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Fungicide resistance in plant pathogens: An emerging threat in Plant Disease Management

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Introduction

Agricultural productivity is greatly hampered by various plant diseases incited by virulent plant pathogens. Broad concepts like integrated disease management strategies have come up for rescue which includes host plant resistance, biological control, surveillance, quarantine, Good Agricultural Practices (GAPs), etc. Among various methods, Chemical control with fungicides still remains focused & quick relievers against a large group of plant pathogens. Fungicides holds a large share in agrochemical market in India. Besides other causes like insufficient dose, faulty application and heavy disease pressure, development of resistance can be one of the causes of poor disease management. Resistance to fungicides has become a challenging problem in the management of crop diseases .

Historical glimpse of resistance development

Practical problems of resistance development to pathogens emerged with introduction & widespread use of first generation site specific systemic fungicides groups like benzimidazoles, dicarboximides. Initially it was observed in *Penicillium* sp. causing citrus storage rots due to aromatic hydrocarbons and laterly in benzimidazoles and dicarboximides in many pathogens.

Fungicide resistance and its Mechanisms

It is referred to as genetic adjustment by a fungus that results in reduced sensitivity to a fungicide. It is result of genetic mutations occurring at low frequencies & subsequently selection pressure acting on resistant propagules found in natural populations. It is mostly established that alteration in biochemical target sites due to mutation leads to resistance which is seen among benzimidazoles, phenylamides , DMIs and QoIs (Brent, 1995). Certain site specific fungicide groups like benzimidazoles, phenylamides creates single gene mutation which is carried by sudden shift called as disruptive selection among wide differing

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responsive populations leading to resistance problems termed as Qualitative resistance. Another case, mutation in multiple genes favour resistant population development which under progressive selection called as directional selection encounters sub-lethal doses of fungicides over a time and becomes resistant termed as Quantitative resistance. Other mechanisms includes cross resistance and multiple resistance. Site specific fungicides groups like benzimidazoles, dicarboximides, phenylamides and strobilurins possess target site activity with different mode of action leading to high resistance risk.

Case Studies

Benzimidazoles

Benzimidazoles traded under name benomyl, carbendazim and thiophanates possess site specific mode of action and systemic movement in plants and showed interference with mitosis (Davidse, 1973) causing dislocation of spitzenkorper and disorganization of fine structure of fungal hyphae (Borck, 1973 and Howard, 1980). At Molecular level, Point mutations in beta- tubulin gene alters amino acid sequences at binding sites which reduces protein complex was confirmed by site directed mutagenesis followed by gene replacement (li *et al*., 1996). Mutations observed at codon 6 & 198 leads to low and high resistance level in *Monilinia fructicola* (Ma *et al*., 2005) and at codon 198 & 200 leads to medium and high resistance level in *Venturia inaequalis* (Koeraadt, 1992). Keinath *et al*. (1998) found resistance development in *D. bryoniae* isolates to benomyl with cross resistance relationships to thiophanate methyl. Khilare *et al*. (2010) found high number of resistant isolates in carbendazim compared to difenconazole and propiconazole due to high selection pressure. Khilare *et al*. (2004) observed increase in total sugars, amino acids, proteins, DNA, RNA contrast to decrease in orthodihydric phenols and total phenols due to infection of resistant isolates in different infected parts of grapes plant.

Dicarboximides

Traded under the name iprodione, vinclozolin act as contact fungicides and moves in systemic manner in plants. it mainly induce membrane lipid peroxidation in fungi by interfering with flavin containing enzymes (Edlich *et al*., 1988) and also causes hyphal swelling and bursting of tips (Eichhorn, 1978) but no effect on ion & water permeability (Yoshinaga, 1993) & negligible effect on respiration, sterol synthesis, nucleic acids were observed. At Molecular level, *adr-1* gene isolated from cosmid library fragment of *U. maydis*

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was confirmed to confer resistance (Orth, 1995). Dry *et al*. (2004) compared iprodione susceptible and resistant isolates of *A. alternate* & suggested premature termination of open reading frame to be cause of resistance. Hubbard *et al*. (1997) observed gradual development of resistance in *S. minor* isolates against iprodione with cross resistance to vinclozolin. Myresiotis *et al*. (2007) observed resistance development in *B. cinerea* against iprodione with strong cross resistance relationships between dicarboximides and anilinopyrimidines.

Phenylamides

Traded under name: Metalaxyl, Furalaxyl etc. Phenyl amides fungicides possess site specific multistep resistance effects which attacks RNA polymerase I & inhibits rRNA synthesis (Davidse, 1995). Thind *et al*. (2010) observed normal pathogenic potential among metalaxyl populations and strong competitive fitness with sensitive populations when inoculated in mixture. Kaur *et al*. (2010) revealed prevalence of metalaxyl resistant to vary levels with high pathogen city with nil cross resistance to novel action fungicides.

Demethylation inhibitors(DMIs)

DMIs also called as sterol biosynthesis inhibitors (SBIs), inhibits sterol C-14 a demethylation of 24 methylene dihydrolanosterol, a precursor of ergosterol in fungi (Brent, 1995). At Molecular level, Delye *et al*. (1997) confirmed single gene mutation in *CYP51* gene of *Uncinula necator* responsible for resistance. Overexpression of *CYP51* gene was also confirmed for resistance (Marichal *et al*., 1997). Hamamoto *et al*. (2000) confirmed 126 bp repeats in *CYP51* gene for resistance development. Stevic *et al*. (2010) showed high sensitivity of *V. inaequalis* to both flusilazoles, Difenconazole. Wong *et al*. (2007) established single discriminatory dose for differentiating sensitive and resistant isolates of *C. cereale*.

Strobilurins

Traded under name : azoxystrobin, Kresoxim- methyl, trifloxystrobin etc. They possess site specific action and inhibits electron transport in mitochondria respiration (Bartlett *et al*., 2002). At Molecular level, Fisher *et al*. (2004) confirmed point mutation at cy b gene changes phenylalanine to leucine which alters enzyme activity by site directed mutagenesis. Vincelli *et al*. (2002) found emergence of QoI resistant biotypes of perennial

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ryegrass infecting strains of *P. grisea.* Ishii *et al*. (2001) showed distribution of pathogen isolates highly resistant to kresoxim-methyl.

Fungicide resistant management

Main principles emphasizes on delaying of resistance development and to keep its level under control. Fungicide resistance management focuses on various strategies as follows: Avoiding sole use of "at risk" fungicides , integration with cultural practices to reduce selection pressure in pathogens (Damicone,1999), Reduction in number of applications, use of new fungicides with novel sites and exploiting negative cross resistance to eliminate other phytopathogens (Hewitt, 1998).

Conclusions

- Development of fungicide resistance in pathogen leads to failure of disease management strategies.
- Single Point mutation and Selection pressure play an important role in resistance development.
- Site specific fungicides like Benzimidazoles, Dicarboximides, Phenylamides and strobilurins characterized at high resistance risk due to target site activity.
- Resistant management strategies emphasizes on prophylactic measures aiming delaying resistant development and approaching for integrated management strategies.

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