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Understanding *Botrytis cinerea* infection and gray mold management: a review paper on deciphering the rose's thorn

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Abstract

Gray mold of roses, caused by the necrotrophic fungal pathogen *Botrytis cinerea,* is an economically notorious disease and a well-known economic menace, leading to substantial annual losses estimated at no less than 30% of production. This disease takes a toll not only on the marketability of cut rose flowers but also on consumer and importer confidence due to the unsightly symptoms it induces. This fungus influences the rose foliage throughout cultivation, transportation, storage, and marketing. The interplay of conducive environmental conditions and genetic factors plays significant roles in developing the rose gray mold on rose flowers during the pre- and post-harvest phases. Nevertheless, the molecular mechanisms underlying *B. cinerea*-rose interactions are poorly understood, and the knowledge of how rose plants defend themselves against *B. cinerea* infection is not comprehensively investigated. It is worth noting that breeding for resistance to discover genetically resistant roses toward gray mold was unsuccessful despite research conducted over the past century. Consequently, synthetic fungicides remain the primary approach to controlling *Botrytis* blight in roses during the pre- and post-harvest stages. However, this measure has several drawbacks, including the emergence of fungicide-resistant *B. cinerea* and endangering human and animal health due to chemical residues in the food chain. This review aims to offer a comprehensive update on recent research findings on the biology and management of *B. cinerea* infection in roses and to propose novel strategies for managing gray mold disease.

Keywords Rose, Biotic stress, *Botrytis cinerea*, Gray mold, Long-term management

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Background

Globally, the rose (*Rosa chinensis* Jacq.) holds significant economic value as an ornamental and medicinal plant (Youren et al. 2015; Hao et al. 2017; Liu et al. 2019; Ullah et al. 2022). In recent years, the economic benefits of rose cultivation have been steadily increasing, leading to a continuous expansion of its cultivation area. However, this growth has been accompanied by the widespread occurrence of diseases, resulting in significant quality and yield losses (Youren et al. 2015). Throughout the cultivation process, this plant is vulnerable to attacks and infections by various pests and pathogens, giving rise to multiple diseases (Bebber et al. 2013; Youren et al. 2015). Among these diseases, gray mold, caused by *Botrytis cinerea* Pers. Fr. [Telomorph: *Botryotinia fuckeliana* (de Bary)], stands out as a devastating threat. It annually accounts for a minimum of 30% of worldwide production loss (Hao et al. 2017; Liu et al. 2018). This pathogen can attack more than 1400 plant species spanning nearly 600 genera worldwide (Elad et al. 2016; Garfinkel 2021), resulting in a staggering annual production loss estimated at $10 to 100 billion (Weiberg et al. 2013; De Angelis et al. 2022). Due to its broad host range, prolific reproductive capabilities, versatile infection modes, and the capacity to endure extended periods as conidia or sclerotia, *B. cinerea* is rightly recognized as a highly destructive pathogen (Williamson et al. 2007; De Angelis et al. 2022) and ranked second among fungal pathogens of economic importance (Dean et al. 2012). As a necrotroph, it predominantly infects and thrives on aging or damaged plant tissues, ultimately leading to tissue death. The ubiquitous *B. cinerea* inoculum (conidia) originates from infected plant tissues, gaining entry into the host through natural openings and wounds (Holz et al. 2004). Attacks on unripe or non-senescing plant organs may cause quiescent infections and minimal damage (Grant-Downton 2016). *B. cinerea* typically undergoes a brief asymptomatic biotrophic phase in the early stages of disease development (Veloso and van Kan 2018), which is often followed by an aggressive necrotrophic phase as plant organs mature or senesce. This necrotrophic phase causes the infected tissues to disintegrate (Veloso and van Kan 2018). Recent research has demonstrated that *B. cinerea* infection is a multilayered process controlled by the interaction of numerous factors that collectively affect the severity and the general course of the disease (Veloso and van Kan 2018; Bi et al. 2023). Hence, by no means a merciless killer, the fungus successfully manipulates and takes advantage of vital biological processes in host plants for its survival. This review compiles the latest research findings on the biology and management of *B. cinerea* infection in roses, while also proposing potential directions for the development of innovative strategies for combating gray mold disease.

Taxonomic status of *Botrytis cinerea*

The fungi *Botrytis* spp. belong to *Ascomycetes* from class *Leotiomycetes*, order *Heliotiales*, and family *Sclerotiniaceae*. Presently, 38 species of *Botrytis* have been formally recognized (Garfinkel 2021); all are necrotrophic except *B. pyriformis,* which is a saprotrophic species (Richards et al. 2021). Different *Botrytis* spp. have been identified on ornamental plants, such as *B. pelargonium* on geranium, *B. convoluta* on the iris, *B. calthae* on the caltha, *B. narcissicola, B. cinerea,* and *B. pseudocinerea* on narcissus, as well as *B. cinerea* and *B. pseudocinerea* on rose (Walker 2016; Muñoz et al. 2019). Based on molecular diagnosis, *Botrytis* spp. have been grouped into two phylogenetic clades. *B. pseudocinerea* and *B. cinerea* infecting plants from dicotyledonous are part of *Botrytis* clade 1, together with host-specific *Botrytis* species, *viz.*, *B. euclypti, B. sinoviticola, B. calthe*, and *B. fabae* (Hyde et al. 2014; Liu et al. 2016; Plesken et al. 2021). *Botrytis* clade 2 includes monocot-specific and phylogenetically more diverse *Botrytis* sp., having narrow host preferences (Staats et al. 2007; Hyde et al. 2014; Plesken et al. 2021). Recent research by Yuan et al. (2023) conducted a systematic study on the characteristics of *Botrytis cinerea* isolates from cut roses in Yunnan, China. They analyzed 100 isolates and found that all isolates belonged to the species *B. cinerea* based on morphological characteristics and phylogenetic analysis of *RPB2*, the gene encoding the second subunit of DNA-directed RNA polymerase, which is commonly used as a genetic marker for species identification. The study revealed insights into the pathogenicity, fungicide sensitivity, mating type, and genomic analysis of these isolates, providing significant support for gray mold control and further research in rose cultivation (Yuan et al. 2024).

Botrytis cinerea strains exhibited significant morphological variation, including variations in conidiation, mycelium development, and sclerotium production (Martinez et al. 2003; Plesken et al. 2021). Furthermore, numerous studies have also shown that populations of *B. cinerea* show substantial levels of genetic diversity (Rowe and Kliebenstein 2007; Fekete et al. 2012; Walker 2016; Plesken et al. 2021). *B. cinerea* was proposed to be a species complex with very controlled gene flow within distinct cryptic genetic groups that may vary with the tissue, host preference, and season (Fournier et al. 2005; Fekete et al. 2012; Muñoz et al. 2019). Early research focusing on fungicide resistance, restriction fragment length polymorphism (RFLP) patterns, and the identification of transposons displayed the presence of distinct *B. cinerea*

genetic groups (Giraud et al. 1999). Particularly, two sympatric sibling species were described based on the presence/absence of transposons: (i) *transposa*, containing *Boty* and *Flipper* transposons, and (ii) *vacuma*, having no transposon (Fekete et al. 2012; Walker 2016; Plesken et al. 2021). Moreover, several studies on nuclear genes have grouped *B. cinerea* populations into two clades (phylogenetic species), Group-I and Group-II, in various gene phylogenies (Albertini et al. 2002; Fournier et al. 2005; Cantu et al. 2008; Bi et al. 2023). The Group I isolates belong to the type *vacuma*, and Group II are from *transposa*, *Boty* (having only *Boty*), *Flipper* (having only *Flipper*), or *vacuma* genotype (Walker 2016; Garfinkel et al. 2019; Garfinkel 2020; Plesken et al. 2021). Group I had a very narrow genetic diversity than Group II, as revealed by vegetative incompatibility and DNA polymorphism studies (Fournier et al. 2005). Moreover, both groups showed differences in host range as well as morphological or phenotypic characteristics, e.g., strains from Group I have a very narrow host range and form significantly larger asexual spores as compared to Group II. Additionally, a subgroup of *vacuma* strains in Group I was found to vary from all other *B. cinerea* strains based on DNA sequencing and genetic markers. This subgroup was ultimately identified as a distinct *Botrytis* species known as *B. pseudocinerea* (Walker et al. 2011). In sympatry with *B. cinerea*, *B. pseudocinerea* is considered a minor species on strawberry and vineyard farms, but it can also infect other species of plants (Plesken et al. 2015).

The wide phenotypic variation and extensive host range exhibited by *B. cinerea* suggest a corresponding genetic diversity. Population genetics studies have revealed evidence of population clusters within *B. cinerea* from various host plants (Plesken et al. 2021). For instance, research conducted in France scrutinized *B. cinerea* populations, revealing that these populations were distinctly influenced by their host plants, such as grapevines and tomatoes (Walker et al. 2015). Further investigations, including pathogenicity trials, unveiled varying degrees of host preferences depending on the origin of the host (Mercier et al. 2019). Similarly, multiple fungicide-resistant *B. cinerea* strains have been discovered on German strawberry farms. Some of these strains showed mutations in *mrr1* (a transcription factor gene), leading to the enhanced expression of *atrB* (a gene for drug efflux transporter) and being partially resistant to fludioxonil and cyprodinil, the most frequently used fungicides against *Botrytis* (Kretschmer et al. 2009). These strains were later classified into a new subgroup of *B. cinerea* named *Botrytis* group-S (*B. cinerea* S) by sequencing *mrr1* and numerous other genes (Leroch et al. 2013). In Germany, *B. cinerea* S strains were dominant in the fields of strawberries but not observed in vineyards. Studies on *B. cinerea* populations in New Zealand's vineyards reported some strains similar to *B. cinerea* S but did not support their separation from other strains of *B. cinerea* (Johnston et al. 2014). Recently, using MLST (multilocus sequence typing) analysis of around 100 *B. cinerea* strains from various geographical origins demonstrated that several strains formed an as yet unidentified population group called group B, different from the other strains of *B. cinerea* in which the gene cluster for the production of botcinic acid (phytotoxin) was absent (Plesken et al. 2021). Moreover, a genetically distinct population group was formed by the strains of *B. cinerea* isolated from *Iris pseudacorus,* an attractive water garden plant, having an intact gene cluster for bikaverin (red pigment) production, usually degenerated in *B. cinerea.* In comparison to other *B. cinerea* strains on iris plants, these were notably more aggressive, providing compelling evidence for varying degrees of host adaptation and intra-specific differentiation within *B. cinerea.*

Rose-*Botrytis* pathosystem

The susceptibility of roses to *B. cinerea* has been investigated in numerous fresh-cut rose cultivars, and all were found to be highly vulnerable to *B. cinerea* infection (Friedman et al. 2010; Ha et al. 2021). *B. cinerea* penetrates the host through natural openings and wounds (Cao et al. 2019; Ha et al. 2021). During flower development, the infection starts with the landing of conidia on petals (Fig. 1). Symptoms of the disease are first visible on floral petals as minute quiescent lesions that later become necrotic and cover the entire corolla (Ha et al. 2021). The infection affects various plant organs and growth stages and reduces the postharvest quality of roses, but the invasion and the infection of the flower petals cause the most significant economic damage (Youren et al. 2015; Ha et al. 2021). The pathogen may cause minor damage and latent infection when it occurs on non-senescent or unripe plant organs. There have been reports of three different types of *B. cinerea* latent infection, including growth arrest after germination or a delay in conidia germination, symptomless endophytic growth in the apoplast, and the colonization of abscising floral parts like petals before expansion into receptacles or ovaries where growth is arrested (Sowley et al. 2010; Petrasch et al. 2019; Ha et al. 2021). Moreover, recent research by Liu et al. (2024) sheds light on the molecular mechanisms underlying the defense response of roses to *B. cinerea* infection. The study characterizes two ethylene- and jasmonic acid-regulated transcription factors, RhEFR005 and RhCCCH12, which bind to the promoter region of *PATHOGENESIS-RELATED 10.1*

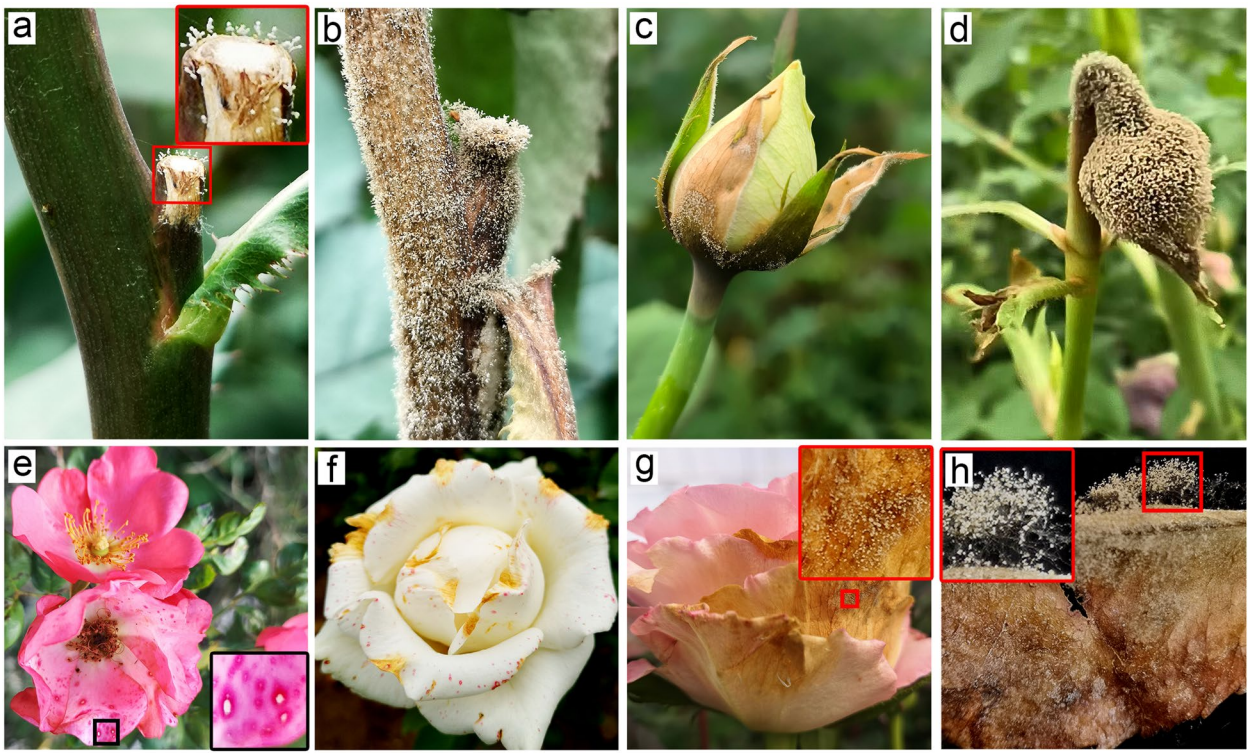

Fig. 1 Disease symptoms on roses caused by *Botrytis cinerea*. **a** The pathogen invaded through the wound stem of roses. **b** The pathogen caused stem death, and a large number of conidia were produced at the late stage of infection.**c** The sepals and outer petals of the bud were infected in the early stage. **d** The bud rotted, and a large number of conidia were produced on its surface at the late stage of infection. **e** White, red, or light-yellow spot symptoms were produced on the petals. **f** The lesion gradually expanded, and the petals began to rot, taking on a water-stained, yellow or brown appearance. **g** The petals were rotted, and conidia were produced on the surface at the late stage. **h** Gray mold infection caused the leaves to wither and fall, and a large number of mycelia and conidia are attached to the surface of the leaves in a humid environment

(*RhPR10.1*) and promote its transcription, leading to decreased susceptibility to *B. cinerea*. The findings suggest that the *RhERF005/RhCCCH12–RhPR10.1* module regulates the cytokinin-induced defense response of roses to *B. cinerea*. This research provides valuable insights into potential strategies for enhancing roses' resistance to *B. cinerea* infection (Liu et al. 2024). Additionally, recent findings by Li et al. (2024) demonstrate that transcription factors RhbZIP17 and RhWRKY30 enhance resistance to *B. cinerea* by increasing lignin content in rose petals. This study further highlights the importance of understanding the regulatory mechanisms underlying plant immunity against *B. cinerea* and offers promising avenues for developing effective strategies to combat this pathogen (Li et al. 2024a). Eventually, integrated proteomic analysis by Li et al. (2024) reveals interactions between phosphorylation and ubiquitination in regulating the rose response to *Botrytis* infection, providing insights into the molecular mechanisms underlying rose resistance to *B. cinerea* and increasing the database of phosphorylation and ubiquitination sites in plants (Li et al. 2024b).

Mechanisms of *B. cinerea* infection

Model organisms have been used to explore the mechanisms of the *B. cinerea* infection (Staats and van Kan 2012; Van Kan et al. 2017), which starts when oval conidia of 50 to 75 μm^3 in size germinate on the plant surface, thereby producing simple appressoria and infection cushions that assist in host penetration (Bi et al. 2023). The pathogen also enters its host via stomata or directly penetrates the cuticle by producing conidial germ tubes (Arya et al. 2021). Two distinct phases have been recognized following the initial contact with the host: an initial stage in which local infection foci are produced but cannot expand or spread, and a late phase where infection spreads and fungal biomass is produced abundantly. The model proposed

by Shlezinger et al. (2011) shows that for the establishment of infection, host cells must be killed to produce a portion of dead tissues in which the pathogen accumulates its biomass before entry into the next infection stages (Shlezinger et al. 2011). It is also believed that the compounds that promote the killing of host cells are critical in completing the early phase of infection. To facilitate local host cell death, *B. cinerea* produces an array of cell-death-inducing proteins (CDIPs) in addition to plant cell wall degrading enzymes (PCWDEs) and toxins that manipulate the plant-regulated cell death (RCD) machinery (Govrin and Levine 2000). In contrast, antimicrobial plant metabolites induce *B. cinerea* to undergo massive RCD towards the end of the early phase and into the intermediate phase of infection (Shlezinger et al. 2011). At this stage, the survival of *B. cinerea* depends on the antiapoptotic machinery, and whether the fungus will develop to the next stage or be blocked is determined by a balance between fungal and plant cell death (Bi et al. 2023).

An alternative model in which the *Sclerotinia sclerotiorum* infection process was proposed is that the fungus initially maintains plant cell viability in a similar way as proposed for *B. cinerea* (Veloso and van Kan 2018; Bi et al. 2023). This model suggested that, due to the suppression of autophagic cell death, which halts the induction of self-destruction, the host cells remain viable following the initial fungal invasion. Once the fungus is established within the host tissues and has accumulated sufficient biomass, the production of RCD-promoting compounds replaces the secretion of autophagy-suppressing molecules, which leads to the death of plant tissues and disease development (Bi et al. 2023). It is unclear whether the early infection stages include a brief biotrophic phase or immediate cell death induction, but both models agree that the initial encounter leads to the formation of an infection court and the accumulation of fungal biomass and that RCD-inducing molecules facilitate lesion spread (Shlezinger et al. 2011; Veloso and van Kan 2018; Bi et al. 2023). Due to the manipulation of regulatory machinery based on hypersensitive response (HR) by putative fungal effectors, cell death induced by toxins and CDIPs keeps spreading (Rai et al. 2023). This is supported by the findings that HR is important for *B. cinerea* infection and protects plants from biotrophic pathogens (Govrin and Levine 2000) and that plants block RCD and prevent infection by expressing antiapoptotic genes (Yu et al. 2023). Moreover, it has also been observed that after initial necrosis and prior to lesion expansion, the infection caused by *B. cinerea* or *S. sclerotiorum* was blocked (Hossain et al. 2023), suggesting that compounds that induced RCD also promoted lesion expansion, whereas the formation of local lesions involved RCD and necrotic cell death.

Plant defense responses

After successful penetration, *B. cinerea* must cope with plant defense mechanisms such as phytoalexins and other plant antimicrobials playing a crucial role in disease progress (de León and Montesano 2013; Bulasag et al. 2023). *B. cinerea* has developed several mechanisms to overcome plant defense compounds, like exporting toxic glucosinolates or camalexin (Stefanato et al. 2009) and the degradation of α-tomatine (Hui et al. 2023). It must overcome plant immune responses once inside the host plant. No host-specific toxins or Avr effectors in the *B. cinerea* toolkit have been found (Bi et al. 2023). As a result, no single gene imparts plant resistance to *B. cinerea*, and effector-triggered immunity (ETI), caused by a specific interaction between a plant receptor (R gene) and pathogen effector, is mainly of no consequence. However, in the absence of complete resistance, a quantitative plant defensive response based on the identification of conserved pathogen-associated molecular patterns (PAMPs) by plant pattern-recognition receptors (PRR) parallels the quantitative virulence of *B. cinerea* (Liao et al. 2022). The most commonly recognized PAMPs as fungal signatures include chitin oligomers, as well as CDIPs that are vital for virulence and recognized by plant receptor-like proteins (RLPs) and receptor-like kinases (RLKs) that initiate plant immune responses. Pathogen perception by receptors leads to the influx of calcium and also initiates a cascade of phosphorylation that can activate mitogen-activated protein kinases (MAPKs), calcium-dependent protein kinases (CDPKs), and cytoplasmic kinases (RLCKs). It has been observed that following *B. cinerea* infection, the earliest induced gene is *Arabidopsis* RLCK gene *BOTRYTIS INDUCED KINASE1* (*BIK1*) (Veronese et al. 2006). Through its function in ethylene signaling BIK1 integrates PAMP-triggered immunity (PTI) signals independently with MAPKs downstream of PRRs thereby connecting plant growth to immune responses (Lal et al. 2018). Plant immune responses, downstream of these signal cascades, include cell wall reinforcement, callose deposition, the production of plant defense compounds (phytoalexins), and reactive oxygen species (ROS) (Veronese et al. 2006; Ahuja et al. 2012), which may reduce the local infection and systemically enhance the immunity of non-infected plant parts, a process commonly known as systemic acquired resistance (SAR).

Botrytis cinerea toolkit

B. cinerea aggressively increases plant susceptibility by utilizing a large array of virulence elements (Nakajima and Akutsu 2014; Petrasch et al. 2019). Initially, it deploys

effector proteins and sRNAs that cause gene silencing and suppress host immunity. This allows the pathogen to establish itself inside the host, accumulating biomass before entering the necrotrophic phase (Veloso and van Kan 2018; Bi et al. 2023). It has also been reported that *B. cinerea* Dicer-like proteins (DCL1 and DCL2) generate sRNAs (small RNAs) that are released from fungi and move into plant cells, where they interact with the host's RNAi (RNA interference) systems to suppress immune response genes of the host plant (Weiberg et al. 2013; Wang et al. 2016). Moreover, some secreted virulence factors, such as toxins, enzymes, and effector proteins, can produce reactive oxygen species (ROS), leading to host cell death (Schumacher 2016). The pathogen also secretes oxalic acid and stimulates the synthesis and activity of fungal pectinases, proteases, and laccases by lowering the pH of host tissues (Sharon et al. 2004; Fernández-Acero et al. 2010), which also results in Ca^{2+} chelation, thereby weakening the pectin substances of cell walls and inhibiting the deposition of callose (Chakraborty et al. 2013). *B. cinerea* also decomposes host cell walls and obtains nutrition through a massive secretion of cell wall degrading enzymes (CWDEs) like cellulases, hemicellulases, and pectinases (Blanco-Ulate et al. 2016; Bi et al. 2023). It has also been demonstrated that the pathogen produces plant hormones or their analogs that might disrupt the cellular metabolism of the host plant (Petrasch et al. 2019).

Disease cycle and epidemiology

The lifecycle of *B. cinerea* consists of three consecutive stages: germination, penetration, and establishment (Elad and Shtienberg 1995; Körner et al. 2014). The conidia of *B. cinerea* released from overwintering sites are the primary inoculum source. These landed spores germinate to develop a germ tube when a thin water layer is formed on the foliage surfaces. In the glasshouse, mainly during the production stage, conidia are deposited on flowers and remain dormant until a thin water coating is accessible for germination (Kerssies et al. 1995). Numerous studies have noted that during the first two phases, the dispersed and newly germinated conidia are extremely sensitive to microclimatic conditions (Williamson et al. 2007). However, the mycelium is affected by the conditions available within the host plant during the last phase. The conidia germinate by forming germ tubes, which may directly penetrate the petals or develop the appressoria (Williamson et al. 2007). However, penetration through natural openings (stomata) and wounded tissues has been reported (Elad 1988; Kerssies et al. 1995). It is believed that dead cells and wounded host tissues could facilitate the infection process (Elad and Shtienberg 1995; Williamson et al. 2007; Ha et al. 2021). The report demonstrated that the exposed spores to free water germinated at 22°C after 4 h of incubation. Additionally, the germination of 50% and 95% of exposed spores was documented after 7 and 11 h of incubation, respectively. Relative humidity (RH) plays a role in the germinating process, in which the spore can germinate at 100% RH after 30 hours, even in the absence of water. Following the germinating event, the penetration phase for *B. cinerea* takes approximately 2–3 h. Consequently, the fungus is exposed for 9 to 10 h to microclimatic conditions (germination 7 h and penetration 2– 3 h). The penetration peg and other infective hyphae continue to develop into the epidermal cells after penetration while releasing various toxins and metabolites that are destructive to the host plant tissues. Usually, during the establishment phase, *B. cinerea* produces a plethora of toxins and enzymes to kill the epidermal and underlying cells. Afterward, the infective hyphae start to spread, resulting in massive tissue maceration and the production of conidia, which are secondary inoculum that infect other host plants or non-infected parts (Schumacher 2016). Under ideal conditions, the establishment phase (i.e., the interval between infection and the expression of disease symptoms) takes 5–8 days (Elad and Shtienberg 1995; Williamson et al. 2007). Following the death of the plant, *B. cinerea,* as a typical necrotroph, feeds on dead plant tissues and produces abundant conidia formed on conidiophores, which may land on other host plants or soil and continue the lifecycle (Fig. 2).

The pathogen overwinters by producing sclerotia inside the dying host tissues. The internal mycelium of sclerotia is encased with β-glucans and melanized rind, which protects sclerotia against extreme conditions and microbial attack over longer periods (Williamson et al. 2007; Schumacher 2016). The sclerotia commence growth by producing conidiophores and multinucleate conidia, which serve as a primary inoculum source. The main inoculum sources of *B. cinerea* in glasshouses are dead foliage and petals accumulating on the soil and greenhouse floor. Furthermore, the dead flowers, leaves, and fruits of perennial crops contain large amounts of mycelium that can form conidia and establish infection. In aging cultures, *B. cinerea* also produces large quantities of microconidia from phialides, which predominantly serve as spermatia. The sexual lifecycle involves the spermatization of sclerotia and the formation of apothecia and asci with eight binucleate ascospores (Beever and Weeds 2004; Williamson et al. 2007).

B. cinerea sporulation on senescent and dead plant tissues happens at a wide range of temperatures; however, a water film on a mycelium-covered tissue inhibits its sporulation. Although particular wavelengths of light encourage the fungus to sporulate, some reports indicate that many field isolates commonly sporulate

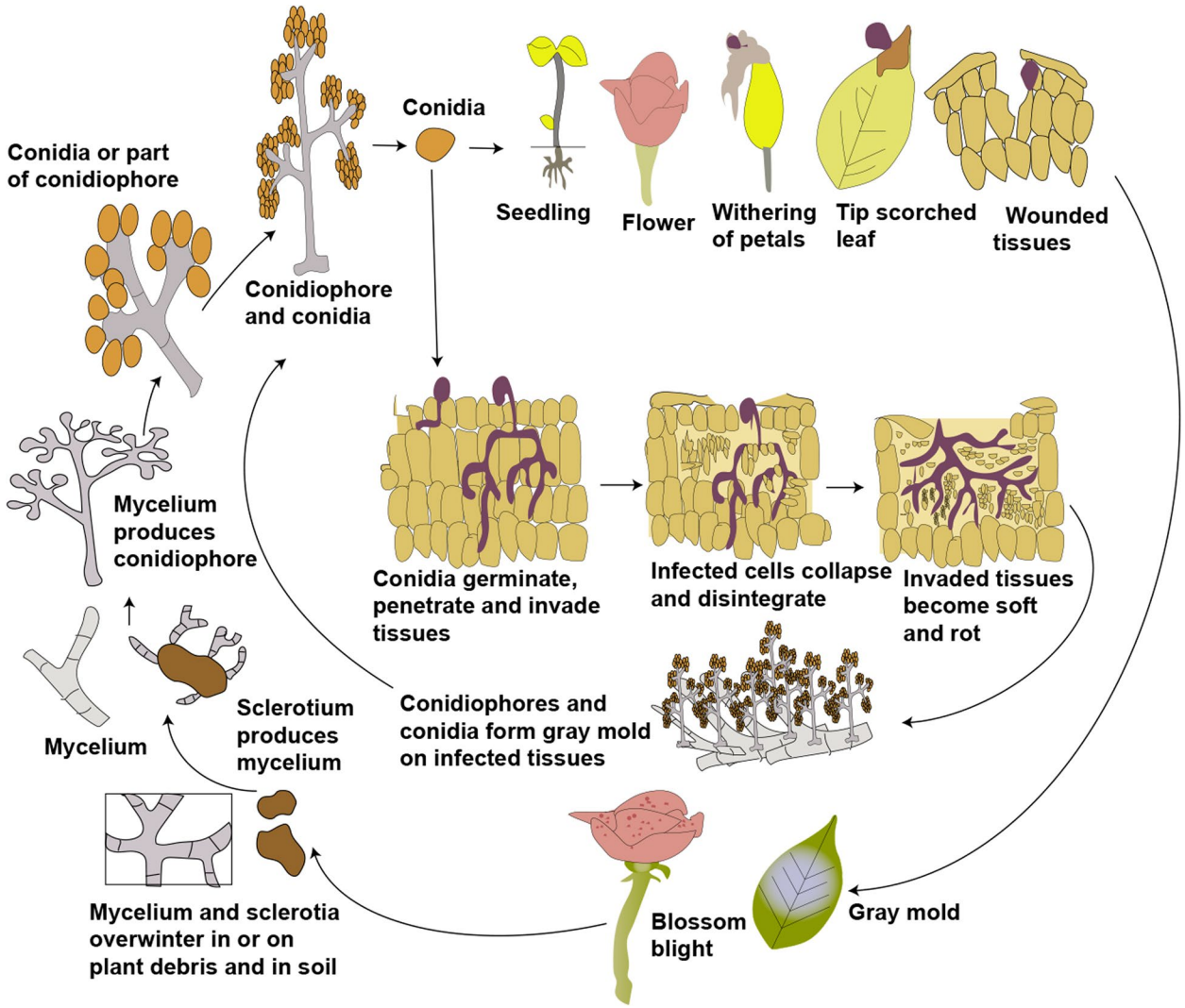

Fig. 2 The disease cycle of *B. cinerea* in Chinese roses. The sources of *B. cinerea* inoculum include diseased leaves, petals, and sclerotia. The depiction of primary infections affecting flowers and secondary infections affecting petals is evident (Petrasch et al. 2019)

under darkness (Elad and Shtienberg 1995; Williamson et al. 2007; Cerón-Bustamante et al. 2023). Conidia formed at the sources of primary inoculum follow a well-defined diurnal cycle of initiation, production, and dispersal; this cycle is regulated by temperature and RH fluctuations. According to a prior study, conidiophores can dry out and twist early in the morning as a result of a sudden reduction in RH and an increase in temperature, which can cause conidia to be released into the air singly or in small clusters (Williamson et al. 2007; Ha et al. 2021). Water droplets are also known to disperse conidia but are not considered a major dispersal mechanism. Although conidia might travel on air currents from nearby crops, most conidia are probably generated from primary sources within the crop.

During epidemics, cool and humid conditions favor *B. cinerea* infection and enhance the host's susceptibility (Elad and Shtienberg 1995; Romanazzi et al. 2016). The most important environmental factors influencing *B. cinerea* infection are high RH and free moisture on plant surfaces (González-Fernández et al. 2021). For germination and germ tube development, the conidia of *B. cinerea* require a thin layer of water. Previous reports documented that a high relative humidity of more than 93% is crucial for conidial germination and infection (Williamson et al. 2007; Thaochan et al. 2020; Ha et al. 2021). The optimum temperatures for successful infection range from 10 to 20°C, but it may occur even at 2°C and above 25°C (Ha et al. 2021).

Disease management

Cultural and physical practices

Cultural and physical measures can serve as potent tools for suppressing rose *Botrytis* blight, particularly in glasshouse conditions where the causal agent's activity highly depends on environmental conditions. Such practices are generally aimed at reducing the conditions conducive to the causal agent and disease development by altering the microclimate in the canopy and around the susceptible organs, preventing the entry of the causal agent into the greenhouse, and providing an environment for the plants, which is less favorable for disease development (Elad and Shtienberg 1995). Researchers and growers recommended constructing the greenhouse in such a way that is equipped with robust ventilation and cooling systems to maintain the greenhouse humidity below 80% and promote drying. More importantly, expanding the space between plants to provide effective air circulation around the plants and avoiding water splashes on foliage during irrigation play an essential role in diminishing disease development.

Furthermore, certain physical interventions can complement cultural practices. For instance, discouraging late evening and overhead irrigation, which wets the leaves, helps prevent conditions conducive to causal agent development and reduces plant susceptibility to disease. It is also reported that UV light (300–400 nm) can trigger the *B. cinerea* sporulation, while blue light (380–530 nm) acts oppositely, inhibiting conidiation of this pathogen, so that applying thermal screens as well as photo-selective polyethylene sheets are proposed in constructing the greenhouses (Reuveni et al. 1989). Nitrogen-based fertilizers should be used at a standard level, as this substance can make the plant more vulnerable to *B. cinerea* by causing the cells to become crispy and providing the pathogen with favorable conditions to develop (Abro 2013). Sanitation practices are integral to managing *B. cinerea* in the rose greenhouse. At the first sign of symptoms, all diseased stems, flowers, and canes should be promptly removed and destroyed to prevent further spread of the disease.

Chemical control

Spraying with synthetic fungicides is the primary strategy for controlling *Botrytis* blight in roses before or after harvest. Benzimidazoles and dicarboximides are two chemical classes commonly used to manage *B. cinerea*. Previously, a procedure was used to dip cut rose flowers in fungicide solutions to prevent the post-harvest development of *B. cinerea*, but this left unsightly residues that hampered this practice widely (Ha et al. 2020). Studies have examined the effectiveness of various fungicides in combating *B. cinerea* in roses. For instance, on 'Sonia' roses infected with *B. cinerea* and incubated at 2.5°C in H_2O-saturated air, spraying with the common dicarboximide Vinclozolin (Ornalin 50 WP) reduced disease severity (Hammer et al. 1990). Elad (1988) investigated the effectiveness of 18 fungicides on the disease development of *B. cinerea* in rose-cut flowers at various temperatures (Elad 1988). Results indicated that several fungicides, including metomeclan, dichlofluanid, myclozolin, N-phenylcarbamate plus carbendazim (NPC + MBC), polyoxin D, prochloraz, and iprodione led to a significant reduction in disease index, ranging between 0.7–1.8 compared with that of the non-treated plants (4.8) at 20°C while the most effective ones at 10°C were NPC + MBC, polyoxin D, and chlorothalonil (Elad 1988). Similarly, a previous study exhibited that weekly applications of polyoxin B, fenpiclonil, and tebuconazole in a rose greenhouse reduced rose branch infection by 50–70% (Elad et al. 1993). Only two days after the treatment, polyoxin B (67%) and fenbuconazole (68%) both applications significantly reduced the severity of petal infection. Additionally, in an experiment where cut rose flowers were collected from commercial greenhouses, sprayed with fungicides, and kept in conditions conducive to *Botrytis*, iprodione plus TMTD, tebuconazole plus dichlofluanid, and polyoxin D could significantly and constantly reduce the post-harvest infection by *B. cinerea* (Elad et al. 1993). More recently, an *in vitro* assay provided solid evidence that Cabrio top (Metiram 55 % + Pyraclostrobin 5 % WG) at a concentration of 300 ppm significantly decreased the linear colony growth (31 mm) of *B. cinerea*, sampled from rose commercial greenhouses, compared with the control treatment (90 mm) (Jatoi et al. 2022).

Nevertheless, applying synthetic fungicides to control *Botrytis* blight in rose flowers and other ornamental plants raises several serious issues. On the one hand, because of the pathogen's high genetic plasticity and adaptability, *B. cinerea* populations are developing resistance to single-site or multi-site fungicides. On the other hand, these substances can persist in food chains and cause health issues in humans and animals. It is important to note that they are expensive, and governmental organizations impose severe limits on their manufacturing and use because these compounds are associated with health and environmental problems. In 1988, resistance to the fungicides benzimidazole (benomyl) and dicarboximides (iprodione) was assessed in 66 *B. cinerea* isolates obtained from the roses. The finding revealed that 23% of the investigated isolates were resistant to both fungicides, while 11% and 16% were just resistant to benomyl and iprodione, respectively (Elad 1988). Fungicide resistance profiles of 49 *B. cinerea* isolates obtained from commercially cut roses were estimated using previously

published discriminating dosages. The results indicated that the examined isolates were frequently resistant to thiophanate-methyl (FRAC 1), iprodione (FRAC 2), cyprodinil (FRAC 9), and boscalid (FRAC 7). Furthermore, no resistance to pydiflumetofen was reported at the discriminative tested doses (Muñoz et al. 2019).

Biological control

There is accumulating evidence demonstrating the significant potential of microorganisms accommodating the phyllosphere to control *Botrytis* blight, a notorious disease of greenhouse-cultivating roses impacting the production and post-harvest quality of cut roses. Furthermore, heightened societal and scientific concerns for sustainable management strategies have spurred considerable efforts to identify promising biological control agents (BCAs). These methods offer an eco-friendly alternative to chemical fungicides, particularly in cases where their widespread use is restricted by regulations imposed by importing countries or due to concerns about the accumulation of residues in food chains, which can jeopardize human and animal health. Several microorganisms have been assessed for their ability to control *Botrytis* in roses to achieve these objectives.

Microbial resources

In an early attempt to manage *Botrytis* blight on roses, researchers evaluated 72 epiphytic microorganisms isolated from rose petals for their potential to reduce lesions on detached petals of the Golden Wave rose cultivar (Table 1). The most effective antagonists, including *Exophiala jeanselmei*, *Cryptococcus albidus*, *Erwinia* sp., and coryneform bacteria, were applied to the entire cut flower one day before infection by a suspension of 1000 conidia of *B. cinerea* per mL. The infection assay demonstrated that the most effective isolate was black yeast, *E. jeanselmei*, which diminished the number of lesions by 63%. This degree was comparable with the level of 74%

Table 1 Microbial resources from the rose petals and their potential to reduce lesions

Microbial Strain	Source of Isolation	Activity	Cultivar Tested	Reference
Fungi				
Trichoderma harzianum	Cucumber fruit	DR: 50%	Florop	(Elad et al. 1993)
Cladosporium oxysporum (FP123)	Rose petals	DR: 52%		(Tatagiba et al. 1998)
Cladosporium oxysporum (FP157)		DR: 52%		
Cladosporium cladosporioide (FP21)		DR: 49%		
Cladosporium cladosporioide (FP139)		DR: 32%		
Ulocladium atrum (302)	Carrot seed	DI: 27.1%	Mistral	(Yohalem 2004)
Ulocladium atrum (385)	Onion	DS: 0.2	Hit Parade	
Clonostachys rosea (NCR27/R)	*Coffea arabica* L./DL	REP: >95%	Sandra	(Nobre et al. 2005)
Clonostachys rosea (NCR28/R)	*Macadamia* sp./DL			
Clonostachys rosea (NCR60/F)	*Eucalyptus globulus*/AL			
Clonostachys rosea (NCR61/F)	*Prunus persica*/AL			
Yeast				
Exophiala jeanselmei	Rose petals	DR: 63%	Golden Wave	(Redmond et al. 1987)
		DS: 3.6	Sonia	(Hammer and Marois 1989)
Cryptococcus albidus		DR: 26%	Golden Wave	(Redmond et al. 1987)
Debaryomyces hansenii (Si29)		DS: 0.1	Vendela	(Zapata et al. 2016)
Rhodotorula mucilaginosa (Co3)		DS: 0.2		
		DI: 20%		
Pichia onychis (F11)		DS: 0.4		
		DI: 20%		
Pichia onychis (F14)		DS: 0.4		
		DI: 20%		
Bacteria				
Corynebacterium sp.	Rose petals	DR: 48%	Golden Wave	(Redmond et al. 1987)
		DS: 5.1	Sonia	(Hammer and Marois 1989)
Erwinia sp.		DR: 25%	Golden Wave	(Redmond et al. 1987)
Bacillus subtilis (BP161)		DR: 53%		(Tatagiba et al. 1998)

Disease Reduction (DR): Percentage reduction in disease incidence. Disease Severity (DS): The severity of the disease, measured on a scale. Disease Incidence (DI): Percentage of plants showing disease symptoms. Suppression of *B. cinerea* sporulation (REP): Repression percentage of *Botrytis cinerea* sporulation.

obtained by employing the fungicide iprodione (Redmond et al. 1987). In a follow-up study, *E. jeanselmei* and a coryneform bacterium controlled the *B. cinerea* infection during storage at 2.5°C. The magnitude of disease reduction was comparable to the level achieved by applying the vinclozolin fungicide, but the biological antagonists were unable to control post-storage disease development at room temperature (21°C) (Hammer and Marois 1989). Elad et al. (1993) reported that spraying a rose greenhouse with *Trichoderma harzianum* once a week yielded a 50% reduction in the disease development of gray mold on rose branches, but this degree was not significantly different from the level achieved by the control. In an attempt to identify microorganisms enabling the control of *B. cinerea* in leaves and residues of rose, *Gliocladium roseum* and *T. inhamatum* decreased the pathogen sporulation by >90% in leaf residues artificially infested with *B. cinerea*. This suggested that these isolates have a strong capacity to suppress inoculum production of *B. cinerea* in rose leaf residue. Furthermore, it was shown that *T. inhamatum*, *C. oxysporum*, and *G. roseum* were leading antagonists against *B. cinerea* in leaf residues naturally infested with the pathogen, and it was demonstrated that *G. roseum* was able to suppress lesion formation by *B. cinerea* in detached petal by more than 90% (Tatagiba et al. 1998). Experiments conducted in a Dutch commercial greenhouse exhibited that the saprophytic fungus *Ulocladium atrum* Preuss significantly suppressed *B. cinerea* sporulation in assays on pot rose (Köhl and Gerlagh 1999). Furthermore, two isolates of *U. atrum* (302 and 385) significantly reduced the disease incidence and the sporulation of *B. cinerea* to a greater extent than the levels achieved by applying the *G. roseum* isolates (201 and K726). In this study, both tested *G. roseum* isolates functioned better than the commercial *Trichoderma* products (Trichodex and Supresivit), which were not significantly different from the negative controls (Yohalem 2004).

G. roseum, a non-pathogenic endophytic agent, is a versatile adversary of *B. cinerea*, causing disease on various ornamental plants. This fungus possesses a strong track record of meeting societal expectations in terms of effectiveness, reliability, and cost-effectiveness in biological control (Sutton et al. 1997). Notably, *G. roseum* was recently reclassified as *Clonostachys rosea* (Link: Fr.) Schroers, Samuels, Siefert, and W. Gams [teleomorph, *Bionectria ochroleuca* (Schw.) Schroers and Samuels] (Schroers et al. 1999). Morandi et al. (2000) revealed that the Canadian *C. rosea* Pg 88-710 germinated and established endophytic growth on both living and dead foliage of roses, ranging from 31% to 98% under controlled conditions, respectively. The average frequency of sporulation on inoculated tissues, including leaves, petals, and dead tissues, was estimated to vary between 41% and 75%. This data underscores that *C. rosea* has a remarkable potential to efficiently control the inoculum generation of *B. cinerea* on rose petals and debris, the central sites of primary inoculation during dormant stages accumulated on the soil or greenhouse floor. Subsequently, similar assays were repeated under a commercial greenhouse condition to investigate the impact of *C. rosea* inoculation on rose leaves and debris, reducing the *B. cinerea* sporulation intensity. This study demonstrated that the sporulation intensity of *B. cinerea* on rose debris consistently declined by 30–50% on plants sprayed fortnightly with *C. rosea* or *C. rosea* plus mancozeb. Based on the two previous studies, continuous application of *C. rosea* on debris, coupled with sanitation practices, is recommended as a complementary and promising practice to reduce *B. cinerea* sporulation and disease incidence in the buds (Morandi et al. 2003). Building on these promising results, further studies aimed to discover native Brazilian *C. rosea* isolates that effectively suppress the sporulation and colonization of *B. cinerea* on roses. They recovered four potential *C. rosea* isolates from various host plants and assessed their capacity to prevent the rose *Botrytis* blight. The isolates of *C. rosea* named NCR59/N, NCR61/F, and NCR28/R colonized the rose leaves similar to the levels achieved by the reference isolate Pg 88-710. Isolates NCR27/R, NCR28/R, NCR60/F, and NCR61/F could inhibit the *B. cinerea* sporulation on rose by more than 95% (Nobre et al. 2005). Later, the impacts of microclimatic factors (air temperature and relative humidity) on the abilities of *C. rosea* and *B. cinerea* to colonize and sporulate on rose debris under a commercial greenhouse were investigated. The obtained data emphasized that *C. rosea* can remarkably suppress inoculum production of *B. cinerea* on rose debris in a wide range of environmental conditions occurring in commercial greenhouses (Morandi et al. 2006). Morandi et al. (2008) examined the impacts of application time and exposure to sunlight on the establishment, survival, and capacity of *C. rosea* to reduce *B. cinerea* sporulation on rose debris. The finding indicated that the germination frequency of *C. rosea* was significantly lower on treatments maintained away from direct sunlight, and this ratio was inversely correlated with the application time, independent of exposure time to the sunlight. Exposure to sunlight resulted in a slight suppression of pathogen sporulation ranging between 94.5–100% and 65–93% in both controlled and uncontrolled conditions, respectively (Morandi et al. 2008). Applying the four yeast isolates, including *Debaryomyces hansenii* Si29, *Rhodotorula mucilaginosa* Co3, and *Pichia onychis* F11 and F14, led to a remarkable decrease in the incidence and severity of *Botrytis* blight on rose petals compared to the untreated control. These isolates

demonstrated a notable tolerance for UV radiation and adhesion to the leaf surfaces, adding value to them and making them attractive options for use as primary ingredients in the creation of commercial products (Zapata et al. 2016).

Bio or (in)-organic compounds

Researchers have recently focused on the role of natural antifungal compounds, such as botanical extracts, offering a plethora of structurally different fungicidal compounds and essential oils (EOs) and natural complex compounds biosynthesized by plants, in controlling *Botrytis* blight. Neem extracts effectively reduced the linear colony growth (LCG) of a *B. cinerea* isolate recovered from a rose *in vitro* by 23 mm compared to the control treatment (90 mm) (Jatoi et al. 2022). The essential oil derived from the medicinal plant *Dysphania ambrosioides* decreased the mycelial growth and spore germination of *B. cinerea* by 60% and 51%, respectively. However, this mixture failed to suppress the establishment of this fungus on a rose petal and caused a color alteration 24 h after the treatments (Salimena et al. 2015; Ignacchiti et al. 2022). The EO extracted from thyme (*Thymus vulgaris* L.) reduced *Botrytis* damage on a rose in such a way that the disease rating was not statistically different from that achieved by using the fungicide fludioxonil, but this was accompanied by flower phytotoxicity.

Switching the EO delivery method from an aqueous solution to a vapor led to lower flower phytotoxicity without further changes in the *Botrytis* damage (Bergmann and Dole 2018). Recent studies showed that the EO derived from *Pelargonium graveolens*, at a concentration of 250 ppm, completely inhibited the mycelial growth of *B. cinerea* recovered from diseased roses under laboratory conditions. An *in vivo* assay using cut rose flowers showed that this EO reduced the disease severity levels (below 1) similar to those in plants treated with the commercial fungicide carbendazim, encouraging the potential application of *P. graveolens* EO to manage *B. cinerea* in rose flowers. The main chemical compositions of the EO determined by gas chromatography-mass spectrometry (GC-MS) were geraniol (24.89%) followed by citronellol (19.50%) (Stegmayer et al. 2022). In a study by Herrera-Romero et al. (2017), *Aloe vera* L. pulp (25%) was used as a coating base, and natural antifungal agents such as chitosan (0.1%), thyme (0.1%), and oregano EOs (1.0%) were combined to manage post-harvest *Botrytis* blight infection in rose flowers. Nevertheless, none of the combinations inhibited the pathogen's development in rose flowers. There is a proposition that additional research is required to determine the best combinations of the applied compounds since the *A. vera* coating permitted natural bud opening and had no detrimental effects on rose blooms (Herrera-Romero et al. 2017). Three carbon and metal nanoparticles (NPs) were applied to evaluate their potential in reducing the *Botrytis* blight on roses *in vitro* and *in planta*. The results demonstrated that Fe_2O_3, CuO NPs, and multi-walled carbon nanotubes (MWCNTs) markedly inhibited the mycelial growth of *B. cinerea* at doses of 50, 100, and 200 mg/L *in vitro*, while reduced graphene oxide (rGO) at concentrations of 100 and 200 mg/L had the greatest inhibitory impact. The petal disc assay further showed that fullerene and CuO NPs (50 mg/L) were able to significantly reduce fungal development on the infected petal in such a way that the achieved level was lower compared to that obtained by applying the conventional fungicide procymidone (Hao et al. 2017).

RNAi-Based Bio-fungicides

RNAi-based bio-fungicides involve the application of double-stranded RNA (dsRNA) molecules to interfere with the expression of essential genes in *B. cinerea*, leading to its inhibition and eventual death. Consequently, this strategy results in a significant reduction in disease severity. These bio-fungicides exploit the natural RNA interference mechanism to target essential genes in the pathogen, offering an environmentally friendly and specific means of disease control (Islam and Sherif 2020). It is worth mentioning that RNAi-based bio-fungicides work by introducing small interfering RNAs (siRNAs) that specifically target genes crucial for the survival and pathogenicity of *B. cinerea* (Fig. 3). The delivery of dsRNA into target cells typically involves mechanisms such as endocytosis or direct penetration through the fungal cell wall. Endocytosis, a fundamental cellular process, involves the uptake of extracellular material by the cell through the formation of vesicles (Saleh et al. 2006). Research has shown that fungal cells, including those of *S. sclerotiorum*, can utilize endocytosis, particularly clathrin-mediated endocytosis (CME), a conserved pathway found in eukaryotic organisms, to internalize exogenous dsRNA molecules. This mechanism facilitates the uptake of dsRNA molecules, allowing them to enter the fungal cells and interact with the RNA interference machinery (Wytinck et al. 2020). Additionally, direct penetration through the fungal cell wall represents another potential route for dsRNA delivery into fungal cells. This mechanism provides an alternative pathway for dsRNA to bypass the cell wall barrier and gain entry into the fungal cytoplasm, where it can interact with the RNAi machinery to exert its biological effects (Šečić and Kogel 2021).

Once inside the pathogen, these siRNAs trigger the degradation of complementary messenger RNAs (mRNAs), thereby silencing the expression of

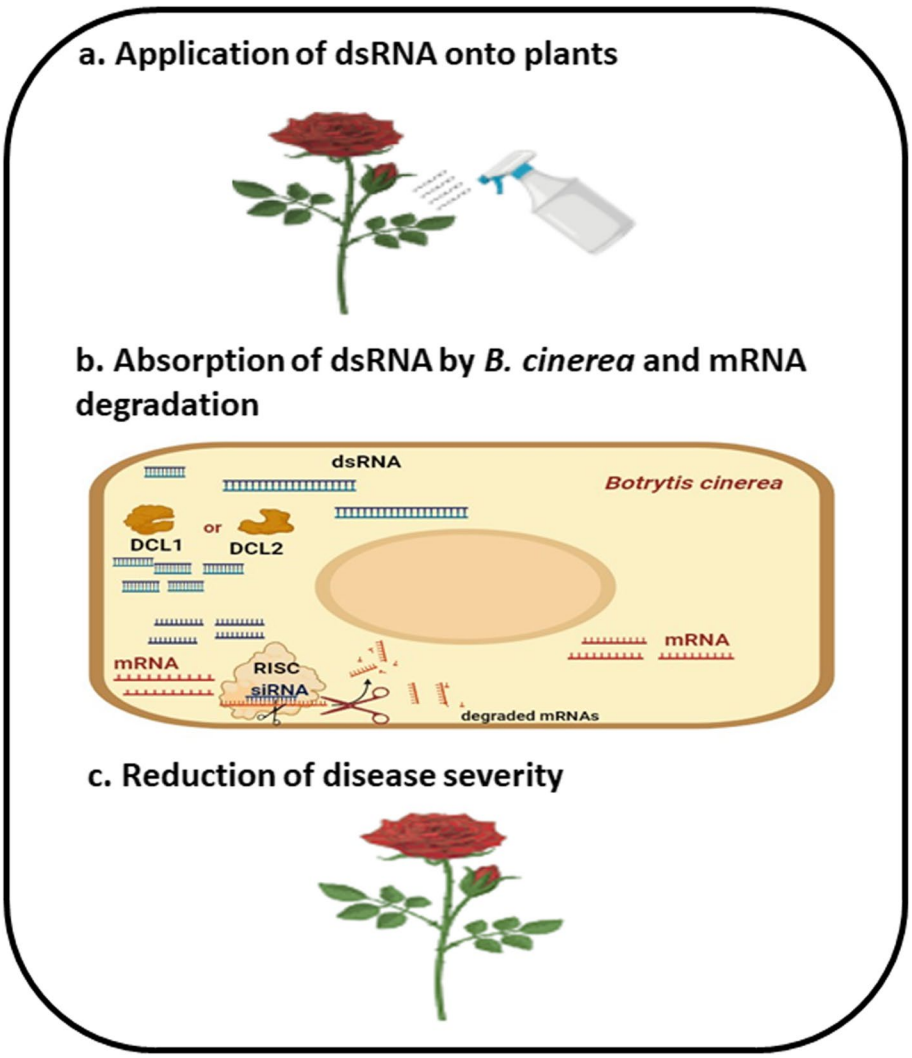

Fig. 3 RNAi-based bio-fungicides for *Botrytis cinerea* control in roses. **a** Application of RNAi construct. In the initial step, the RNAi-based bio-fungicide, designed to target *Botrytis cinerea*, is applied to the rose plants using a hand sprayer. The construct contains double-stranded RNA (dsRNA) molecules tailored to disrupt specific gene expression in the pathogen. **b** Absorption and mRNA degradation. Upon application, *B. cinerea* absorbs the dsRNA from the RNAi construct. Inside the fungal cells, the dsRNA is processed, leading to the degradation of target messenger RNA (mRNA). This interference disrupts vital gene expression in the pathogen, hindering its ability to cause infection. **c** Reduction of disease. The culmination of the RNAi mechanism results in a significant reduction in *B. cinerea*-induced disease symptoms. This innovative approach offers an effective and environmentally friendly strategy for controlling *Botrytis cinerea* in roses, showcasing the potential of RNAi-based bio-fungicides in sustainable plant disease management

fungal genes and disrupting the pathogen's normal physiological processes. One of the main advantages of RNAi-based bio-fungicides is their high specificity, targeting only the intended pathogen without affecting beneficial organisms or non-target species (Fig. 4). This specificity reduces the risk of off-target effects and environmental harm, making them safer alternatives to traditional chemical fungicides. This approach can target multiple central genes simultaneously, making it difficult for the pathogen to develop resistance compared to single-target fungicides. Previous studies demonstrated that exogenous application of siRNA and dsRNA targeting *Bc-DCL1* and *Bc-DCL2* silenced both genes, resulting in attenuating fungal pathogenicity and growth and gray mold diseases on strawberry and tomato fruits (Choquer et al. 2007; Wang et al. 2016). Additionally, topical applications of the dsRNAs that target *B. cinerea* genes encoding thioredoxin reductase and mitochondrial import inner membrane translocase subunit TIM44 led to fewer necrotic

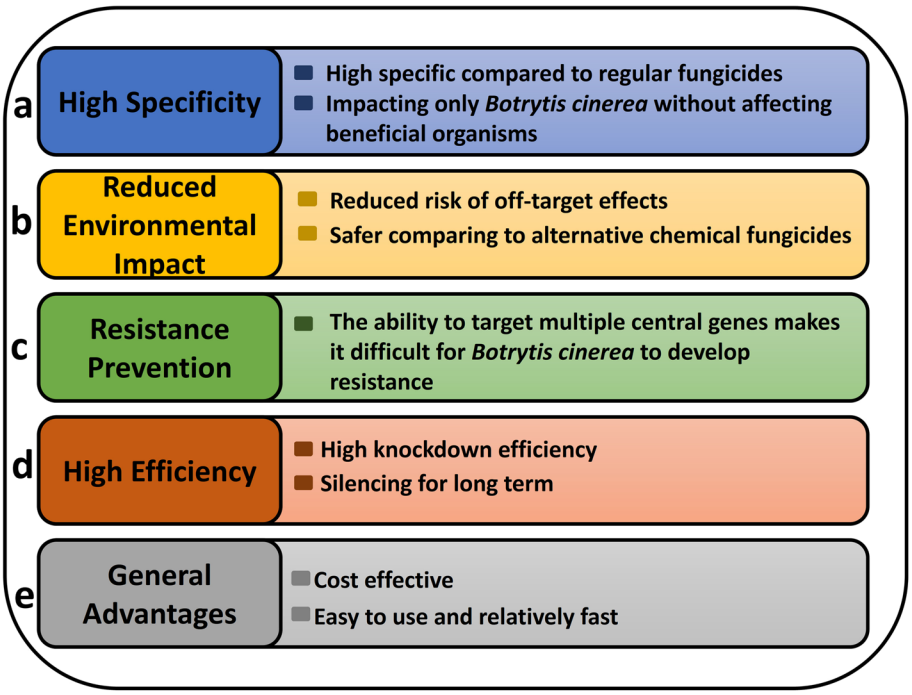

Fig. 4 Specificity and advantages of RNAi-based bio-fungicides in *Botrytis cinerea* management. This figure elucidates RNAi-based bio-fungicides' specificity and key advantages in mitigating *Botrytis cinerea*, a common pathogen affecting roses. **a** Precision targeting of *Botrytis cinerea*. The designed RNA molecules specifically target crucial genes in the pathogen, ensuring minimal impact on non-target organisms. **b** Reduced environmental impact in rose cultivation. Compared to traditional chemical fungicides, these bio-fungicides offer a more sustainable and eco-friendly alternative. The targeted nature of RNA interference minimizes the ecological footprint, contributing to a healthier and more balanced rose-growing ecosystem. **c** Resistance management strategies against *Botrytis cinerea*. The targeted mode of action reduces the likelihood of *B. cinerea* developing resistance. This property enhances the long-term sustainability of RNAi-based bio-fungicides as a reliable tool in integrated pest management programs for roses. **d** Promotion of enhanced rose health. By suppressing *B. cinerea*, these bio-fungicides contribute to improved rose vigor, flower quality, and overall plant health, fostering resilient and productive rose cultivation. **e** General advantages. RNAi-based bio-fungicides offer general advantages such as cost-effectiveness and ease of use, making them a practical and accessible solution for rose growers

lesions formation on detached leaves of oilseed rape (McLoughlin et al. 2018).

Concluding remarks and future directions

In conclusion, the effective management of gray mold in roses is a pivotal factor in maintaining these cherished plants' vitality and aesthetic appeal. Gray mold, caused by the fungal pathogen *B. cinerea*, can significantly impact the overall health and aesthetic value of rose gardens if left unchecked. A comprehensive and integrated approach is essential to effectively controlling and preventing the spread of this destructive disease. Chemical management, while an option, should be used judiciously and in combination with cultural practices. Fungicides specifically formulated to target *B. cinerea* should be applied preventatively or at the first sign of disease to optimize their efficacy. Cultural practices, on the other hand, assume a fundamental role in managing gray mold disease. Ensuring proper plant spacing, enhancing air circulation, and facilitating adequate sunlight exposure collectively foster an inhospitable environment for the pathogen to flourish. Routine pruning of dead or infected plant materials also helps reduce the disease's spread and provides a healthier environment for the roses. Furthermore, early detection and continuous monitoring of gray mold disease are paramount. Regular inspections of roses for telltale signs of infection, such as browning petals, fuzzy gray mold growth, and wilting, enable timely intervention and contain the disease's expansion. Educating gardeners and rose enthusiasts on the importance of disease prevention and management is crucial to safeguarding their beloved roses. Sharing information on identifying symptoms, implementing proper cultural practices, and using fungicides responsibly will empower individuals to take proactive measures to protect their plants. The integration of RNAi-based bio-fungicides into the arsenal of management strategies, complementing cultural practices, biological control agents, and conventional fungicides, plays a pivotal role within an integrated pest management (IPM) framework to bolster disease control (Fig. 5). However, the commercialization and adoption

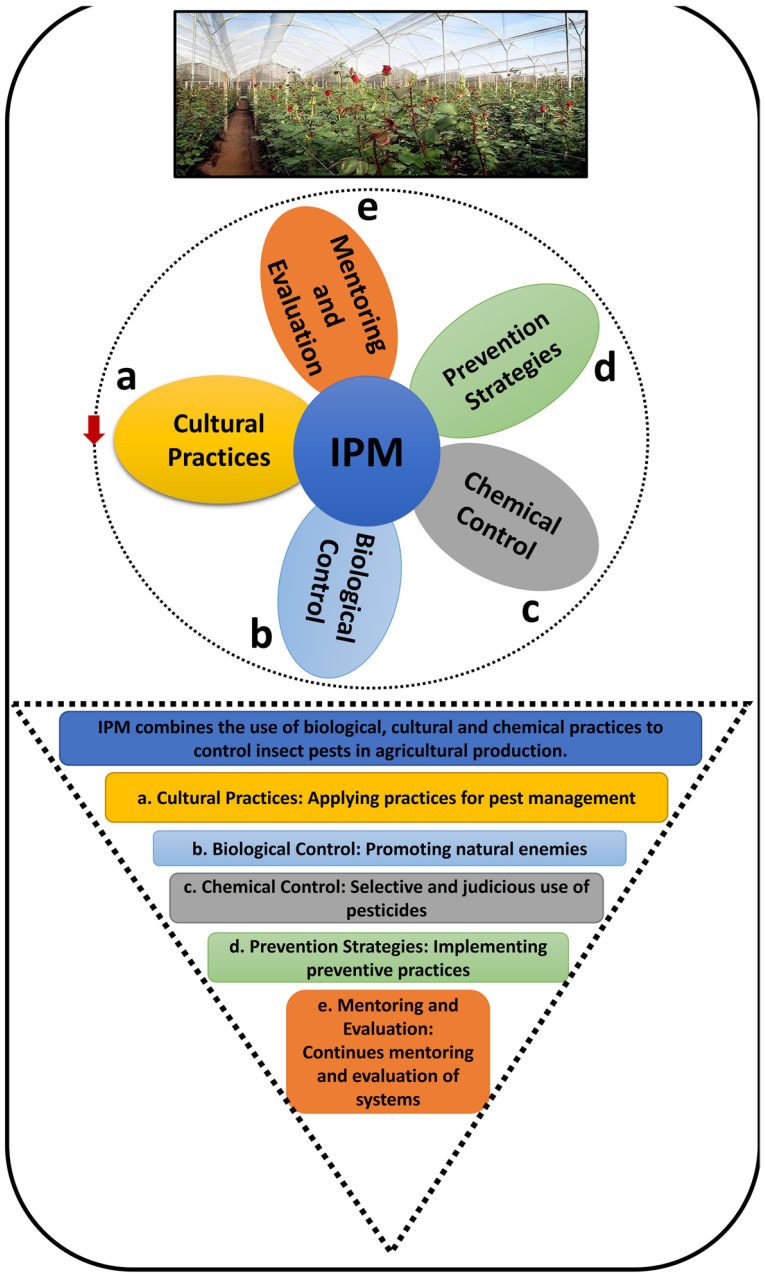

Fig. 5 Integrated pest management (IPM) in roses. This figure illustrates the holistic approach of integrated pest management (IPM) in cultivating roses, combining cultural, biological, chemical, and systematic practices for effective pest control. **a** Cultural practices. These include strategic choices in planting, irrigation, and overall crop management aimed at creating an environment that minimizes pest pressure. Culturally resilient rose varieties, proper spacing, and sanitation practices contribute to a robust foundation for pest management. **b** Biological control methods. Beneficial organisms, such as fungi and bacteria, play a crucial role in suppressing pest populations. **c** Chemical intervention. Carefully selected and applied pesticides are integrated into the management plan, considering their impact on non-target organisms and the environment. This strategic use of chemicals aims to control pest populations while minimizing negative consequences. **d** Prevention strategies. Proactive steps, such as regular scouting for pests and diseases, implementing quarantine practices, and employing resistant rose varieties, contribute to preventing potential pest outbreaks. This anticipatory approach reduces the reliance on reactive control measures. **e** Monitoring and evaluation. Regular assessments of pest populations, disease incidence, and the overall health of rose plants enable growers to make informed decisions. Continuous evaluation ensures the effectiveness of implemented strategies and allows for timely adjustments to optimize pest management

of RNAi-based bio-fungicides in agricultural practices necessitate strict adherence to regulatory guidelines to ensure their safety, efficacy, and environmental compatibility. In summary, staying abreast of the latest developments in gray mold management and embracing sustainable methodologies contribute to a more holistic and environmentally responsible approach to disease control.

Abbreviations

ETI	Effector-triggered immunity
PRR	Pattern-recognition receptors
PAMPs	Pathogen-associated molecular patterns
RLPs	Receptor-like proteins
MAPKs	Mitogen-activated protein kinases
CDPKs	Calcium-dependent protein kinases
BIK1	BOTRYTIS INDUCED KINASE1
PTI	PAMP-triggered immunity
ROS	Reactive oxygen species
SAR	Systemic acquired resistance
CWDEs	Cell wall- degrading enzymes

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Authors' contributions
IU, AG, and HW designed and conceptualized the study. FL, MK, MU, WY, AA, AG, and IU contributed to literature collection, methodology, software, and validation. IU, WY, HK, MK, AG, and HW wrote the original draft, review, and editing. IU, AA, and HK created the visualizations. AMG and HW supervised the study. HW secured funding. All authors have read and approved the published version of the manuscript.

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Availability of data and materials
All the data is presented in the main text.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
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Competing interests
The authors declare that they have no competing interests.

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