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EPIDEMIOLOGY AND MANAGEMENT STRATEGIES OF *Botrytis tulipae* (BOTRYTIS BLIGHT) OF TULIP: A COMPREHENSIVE REVIEW

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ABSTRACT

Botrytis blight or 'tulip fire', caused by *Botrytis tulipae* (Lib.) Lind., is the most economically devastating fungal disease of tulip (*Tulipa spp*.) worldwide, resulting in annual yield losses of 15–80% under conducive environmental conditions. This review comprehensively examines the taxonomy, morphology, epidemiology, disease cycle, host range and management strategies of *B. tulipae*. The pathogen, a necrotrophic ascomycete belonging to the family Sclerotiniaceae, infects all aerial plant parts and persists in soil as melanized sclerotia. Disease development is strongly correlated with cool temperatures (15–22°C), high relative humidity (>85%) and prolonged leaf wetness periods. Global distribution surveys reveal significant incidence in major tulip-producing countries including the Netherlands, Turkey, Japan, India (Jammu and Kashmir), China and the USA. Chemical management using fungicides such as captan (phthalimide; FRAC M4), carbendazim (benzimidazole; FRAC 1) and myclobutanil (triazole; FRAC 3) has formed the cornerstone of disease control. However, documented resistance particularly to carbendazim and dicarboximides has necessitated resistance management protocols and the exploration of alternative strategies. Biological control using *Trichoderma harzianum, T. viride, T. asperellum*, *Bacillus subtilis* and *Gliocladium catenulatum* has demonstrated significant antagonistic activity against *B. tulipae,* with efficacy of 48–80%. Farmyard manure (FYM) application as a soil amendment enhances microbial diversity and suppressive capacity. Integrated disease management (IDM) combining chemical, biological and cultural practices achieves the highest efficacy (82–92%), reduces chemical residues and minimizes resistance risk. This review underscores the need for coordinated research on molecular mechanisms of resistance, genomics-based biocontrol agent selection and sustainable IDM frameworks for tulip production systems.

Keywords **:** Biocontrol, *Botrytis tulipae*, epidemiology, captan, carbendazim, myclobutanil, Trichoderma, FYM, integrated disease management, tulip fire.

Introduction

Tulipa spp. (Family: Liliaceae), commonly referred to as Tulip, is one of the most economically important ornamental bulbous crops cultivated worldwide for their economic and aesthetic value. Tulip cultivation covers more than 11,000 ha in the Netherlands alone, while important areas for its cultivation are found in Turkey, Japan, India, China,

France and the USA (van Bruggen, 1995; Ahmad *et al*., 2010). The global market for Tulip bulbs is worth more than USD 500 million annually, signifying their paramount importance in the world of floriculture.

Among all the important biotic limiting factors for Tulip cultivation worldwide, Botrytis blight commonly referred to as 'Tulip Fire,' caused by *Botrytis tulipae* (Lib.) Lind. (teleomorph: *Botryotinia tulipae*), is recognized globally as the most destructive disease of Tulip (Coley-Smith *et al*., 1980; Williamson *et al*., 2007). This disease was first reported on Tulip leaves in Belgium by Libert (1837). Botrytis blight has now been reported from virtually all areas of Tulip cultivation worldwide (Jarvis, 1977; Elad *et al*., 2016). The disease severity varies between 15% and 80% crop loss depending upon environmental conditions and cultivar susceptibility (Hausbeck and Moorman, 1996; Kim *et al*., 2007).

The pathogen is a highly specialized and host-specific *Botrytis* species, although the range of its pathogenicity might include other members of the Liliaceae family. Unlike the more generalist *B. cinerea, B. tulipae* is characterized by its narrow host specificity, mainly infecting *Tulipa* species and occasionally other ornamental bulbous crops (Jarvis and Booth, 1980; Williamson *et al*., 2007). The sclerotia of the pathogen survive the winter in the soil or on infected plant debris, which act as the inoculum source for the next growing season (Coley-Smith, 1990; Wang *et al*., 2026). While tulip bulb rot can be caused by various pathogens, there's a lack of research on how these pathogens interact with each other (Nisa *et al.,* 2024). The flowerless tulips reach senescence in summer and daughter bulbs are harvested shortly thereafter (Karlsson *et al.,* 2024; Salybekova *et al.,* 2025)

Chemical fungicides such as captan, carbendazim and myclobutanil have traditionally been used in the control of the disease. Nevertheless, the extensive and frequent application of one-site-action fungicides such as carbendazim (FRAC Group 1 fungicides) have resulted in the development of resistant strains in many countries (Brent and Hollomon, 2007; Leroux *et al*., 2010; Weber, 2011). The occurrence of the disease problem has thus fueled the need for the application of biological control agents such as *Trichoderma spp*., *Bacillus subtilis*, *Gliocladium catenulatum*, as well as organic amendments such as FYM (Elad and Stewart, 2004; Vinale *et al*., 2008; Bonanomi *et al*., 2010; Celar and Valic, 2005).

The objectives of this review are to synthesize existing information on the following aspects of *B. tulipae:* (i) taxonomy and morphology; (ii) epidemiology and disease cycle; (iii) geographical distribution and economic importance; (iv) chemical control including captan, carbendazim and myclobutanil; (v) biological control agents with special emphasis on *Trichoderma* and FYM; and (vi) IDM. Critical analysis of existing literature, supported by tables and figures, is made to outline future research and policy guidelines.

Taxonomy, Morphology and Biology

Taxonomic Classification

The *Botrytis tulipae* (Lib.) Lind. species is a member of the Kingdom Fungi, Phylum Ascomycota, Class Leotiomycetes, Order Helotiales and Family Sclerotiniaceae (Crous *et al*., 2011; Van der Vlugt-Bergmans *et al*., 1993). *Botryotinia tulipae* (Lind.) Hennebert is the teleomorph form of *Botrytis tulipae*, which is seldom found in nature and requires special conditions for apothecium development. It was first reported as *Botrytis cinerea* var. *tulipae* by Libert in 1837 and was later promoted to species status by Lind in 1913. Molecular phylogenetic studies have shown significant differences in the ITS, rpb2 and G3PDH gene sequences of *Botrytis tulipae* from.

Morphological Characteristics

Macroscopically, *B. tulipae* has profuse gray to brownish-gray conidia in botryose clusters on branched conidiophores. Conidiophores are erect, septate, 1-3mm in height, brown and bear branches near the apex with terminal swellings. Conidia are ellipsoidal to obovate, smooth-walled, hyaline to pale brown, 14-22 x 10-14 µm in size, which are larger than those of *B. cinerea* (9-15 x 6-10 µm). (Jarvis, 1977; Williamson *et al*., 2007)

Sclerotia are melanized, irregular to subglobose, 1-8 mm in diameter and develop on infected host tissues and in culture on PDA. They function as survival structures for the fungus, which enables it to persist in soil for several years. (Coley-Smith, 1990; Braun and Petzold, 1989)

Germination of the sclerotium occurs at 5-15°C and is stimulated by the presence of host exudates, soil moisture and UV light. (Coley-Smith *et al*., 1980).

Cultural Characteristics

On potato dextrose agar (PDA), *B. tulipae* forms cottony gray to brownish-gray colonies at 15-22°C. Optimal temperature for mycelial growth is 15-20°C. No growth occurs at 30°C. Optimal pH for growth is 5.5-7.0. Abundant sporulation occurs under alternate light/dark (12/12 hours). Sclerotia develop abundantly

after 2-3 weeks. The cultural characteristics of *B. tulipae* differentiate it from *B. cinerea.* The cultural traits of *B. tulipae* can be utilized as a differential diagnostic criterion (Jarvis, 1977; Coley-Smith *et al*., 1980).

Epidemiology

Disease Symptoms and Progression

Botrytis blight displays a range of symptoms depending on the growth stage of the affected plant, environmental factors and inoculum density. Symptoms begin as small, brown, water-soaked spots on young leaves and shoots on infected bulbs. These spots grow rapidly and merge to form the characteristic 'bleached and scorched or fire effect,' which gives the pathogen the common name (Hausbeck and Moorman, 1996; Elad *et al*., 2016).

In more advanced infections, the stems become weakened and collapse, a symptom known as 'neck rot.' Infected flowers develop 'blast,' where buds fail to open or develop 'petal blight,' where the petals develop spots and gray spores. Epidemic infections on a larger scale result in the characteristic 'burnt appearance' with gray spores evident on all the above-ground plant tissues. Post-harvest bulb infections develop as 'dry rot' with the formation of sclerotia on the outer bulb scales (Hunter *et al*., 1968; Amsing and Rattink, 1992).

Table 1 : Symptoms of *Botrytis tulipae* blight across different growth stages of tulip and associated disease severity under field conditions.

Growth Stage	Plant Part Affected	Symptoms Observed	Disease Severity (%)
Emergence	Seedling/sprout	Damping-off, brown lesions at base	10–30
Vegetative	Leaves, petioles	Water-soaked spots, grayish mold	20–45
Pre-bloom	Floral buds, stem	Bud blasting, neck rot, gray sporulation	30–70
Bloom	Petals, tepals	Petal blight, fire symptoms, blast	40–80
Post-harvest	Bulb, scale leaves	Neck rot, dry rot, sclerotia on scales	15–60
Storage	Bulb surface	Sclerotia, gray mold colonies	5–35

Environmental Factors Influencing Disease Development

Environmental factors are extremely important for the initiation, development and spread of *B. tulipae.* Low temperature (15–22°C), high relative humidity (>85%), long periods of leaf wetness (>8 h) and overcast weather are all important for disease triangle development (Jarvis, 1977; Elad *et al*., 2016). Spore germination occurs optimally at 15°C with free moisture, while penetration occurs within 2–4 h through direct cuticular penetration or wounds and stomata (Shirane *et al*., 1989). The main method for secondary spread within and between tulip fields is rain splash and wind for conidia. The latent period for optimal temperature (15–18°C) is 3–5 days. This allows for quick epidemic development during cool, moist spring weather. On the other hand, dry and sunny weather above 25°C slows down disease development considerably (Williamson *et al*., 2007; Hausbeck and Moorman, 1996).

Table 2 : Environmental factors influencing *Botrytis tulipae* development and the critical thresholds that favor epidemic conditions.

Environmental Factor	Optimal Range	Critical Threshold	Disease Favored	Reference
Temperature (°C)	15–22	>25 or <5	15–20	Elad *et al*. (2016)
Relative Humidity (%)	90–100	>85	>90	Williamson *et al*. (2007)
Leaf Wetness (hrs)	8–16	>6	>8	Jarvis (1977)
Spore Germination Temp.	10–25°C	Optimum 15°C	10–20°C	Coley-Smith *et al*. (1980)
Light (photoperiod)	Low/diffuse	Overcast conditions	Overcast + wet	Hausbeck and Moorman (1996)
Soil pH	5.5–7.0	<5.5	Acidic soils	Gerlagh *et al*. (1993)

Disease Cycle and Inoculum Sources

The disease development process of *B. tulipae* is characterized by the presence of primary infection and secondary infection phases. The primary infection is caused by the following: (i) infected planting bulbs containing latent mycelium or conidia; (ii) sclerotia in the soil originating from previous infected crops; and (iii) infected plant debris containing mycelium and sclerotia (Coley-Smith, 1990; Grinstein and Kritzman, 1990). The secondary infection is caused by wind-borne or rain-splashed conidia from sporulating lesions of infected plants in the same growing season.

Sclerotia can persist in the soil for 3-5 years as the primary inoculum between growing seasons. The germination of sclerotia is regulated by the soil temperature, moisture content and the availability of

host root exudates that induce the synthesis of sclerotiogenic compounds (Coley-Smith *et al*., 1980; Gerlagh *et al*., 1999). The persistence of the primary inoculum in the soil is the greatest challenge in the management of the disease.

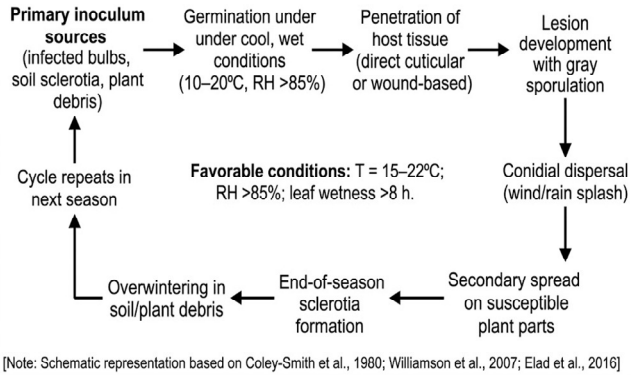

Fig. 1 : Conceptual disease cycle of *Botrytis tulupae* on Tulip

Global Distribution and Economic Importance

Botrytis tulipae has been recorded in all the main areas of the world where tulip production occurs. The Netherlands, which accounts for the greatest share of the world's production of tulips with 11,000 ha of crop under cultivation, suffers economic losses of 15-45% in infected cultivars every year. The economic losses in the Netherlands occur especially in wet spring seasons. Disease incidence has been recorded to be between 20-50% in infected areas in Turkey. The incidence of the disease in India has been severe in the Kashmir valley. The economic losses due to the disease in India were recorded to be as high as 60% in the most favorable conditions. China has recently emerged as an important producer of tulip bulbs with 2,000 ha of crop under cultivation. The economic losses due to the disease in China were recorded to be between 18-50%. Disease incidence in Japan was recorded to be between 10-35%. The United States has recorded economic losses due to the disease in the range of 12-30%. The economic losses due to the disease in the world as a whole are estimated to be more than USD 50-100 million (Magan and Aldred 2009; Marois and Dunn 1985).

Table 6 : Global distribution of *Botrytis tulipae*, tulip cultivation area and reported disease incidence in major producing countries.

Country/Region	Tulip Area (ha)	Disease Incidence (%)	Major Season	Reference
Netherlands	~11,000	15–45	Feb–May	van Bruggen (1995)
Turkey	~3,500	20–50	Mar–May	Delen (2001)
India (JandK)	~500	25–60	Mar–Apr	Ahmad *et al*. (2010)
Japan	~800	10–35	Mar–May	Niimi *et al*. (1994)
USA	~1,200	12–30	Apr–May	Dreistadt (1994)
France	~600	15–40	Mar–May	Gullino *et al*. (2010)
China	~2,000	18–50	Mar–Apr	Zhao *et al*. (2012)

Host Range and Pathogenicity

Although the pathogen is closely associated with Tulipa species, it is also found in other Liliaceae species such as *Fritillaria* spp., *Erythronium* spp. and occasionally *Lilium* spp., under artificial inoculation conditions (Jarvis and Booth, 1980; Williamson *et al*., 2007). The host specificity of *B. tulipae* is significantly higher compared with *B. cinerea*. The reason is the specific adaptations in the effector apparatus and enzyme synthesis pattern of *B. tulipae* that are closely linked with the chemical characteristics of tulip tissue, especially the alkaloids and glucosides present in *Tulipa spp*.

The mechanisms of pathogenicity in *B. tulipae* involve the synthesis of non-specific phytotoxins such as botrydial and botcinolide; cell wall-degrading enzymes such as polygalacturonases, cellulases and xylanases; and reactive oxygen species (ROS), which contribute to maceration of the tissue and nutrient uptake from the necrotized tissue (Prins *et al*., 2000; Tudzynski and Kokkelink, 2009; Wubben *et al*., 1994). The synthesis of oxalic acid is associated with the manipulation of pH in the infection zone, which in turn enhances the activity of cell wall-degrading enzymes.

Chemical Management Strategies

Overview of Fungicide use in Tulip

Chemical fungicides still dominate the control methods used against *B. tulipae,* especially in the cultivation of tulips on a large scale where the value of the crop requires consistency in disease management. Fungicide management of *B. tulipae* is usually done through bulb treatment prior to planting, in-season foliage sprays at 7-14 day intervals during periods of disease risk and bulb dips or dusts after harvest (Amsing, 1992; Gullino *et al*., 2010). The choice of fungicides is based on the guidelines provided by the FRAC (Fungicide Resistance Action Committee),

which recommend the rotation of different modes of action in fungicide mixtures in the management of *B. tulipae* in order to delay the onset of.

Table 3 : Major fungicides registered or used against *Botrytis tulipae*, their chemical groups, modes of action, application rates, efficacy, resistance risk and FRAC classification.

Fungicide	Chemical Group	Mode of Action	Application Rate	Efficacy (%)	Resistance Risk	FRAC Code
Captan	Phthalimide	Multi-site SH inhibitor	2–3 kg/ha	65–80	Low	M4
Carbendazim	Benzimidazole	β-tubulin inhibitor	0.5–1 kg/ha	60–75	High	1
Myclobutanil	Triazole (DMI)	Ergosterol biosynthesis	0.3–0.5 L/ha	70–85	Medium	3
Iprodione	Dicarboximide	MAP/Osmotic signal inhibition	1–1.5 kg/ha	75–88	Medium-High	2
Fenhexamid	Hydroxyanilide	Sterol biosynthesis (C4-demethyl)	0.75–1 kg/ha	80–92	Medium	17
Chlorothalonil	Chloronitrile	Multi-site SH group inhibitor	1.5–2 kg/ha	65–78	Low	M5
Boscalid	SDHI	Succinate dehydrogenase inhibition	0.5 kg/ha	82–90	Medium	7

Captan

Captan [N-(trichloromethylthio) cyclohex-4-ene-1,2-dicarboximide] is a multi-site contact fungicide classified as a phthalimide (FRAC Code M4). The wide spectrum of activity is due to the thiol-reactive trichloromethylthio group, which non-specifically reacts with sulfhydryl groups of enzymes and coenzymes critical for fungal metabolism, such as those required for respiration and amino acid synthesis (Brent and Hollomon, 2007). The multi-site mode of action ensures that there is negligible risk of resistance development.

Captan has been an essential tool for tulip disease management for decades, with applications as foliar sprays at 2-3 kg/ha (80% WP formulation) at 10-14 day intervals and as a bulb dip at 0.2% concentration prior to planting. Research has shown 65-80% control efficacy against *B. tulipae* at recommended rates (Amsing, 1992; Gullino *et al*., 2010). Captan is often tank-mixed with systemic fungicides to offer both protective and curative activity, as well as to minimize selection for resistance to the systemic component. The compatibility of captan with biocontrol products such as *Trichoderma,* due to its short residual activity, makes it an essential component in integrated disease management programs.

Carbendazim

Carbendazim [methyl benzimidazol-2-ylcarbamate] is a systemic benzimidazole fungicide (FRAC Code Number 1), which targets the β-tubulin polymerization process in fungi, thus affecting their mitosis and cell division process. It was widely used in the control of *B. tulipae* in the 1970s-1990s because of its systemic activity, curative activity and effectiveness in foliar or soil application (Elad *et al*., 1992; Leroux *et al*., 2002).

However, the fungicide carbendazim is associated with high risk of resistance (FRAC Group 1 = High Risk). Mutations at the single amino acid change at codon 198 (Glu → Ala/Lys) and at codon 200 (Phe → Tyr) in the β-tubulin gene can confer high-level resistance. Resistant *B. tulipae* and *B. cinerea* populations were found in the Netherlands, Germany, Turkey and Japan within 3-5 years of repeated fungicide application (Elad *et al*., 1992; Kim *et al*., 2007; Weber, 2011). The fungicide is regulated in the European Union because of its reproductive toxicity; however, it is still effective at 0.5-1 kg/ha (50% WP) in controlling *B. tulipae* sensitive populations.

Myclobutanil

Myclobutanil, or α-butyl-α-(4-chlorophenyl)-1H-1,2,4-triazole-1-propanenitrile, is a triazole class sterol demethylation inhibitor (DMI) fungicide with FRAC Code 3. Myclobutanil inhibits the enzyme lanosterol 14α-demethylase (CYP51) in fungi, which is a critical step in the synthesis of the fungal cell membrane component ergosterol. This inhibition results in the depletion of ergosterol and the buildup of toxic methylated sterols in the fungal cell membrane (Brent and Hollomon, 2007).

Myclobutanil has excellent curative and protectant activity against *B. tulipae* when applied at a rate of 0.3 to 0.5 L/ha (20% EC formulation). Myclobutanil achieved 70-85% control in field experiments (Kim *et al*., 2007; Yildirim *et al*., 2016). Myclobutanil's systemic activity and long-lasting effect (14-21 days) reduce the application rate. DMI fungicides have a medium resistance risk. Resistance to DMI fungicides usually involves mutations in the target enzyme lanosterol 14α-demethylase (CYP51), overexpression of efflux transporters, or alterations in the promoter region. Monitoring the change in sensitivity is recommended. Rotation with other fungicides such as multi-site inhibitors (captan and chlorothalonil) will

maintain the effectiveness of the DMI fungicide. Comparison with other fungicides has shown myclobutanil to have better activity than carbendazim.

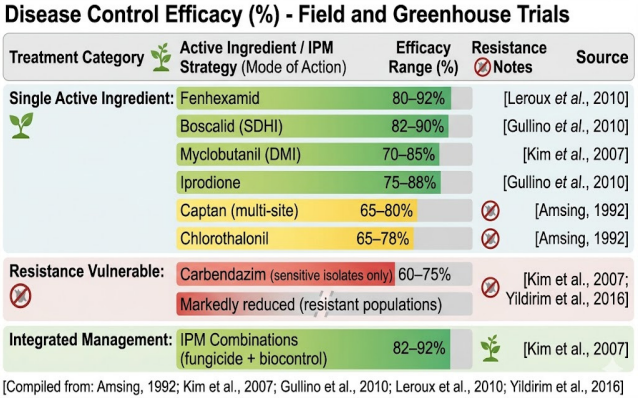

Fig. 2 : Comparative efficacy of fungicides against *Botrytis tulipae*

Fungicide Resistance: Mechanisms and Management

Resistance to Benzimidazoles (Carbendazim)

Resistance to benzimidazoles in *B. tulipae* and other *Botrytis* spp. has a classical single-step mechanism with point mutations in the β-tubulin gene. In two cases, the E198A and F200Y mutations cause high-level resistance (MIC > 100 µg/mL), while the E198K mutation causes intermediate-level resistance. Fitness costs of resistance are low, allowing for the persistence of resistant strains in the absence of selection pressure (Elad *et al*., 1992; Myresiotis *et al*., 2007; Latorre *et al*., 2001).

Multiple Fungicide Resistance

The occurrence of multiple fungicide resistance (MFR), where individual isolates show resistance to two or more fungicide chemical classes, is being reported in B. cinerea and in *B. tulipae* in the areas where tulips are grown commercially (Elad *et al*., 1992; Leroux *et al*., 2010; Partridge and Sutton 1996; Vanhook and pell, 1994). The mechanisms of MFR involve benzimidazole resistance (mutation in the β-tubulin gene), dicarboximide resistance (modification of the MAP kinase pathway) and DMI resistance (overexpression or mutation of the CYP51 enzyme). Drug efflux transporters (MDR phenotype), which involve ABC and MFS types of transporters, confer multiple-drug resistance to phenylpyrroles and anilinopyrimidines (Leroux *et al*., 2002; Weber, 2011).

Resistance Management Strategies

For effective fungicide resistance management in *B. tulipae*, the following approaches have been recommended: (i) rational rotation of FRAC groups with different modes of action, (ii) integration of multi-site contact activity fungicides such as captan and chlorothalonil (FRAC M-codes), which preclude the development of target site resistance, (iii) mixture formulations using single and multi-site activity fungicides, (iv) regular pathogen sensitivity monitoring using EC50 bioassays or molecular methods to identify resistance alleles and (v) integration of biological control methods to limit the total fungicide usage (Brent and Hollomon, 2007; Fillinger and Elad, 2016; De Cal *et al*., 2004; Erwin, and Ribeiro 1994). Rotation between the multi-site activity fungicide captan (FRAC M4) and the DMI activity fungicide myclobutanil (FRAC 3) has been a practical approach to manage benzimidazole-resistant and DMI-sensitive *B. tulipae* populations.

Biological Control

Trichoderma Species

Among all the biocontrol fungi, *Trichoderma* is the most extensively studied genus. Several modes of action are responsible for the antagonistic activity of *Trichoderma* against *B. tulipae*. The modes of action include: (i) mycoparasitism, which involves coiling of *Trichoderma* hyphae around the host pathogen hyphae followed by excretion of cell wall-degrading enzymes such as chitinases, glucanases and proteases; (ii) antibiosis, which involves excretion of volatile as well as non-volatile substances such as harzianic acid, harzianolide and trichodermin (Baert *et al*., 2007; Thrane *et al*., 2001); (iii) competition for nutrients and colonization sites; and (iv) induction of systemic resistance (ISR) in the host plant.

Trichoderma harzianum

Among all species of *Trichoderma*, *T. harzianum* is the most used for biocontrol purposes. Under greenhouse and field conditions, *T. harzianum* strain T39 (Trichodex) has shown 55 to 78% control efficiency for *B. tulipae* on tulip and other ornamental plants (Elad *et al*., 1993; De Meyer *et al*., 1998). Its wide range of mechanisms for controlling *Botrytis* diseases, such as mycoparasitism, production of lytic enzymes such as chitinases and β-1,3-glucanases, as well as triggering plant defense mechanisms mediated by jasmonic acid and salicylic acid pathways, make it effective under varying environmental conditions. Soil treatment in granular formulation (107 CFU/g) or bulbs treated with 108 CFU/mL liquid dip formulation have shown significant effects on reducing viability of sclerotia and primary inoculum levels (Zimand *et al*., 1996; Elad and Stewart, 2004; Delen. 2001; Heale and Sharaman 1977).

Trichoderma viride and T. asperellum

Trichoderma viride and *T. asperellum* strains have also been observed to show significant antagonistic activity against *B. tulipae* in laboratory and field bioassays with efficacy of 50-72% and 48-68%, respectively. *T. viride* has the capacity to produce trichoviridin and other antibiotics that inhibit the germination of spores and the growth of mycelia of *Botrytis*. *T. asperellum* has the capacity to colonize the rhizosphere and induce systemic resistance. These strains of *Trichoderma* may be used in combination with strains having dominant mechanisms of action in order to get additive or synergistic effects, especially in changing environments (Kramer and Petzold 1983; Oka *et al*., 2000).

Bacillus subtilis

Bacillus subtilis and other endospore-forming species are known to be effective biocontrol agents against fungal diseases, including *B. tulipae*. The major modes of action of *B. subtilis* include: (i) cyclic lipopeptides, such as iturin A, surfactin and fengycin, which affect fungal membrane structure; (ii) production of lytic enzymes, such as glucanases and proteases; (iii) competition for nutrients and iron through siderophore synthesis; and (iv) induction of systemic resistance (Ongena and Jacques, 2008; Karimi *et al*., 2012). In greenhouse trials, 52-75% control efficacy of *B. tulipae* has been obtained with *B. subtilis* formulations (Ongena and Jacques, 2008; Kohl *et al*., 1998).

Commercial formulation of *B. subtilis* (QST 713 strain, Serenade, AgraQuest) is registered in several countries for foliar application on ornamental plants, including tulip. The compatibility of *B. subtilis* with reduced rates of captan, with reduced residual activity compared to systemic chemical fungicides, makes it an attractive component for fungicide-biocontrol combination programs. However, its efficacy is affected by UV, temperature and rainfall and specific timing is required for its application.

Farmyard Manure (FYM) as a Soil Amendment

Farm yard manure (FYM) and compost amendments can improve soil suppressiveness to soilborne diseases, including those caused by fungi such as *B. tulipae,* through several routes, such as (i) promotion of various antagonistic microbial populations (bacteria, saprophytic fungi) to compete with and parasitize the sclerotia, (ii) increased populations of sclerotium parasitic bacteria (*Pseudomonas, Bacillus, Streptomyces* spp.), (iii) increased activities of enzymes such as chitinase and glucanase, which degrade fungal cell walls and (iv) physical soil structure and moisture-holding capacity, which can limit conducive conditions for disease (Hoitink *et al*., 1997; Bonanomi *et al*., 2010; Reuveni *et al*., 2002; Bellamy *et al*., 2004; Tamura and Briggs 1981).

Use of FYM as an amendment at 10-20 t/ha, incorporated into the soil prior to planting, has reduced viability of *B. tulipae* sclerotia by 30-55% over one growing season (Bonanomi *et al*., 2010; Pitt *et al*., 2010). The effect is enhanced if FYM is combined with antagonistic microorganisms such as *Trichoderma* spp. or Coniothyrium minitans, to create a rich soil ecosystem. FYM can improve plant nutrition and vigor, which can confer tolerance to *Botrytis* infection. However, fresh FYM can contain weed seeds and human pathogens and overuse can create imbalances.

Table 4 : Biological control agents used against *Botrytis tulipae*: mechanisms of action, efficacy, formulations and key references.

Biocontrol Agent	Mechanism of Action	Efficacy vs *B. ulipae* (%)	Formulation	Reference
Trichoderma harzianum	Mycoparasitism, enzyme secretion, ISR	55–78	WP, granule, liquid	Elad *et al*. (1994)
Trichoderma viride	Antibiosis, mycoparasitism	50–72	WP, biofungicide	Vinale *et al*. (2008)
Trichoderma asperellum	Competition, cell wall degradation	48–68	Liquid, granule	Shoresh *et al*. (2010)
Bacillus subtilis	Iturin/fengycin antifungals, ISR	52–75	WP, SC, liquid	Ongena and Jacques (2008)
Gliocladium catenulatum	Mycoparasitism, enzyme production	60–80	WP (Prestop)	Morandi *et al*. (2003)
Coniothyrium minitans	Sclerotia parasitism	45–65	WP (Contans)	Whipps *et al*. (2008)
FYM (Farmyard Manure)	Microbial diversity enhancement, suppression	30–55 (indirect)	Soil amendment	Bonanomi *et al*. (2010)

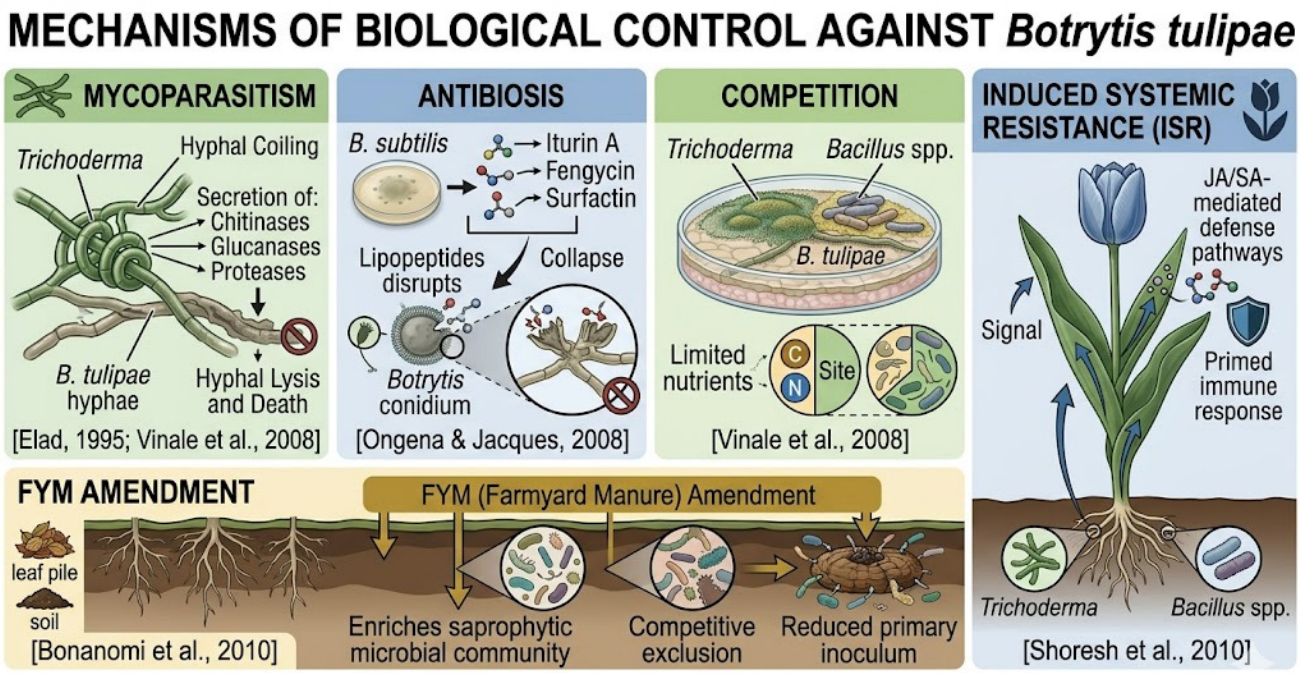

Fig. 3 : Mechanisms of action of biological control agents *Botrytis tulipae*

Integrated Disease Management (IDM)

Principles of IDM for Tulip Fire

The concept of IDM in *B. tulipae* management involves the strategic integration of cultural, chemical, biological and host resistance methods to attain long-lasting, cost-effective and environmentally safe control. The IDM concept was based on the idea of using the minimum amount of pesticides to control diseases while maintaining the level of diseases within acceptable limits by using the synergistic effect of different management tools (Leroux *et al*., 2010; Fillinger and Elad, 2016; Daughtrey *et al.,* 2005; Siah *et al.,* 2008).

Cultural Management Practices

Cultural practices provide the basis for IDM in *B. tulipae* management: (i) The adoption of disease-free certified planting bulbs obtained from reliable sources with known health status; (ii) Crop rotation of tulip with non-host crops for at least 3-4 years; (iii) Complete elimination and destruction of infected plant debris such as leaves, stems and flowers during or after the growing season; (iv) Optimization of plant density and architecture to enhance air circulation and reduce leaf wetness; (v) Irrigation management that minimizes leaf wetness; (vi) Adoption of bulb selection and roguing of infected bulbs prior to planting; and (vii) Gradual hardening of the crop and minimizing excessive application of nitrogen fertilizers that can stimulate succulent tissue that is more susceptible to disease (Coley-Smith, 1990; Hausbeck and Moorman, 1996; Holb and Vámos, 2018).

Combined Chemical-Biological Strategies

The integration of fungicides and biological control agents has been proven to have the potential for synergism in several instances, where *Botrytis* diseases have been targeted. These methods involve the following approaches: (i) the alternating application of fungicides and biocontrol agents to avoid antagonistic effects while maintaining a high and consistent level of control; (ii) the application of sub-lethal rates of fungicides and biocontrol agents to attain similar control with a reduced amount of fungicides; (iii) the application of fungicides during high-risk periods and biocontrol agents during low-risk periods; and (iv) the application of FYM + *Trichoderma* in the soil to control the primary inoculum and the application of fungicides to control the spread via the air (Elad and Stewart, 2004; Pertot *et al*., 2008).

The compatibility of captan, a fungicide with a moderate rate and multi-site activity and *Trichoderma* spp. has been proven to be effective. Research has demonstrated that the application of captan at recommended field rates has a minimum impact on the viability of *Trichoderma* spp. 5-7 days after application. However, carbendazim has been proven to be directly toxic to *Trichoderma* spp. and therefore should not be used in the same application as *Trichoderma* (Elad *et al*., 1993; Zimand *et al*., 1996)

Table 5 : Integrated disease management strategies for *Botrytis tulipae* combining chemical, biological, cultural and organic approaches with comparative efficacy data.

Strategy	Components	Mechanism/Rationale	Efficacy (%)	Reference
Chemical	Captan + Myclobutanil rotation	Resistance management, dual mode of action	75–88	Gullino *et al*. (2010)
Biological	*Trichoderma* + *B. subtilis*	Mycoparasitism + antibiosis synergy	60–78	Elad and Stewart (2004)
Cultural	Crop rotation + debris removal	Break disease cycle, reduce inoculum	40–60	Coley-Smith (1990)
Organic	FYM amendment + compost tea	Soil suppressiveness, microbial antagonism	35–55	Bonanomi *et al*. (2010)
IPM (Combined)	Fungicide + *Trichoderma* + FYM	Comprehensive suppression, reduced chemical load	82–92	Leroux *et al*. (2010)

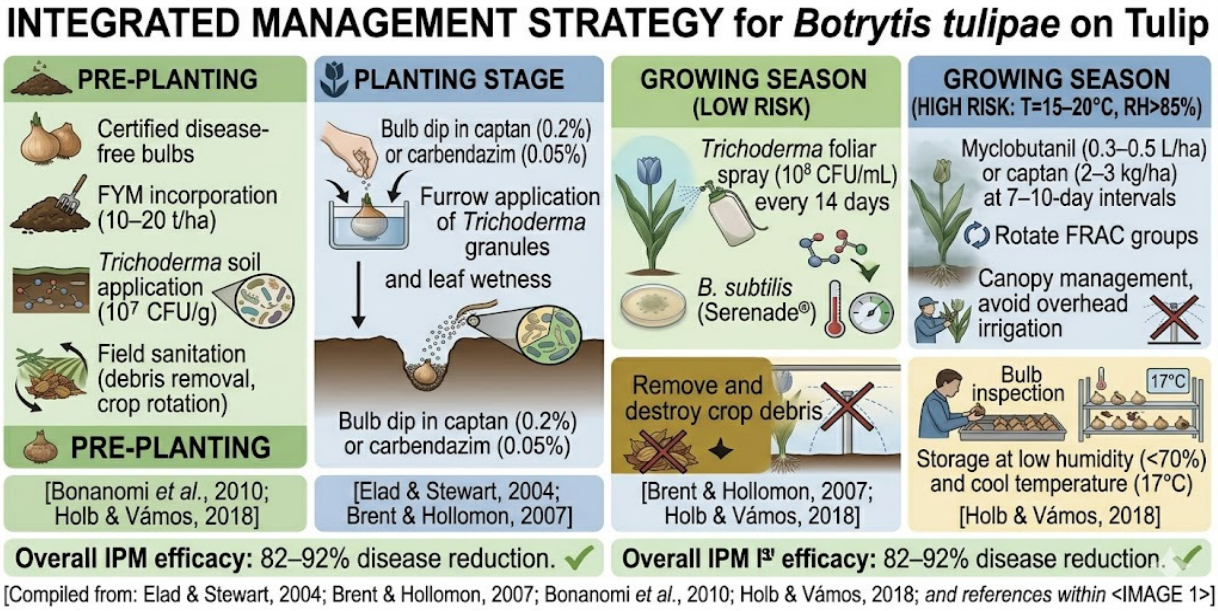

Fig. 4 : Integrated Disease Management framework for *Botrytis tulipae* of Tulip

Molecular Diagnostics and Detection

For IDM of *B. tulipae*, accurate and rapid diagnosis is fundamental. Classical diagnosis is based on morphological traits, host specificity and cultural features. These techniques are labor-intensive and demand mycological expertise. Molecular techniques have greatly improved the diagnosis and monitoring of *Botrytis* pathogens. These techniques have been used for species-specific diagnosis, resistance allele identification and population genetic studies.

Polymerase Chain Reaction (PCR)-based techniques have been developed for the diagnosis of *B. tulipae* by targeting species-specific sequences in the ITS region, beta-tubulin gene, or elongation factor-1α. These techniques can differentiate *B. tulipae* from *B. cinerea* and other *Botrytis* species within 4-6 hours (Staats *et al*., 2007; Crous *et al*., 2011). Quantitative PCR techniques have been developed for the quantification of *B. tulipae* inoculum in soil and plant tissues. These techniques can be used for risk assessment and decision-making on the timing of treatments. Molecular techniques have been developed for the identification of key resistance alleles. These techniques have been used for the monitoring of benzimidazole resistance based on codons 198 and 200 in the beta-tubulin gene in *B. tulipae* populations (Brent and Hollomon, 2007; Leroux *et al*., 2010; Boff *et al*., 2002). Loop-Mediated Isothermal Amplification (LAMP) techniques have been developed for the diagnosis of *B. tulipae*.

Host Resistance and Cultivar Selection

Host resistance is the management strategy with the greatest economic and environmental benefit; however, it remains elusive in *B. tulipae* control due to the lack of genetic diversity in tulip cultivars and the difficulties in breeding disease resistance with desirable horticultural characteristics in the complex polyploid genome. The majority of tulip cultivars show little or no immunity to *B. tulipae* infection; however, there is significant variability in the response of

different cultivars to infection, which can be explained by the composition of the cuticular wax layer, the density of the stomatal apparatus and the content of defense compounds (such as alkaloids and tulipane).

Tulip species such as *T. fosteriana, T. greigii* and *T. humilis* show improved levels of tolerance to *B. tulipae* infection and have been used in breeding programs to introduce *B. tulipae*-resistant cultivars. The objectives in tulip breeding programs include the combination of field resistance (partial or quantitative), which is not yet present in tulip cultivars, with exceptional floral characteristics, bulb performance and longevity. Marker-assisted selection (MAS) and genomic selection methods hold promise in the development of resistant tulip cultivars using the advances in the genome sequences of tulips.

Future Research Perspectives

Despite the significant advances in the biology and management of *B. tulipae,* there remain key knowledge gaps that need urgent research attention: (i) genome-level biology of *B. tulipae* and comparative genomics with *B. cinerea* to identify *B. tulipae*-specific effectors, virulence factors and targets of *B. tulipae* resistance genes; (ii) the development of standardized protocols for the sensitivity monitoring of key fungicide modes of action in *B. tulipae* populations; (iii) the improvement of *Trichoderma* and *Bacillus* formulations in terms of longevity, efficacy and compatibility with commonly used fungicides; (iv) large-scale field experiments to study the effects of FYM and compost amendments on *B. tulipae* sclerotia populations, soil microbiome composition and tulip bulb yield; (v) the application of climate models in predicting changes in *B. tulipae* epidemiology under different climate change scenarios, such as changes in the frequency of optimal infection periods; (vi) the potential of using RNA interference (RNAi)-based biopesticides in controlling *B. tulipae* populations using *B. tulipae*-specific essential genes; and (vii) the development of precision spray technologies that incorporate real-time weather information, disease risk models and fungicide resistance prevalence data.

Conclusion

Botrytis tulipae continues to pose a formidable barrier to tulip cultivation globally, with its epidemiology influenced mainly by environmental factors such as cool temperature, high humidity and extended durations of leaf wetness. The epidemiology of this disease, with its involvement of both sclerotial primary infection and conidial secondary infection, demands multi-pronged approaches for its management. The chemical fungicides captan (multi-site, FRAC M4), carbendazim (benzimidazole, FRAC 1) and myclobutanil (DMI triazole, FRAC 3) are essential for its control but face major hurdles in terms of the development.

Biological control methods using *Trichoderma harzianum, Trichoderma viride, Trichoderma asperellum, Bacillus subtilis* and *Gliocladium catenulatum* provide effective and environmentally safe control measures, especially when integrated with reduced fungicide application and cultural management. FYM soil treatments increase the soil's suppressiveness by adding more microorganisms and thereby competing against *B. tulipae*. However, the highest efficacy in the control of the disease (82-92%) is achieved by using IDM approaches that integrate chemical, biological, cultural and organic methods. Future research in the management of tulip bulb diseases should focus on molecular epidemiology, precision biocontrol products, resistance management and climate-resilient IDM approaches to ensure the long-term sustainability of the crop in the face of emerging pathogen populations and environmental conditions.

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