harzianum* Rifai KRL-AG2 indirectly enhanced association by AM fungi with wheat plant roots (cv. Avocet S) and the wheat protein homologous to arbuscular mycorrhizal protein was over-expressed which might have a role to play in the AM–plant interaction. The phytohormone levels particularly auxin and gibberllin were accumulated significantly high in soybean plants when treated with *T. harzianum* and *Glomus mosseae* (an AMF) in combination compared to untreated plants and plants treated with *T. harzianum* and *G. mosseae* individually. The observations suggest that there was a synergistic interaction between *T. harzianum* and *G. mosseae* that resulted in better plant growth [63]. Saprophytic and nonpathogenic strains of *Trichoderma* reduce the fungal diseases in a variety of plants. Natural inhabitant saprophytic fungi has both positive and negative effect on spore germination, growth,

development and function of AMF e.g. *T. harzianum* have different types of effects such as antagonistic, stimulating and neutral effect on AMF [64]. It has been reported earlier that consortia of *T. harzianum* and *G. intraradices* is more effective in lowering the disease severity of Fusarium crown and root rot of tomato and Fusarium wilt of Giotto melon (*Cucumis melo* L.) caused by *Fusarium oxysporum* than individually. A number of strains of *T. pseudokoningii* were found to inhibit germination of AMF e.g. *G. mosseae* and *Gigaspora rosea*. Different soluble and volatile substances secreted by *T. pseudokoningii* were attributed to play crucial roles in inhibition of AMF spore germination. While inhibition of spore germination of *Gi. rosea* is attributed to soluble substances secreted by *T. pseudokoningii*, volatile substances can be attributed to inhibition of spore germination in *G. mosseae*. Different strains of *T. pseudokoningii* were observed to reduce root colonization percentage by *Gi. rosea* when treated in soybean culminated in reduced accumulation of shoot dry matter. However, no evidence was generated whether AMF had any adverse effect on propagules of *Trichoderma* species in most of the instances [64]. This is an interesting area to work further to understand the molecular mechanisms of synergistic and antagonistic interactions between *Trichoderma* and AMF which is currently very poorly understood [65].

CONCLUSION

Fungi may interact with each other in a variety of ways. Interactions between two fungal species may be of antagonistic, parasitic and synergistic types. An interaction is normally mediated through cross talk between biomolecules released/secreted from one fungal species that acts as messenger and perceived by receptors present on the other interacting fungal species. A signal is generated at the perception site and further downstream actions follow through a signal transduction mechanism. Downstream transduction of signals further helps in expression of a set of genes that governs the type of interaction that is going to have between the two fungal species. A number of mechanisms had been revealed that are involved in a variety of interactions between the fungal species. More specifically, the interactions between *Trichoderma* species with other soil fungi has been studied deeper compared to other types of fungal interactions. However, a lot more is needed to be understood about the mechanisms between fungus-fungus interactions particularly mode of interactions between *Trichoderma* species and AMF as both are essential for promoting plant health and reducing diseases in agricultural crops.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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