



Fusarium crown rot in wheat: advances in the biology, pathogenicity, and management of *Fusarium pseudograminearum*

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Abstract

Fusarium crown rot (FCR), predominantly caused by the soil-borne pathogen *Fusarium pseudograminearum*, poses a severe and escalating threat to global wheat (*Triticum aestivum* L.) production and food security. This review consolidates recent advances in understanding the pathogen's biology, infection strategies, host–pathogen interactions, and sustainable management practices. *F. pseudograminearum* deploys trichothecene mycotoxins, particularly deoxynivalenol (DON), to undermine host immunity, with its virulence governed by transcription factors and functional proteins involved in fungal growth, toxin biosynthesis, and stress adaptation. In response, wheat initiates a multilayered defense mechanism, primarily by upregulating genes related to toxin detoxification and transport, hormonal signaling, and structural reinforcement. Integrated disease management (IDM) practices aimed at mitigating disease severity include agronomic interventions, fungicides, biocontrol agents, and breeding efforts supported by quantitative trait locus (QTL) mapping, genome-wide association (GWAS) studies, and genome editing. To address the dynamic nature of *F. pseudograminearum* populations under changing environmental conditions, future directions should prioritize climate-resilient strategies, integrated multi-omics approaches, drone-based early detection technologies, and global pathogen surveillance systems. This review provides an updated framework to guide sustainable and effective FCR control.

Keywords *Fusarium pseudograminearum* · Fusarium crown rot · Trichothecene mycotoxins · Integrated disease management

1 Introduction

Wheat (*Triticum aestivum* L.) is the most widely cultivated cereal globally, playing a pivotal role in ensuring food security. Among the major damaging diseases threatening wheat production, Fusarium crown rot (FCR), primarily caused by *Fusarium pseudograminearum*, persistently impairs yield and grain quality (Scott and Chakraborty 2006). The pathogen has a global distribution includes Australia (Akinsanmi

et al. 2004; Klein et al. 1991), U.S. Pacific Northwest (Cook 1980; Poole et al. 2012; Smiley et al. 2005a), China (Fan et al. 2021; Ji et al. 2016a; Li et al. 2012), South America (Castañares et al. 2012), the Middle East (Alkadri et al. 2013; Hameed et al. 2012; Shikur Gebremariam et al. 2018), and Northern and Southern Africa (Abdallah-Nekache et al. 2019; Gargouri et al. 2011; Kammoun et al. 2009; Lamprecht et al. 2006). Climate changes and shifts in agronomic practices have further intensified its impact (Poole et al. 2013; Xu et al. 2018).

FCR is of particular concern due to two characteristics: its persistence in crop residues and the production of harmful secondary metabolites (SMs), notably the trichothecene mycotoxins deoxynivalenol and nivalenol (NIV), which contaminate the grains and enter the food chain (Blum et al. 2019). Currently, no wheat cultivars exhibit robust resistance or immunity, and the efficacy of chemical control is diminishing due to fungicide resistance (Moya-Elizondo and Jacobsen 2016; Rahman et al. 2021; Xu et al. 2025). While regional reviews (Li et al. 2022) have addressed FCR

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in specific geographies, no comprehensive global analysis has integrated recent advances in pathogen genomics, host resistance mechanisms, or climate-adaptive management since the 2018 review by Kazan and Gardiner (Kazan and Gardiner 2018). Our review fills this five-year gap by synthesizing breakthroughs across disciplines, from CRISPR-enabled breeding to AI-driven pathogen surveillance, and proposes a scalable framework that prioritizes climate resilience, thereby offering a timely roadmap for future research and sustainable control.

2 Global distribution and economic impact of FCR

Fusarium species significantly reduce wheat yield and quality worldwide (Matny 2015). In Australia, *F. pseudograminearum* reduces bread wheat yield by an average of 25% and durum wheat yields by up to 58%, translating to annual losses of nearly \$80 million (Murray and Brennan 2009). In the U.S. Pacific Northwest, winter wheat yield losses has reached up to 61% (Li et al. 2022; Smiley et al. 2005b). This rotation system has been particularly conducive to the spread of FCR in major wheat growing regions. The disease was first observed in Henan Province wheat maize rotation fields in 2011 (Li et al. 2012). Subsequently, this disease has spread widely across the Huang-Huai and East China wheat regions, leading to significant yield reductions and quality deterioration (Ji et al. 2016b; Xu et al. 2016; Zhao et al. 2022c). According to statistics, from 2013 to 2016, yield losses due to FCR in Henan Province ranged from 38.0% to 61.3% (Xu et al. 2016). During 2017–2021, the average annual wheat yield loss in Henan was 36,000 tonnes, reaching up to 44,000 tonnes in the worst year (Zhao et al. 2022c). In 2015, a survey in central and southern Hebei Province revealed that nearly 50% of fields with FCR had white head rates of 1–10%, and severely affected fields exhibited patches of completely white heads (Ji et al. 2016b). In 2020, the affected area in Shandong Province reached 0.8 million hectares (Li 2021). These regional variations highlight the differential impact of FCR across wheat-growing areas in China.

Beyond yield impacts, FCR poses substantial risks to food and feed safety. The stable and highly toxic mycotoxins produced by *F. pseudograminearum*, contaminate grains, straw, and forage (Champeil et al. 2004; Pestka 2010), and persist through environmental conditions and food processing. These mycotoxins cause acute and chronic toxicity in humans and animals by inhibiting protein and DNA synthesis, disrupting ribosomal and mitochondrial function, and inducing immunosuppression and DNA damage (Ueno et al. 1973). High humidity and rainfall further increase FCR incidence by enhancing spore dispersal and fungal colonization of

crop residues, elevating the risk of mycotoxin contamination (Champeil et al. 2004).

3 Disease symptoms and diagnosis

FCR symptoms caused by *F. pseudograminearum* begin with browning at the crown and basal stem nodes. As infection progresses, discoloration ascends to the third to fifth internodes, often producing the diagnostic “soy sauce stick” appearance. Pink or white fungal mycelia often colonize the central pith of affected internodes, while roots exhibit brown necrosis and rot, rendering internodal tissues fragile and prone to breakage. At the heading stage, severely infected plants may desiccate prematurely, producing white heads and shriveled or aborted grains (Kazan and Gardiner 2018; Mudge et al. 2006; Zhang et al. 2022b).

Accurate FCR diagnosis is complicated by symptom overlap with other *Fusarium* spp. and soilborne pathogens. FCR symptoms caused by *F. pseudograminearum* often overlap with those induced by other *Fusarium* spp., including *F. culmorum*, *F. graminearum*, *F. equiseti*, *F. acuminatum*, *F. avenaceum*, *F. oxysporum*, *F. proliferatum*, *F. solani*, *F. tricinctum*, *F. brachygibbosum*, *F. hostae*, *F. redolens*, *F. torulosum*, *F. flocciferum*, *F. incarnatum*, *F. reticulatum*, as well as other soilborne pathogens like *Bipolaris sorokiniana*, *Gaeumannomyces graminis*, and *Pythium* spp. (Kazan and Gardiner 2018; Li et al. 2022; Shikur Gebremariam et al. 2018). To address this diagnostic challenge, molecular tools such as a TaqMan qPCR assay targeting the *FpAH1* gene enable rapid and specific differentiation of *F. pseudograminearum* from other *Fusarium* spp. This assay, validated against 12 *Fusarium* species and 10 other wheat pathogens, demonstrates high specificity and allows precise quantification of pathogen biomass in plant and soil samples (Yan et al. 2026). Field monitoring over two growing seasons further revealed pathogen dynamics under different seed treatments, underscoring its practical value for disease management. Compared to *F. graminearum*, *F. pseudograminearum* infections result in more severe reductions in root length, root dry weight, leaf number, shoot biomass, and leaf area (Saad et al. 2023). Notably, *F. pseudograminearum* thrives particularly under warm, dry environmental conditions, distinguishing its epidemiology from other *Fusarium* pathogens and leading to regionally variable disease dynamics (Chakraborty et al. 2006; Poole et al. 2012).

4 Biology and lifecycle of *F. Pseudograminearum*

Previously grouped with *F. graminearum*, *F. pseudograminearum* is now recognized as a distinct species based on DNA and isozyme analyses (Aoki and O'Donnell 1999; Francis and

Burgess 1977; Laday et al. 2000). The telomorphic form of *F. pseudograminearum*, *Gibberella coronicola*, possesses two mating types (MAT-1 and MAT-2) and exhibits a hemibiotrophic lifestyle. Unlike *F. graminearum*, *F. pseudograminearum* typically produces few or no perithecia in culture (Xie et al. 2022). Despite substantial genomic similarity, chromosomal rearrangements, especially at the telomeric regions, render *F. pseudograminearum* genetically distinct (Gardiner et al. 2018).

The lifecycle of *F. pseudograminearum* includes survival in soil and crop residues as mycelia, conidia, and chlamydospores, with viability exceeding two years. Overwintering primarily occurs in infected debris, where mycelia and conidia serve as major inocula for the following growing season (Bragard et al. 2022; Kazan and Gardiner 2018). While chlamydospores are rarely observed under field conditions, the exact infection sites on wheat remain poorly defined and likely depend on spatial distribution in soil (Aoki and O'Donnell 1999; Li et al. 2022).

5 Infection process of *F. Pseudograminearum*

F. pseudograminearum can infect wheat at all developmental stages (Li et al. 2012). However, whether infection rates differ significantly across specific growth stages remains to be systematically investigated. Post-germination, seedlings can be invaded by soilborne spores or mycelia near the seed level, leading to seedling blight and root necrosis (Li et al. 2012). More commonly, infection initiates through coleoptile sheaths, progressing to root and leaf sheaths before reaching stem epidermal tissues (Knight and Sutherland 2016).

Following penetration, *F. pseudograminearum* spreads through stomata or intercellular spaces to colonize hypodermal and vascular tissues. It subsequently invades the stem via thin-walled pith cells and the central pith cavity, with rapid colonization along subepidermal and parenchymal tissues (Mudge et al. 2006). Early infection is primarily intercellular, but intracellular colonization becomes more prominent in later stages. In prematurely senescing stems, the pathogen enters xylem vessels and later invades the phloem, severely disrupting water and nutrient transport, a key factor in the formation of the hallmark "white head" symptom (Knight et al. 2017) (Fig. 1).

6 Mechanism of *F. Pseudograminearum* infection

6.1 Transcription factors involved in infection

The pathogenicity of *F. pseudograminearum* is orchestrated by a network of transcription factors and functional proteins

that regulate growth, toxin biosynthesis, and stress adaptation through complex molecular networks. The transcription factors implicated in virulence regulation include the Cys2 Zinc-Finger (CZF) proteins FpCzf14 and FpCzf7, which modulate conidiation and mycotoxin synthesis. FpCzf14 is essential for conidia formation (Chen et al. 2020b), whereas FpCzf7 regulates deoxynivalenol (DON) biosynthesis (Zhao et al. 2022b). The Zn2Cys6-type transcription factor Fp487 balances fungal growth and pathogenicity (Yang et al. 2024b). APSES-family transcription factors, such as FpAPSES1 and FpAPSES4, are involved in vegetative growth, conidiation, and virulence, with expression peaking during host colonization (Zhao et al. 2021). Likewise, basic helix-loop-helix (bHLH) transcription factors (*e.g.*, FpbHLH9) are crucial for infectivity. The loss of FpbHLH9 significantly reduces pathogenicity in susceptible wheat (Chen et al. 2019b). Basic leucine zipper (bZIP) proteins like Fpkapc regulate critical cellular functions, including carbon metabolism, mitosis, ion transport, and stress responses (Zhao et al. 2022a), whereas FpAda1 governs hyphal extension, conidiation, pathogenicity, and cell cycle progression (Chen et al. 2020a).

6.2 Functional proteins and cellular pathways

Functional proteins associated with organelle activity and cellular metabolism further modulate infection dynamics. Peroxisome biosynthetic receptor proteins FpPEX5 and FpPEX7 mediate fatty acid metabolism and secondary metabolite transport, thereby influencing growth and virulence (Bi et al. 2024). Endoplasmic reticulum (ER) luminal heat shock protein 70 s (Hsp70s), FpLhs1, are vital for mycelial development and spore generation (Chen et al. 2019a). Similarly, FpHsp104 supports thermotolerance, growth, and virulence (Xia et al. 2021).

Programmed cell death (PCD) and autophagy pathways also modulate fungal development and infection. The apoptosis inhibitor gene *FpBIR1* and the apoptosis-promoting gene *FpNUC1* regulate spore germination and early infection (Chen et al. 2021). The autophagy-related gene *FpAtg3* governs growth and stress resilience, while also interacting with hyphal fusion proteins such as FpHam-2, FpHam-3, and FpHam-4 (Chen et al. 2024; Dong et al. 2024). Disruption of these fusion-related proteins impairs conidiation and reduces virulence, underscoring a functional link between autophagy and pathogenic development (Chen et al. 2024).

6.3 Virulence factors: the role of secondary metabolites

F. pseudograminearum employs a range of secondary metabolites (SMs) to subvert host defenses and establish infection. Among these, DON and its acetylated derivatives

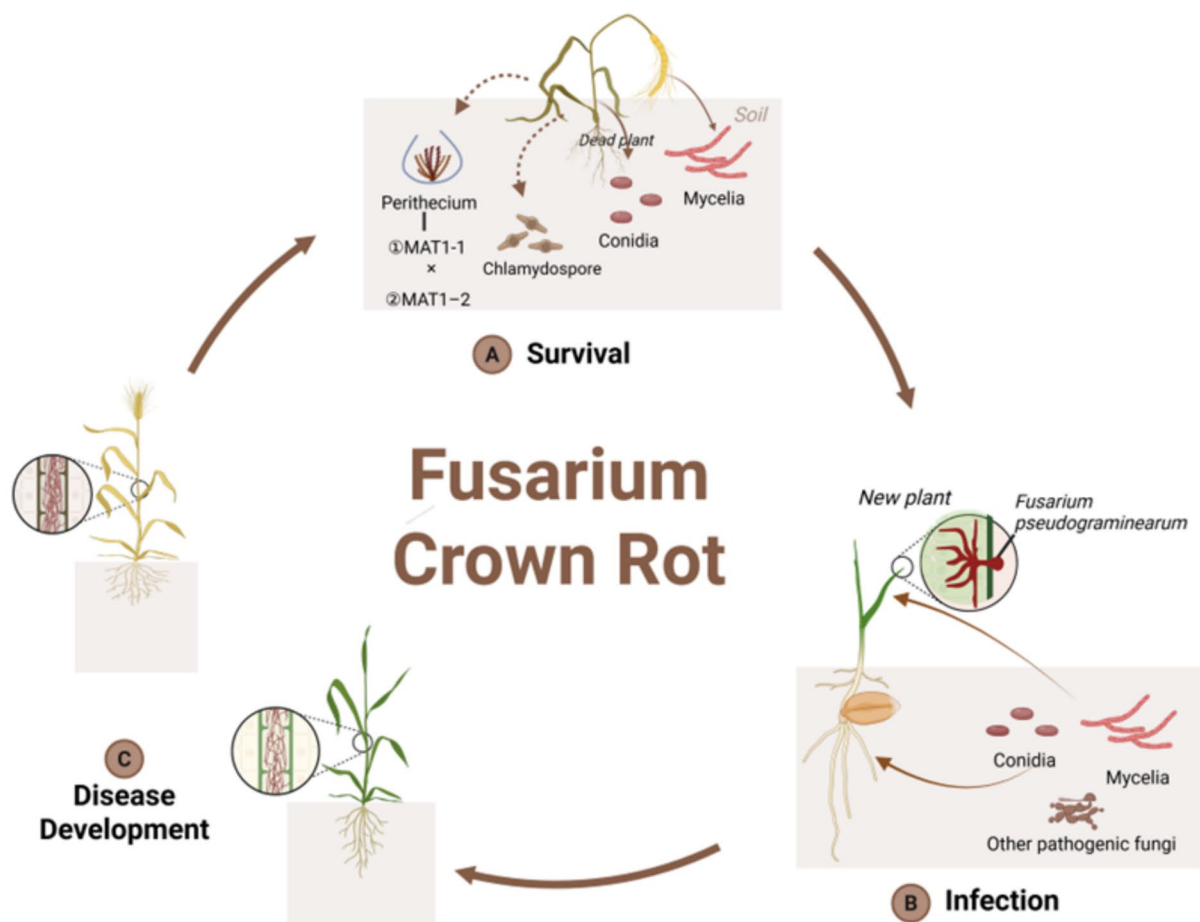


Fig. 1 Life cycle and pathogenic progression of *Fusarium pseudograminearum*. *a*, Survival phase: the pathogen persists in soil and crop residues as mycelia, conidia, and chlamydospores. Under certain conditions, it forms sexual structures (MAT1-1 and MAT1-2), enhancing genetic diversity and environmental resilience. *b*, Infection

phase: conidia and hyphae germinate and penetrate wheat via roots, stem bases, or wounds, often in conjunction with other pathogens. *c*, Disease development phase: colonization of vascular tissues leads to stem browning, upward hyphal spread, “whitehead” formation, and plant death

3ADON and 15ADON are central FCR virulence factors (Mudge et al. 2006). DON facilitates systemic colonization by acting as a mobile virulence factor that enables the fungus to migrate through vascular tissues (Khudhair et al. 2021, 2019). Concentrations of DON are typically higher in the lower stem than in the apical regions, and its production is significantly elevated during infection compared to saprophytic growth (Tunali et al. 2012). Targeted deletion of *Tri5*, a key gene in DON biosynthesis, inhibits hyphal spread through stem tissues, underscoring DON’s essential role in systemic infection (Mudge et al. 2006). Additional SM classes of virulence factors, such as polyketides (PKs) and non-ribosomal peptides (NRPs), further intensify disease progression (Hansen et al. 2015).

Regulation of DON and other SMs occurs via multilayered networks. The global regulator FpLaeB orchestrates cell wall remodeling, membrane integrity, carbohydrate

metabolism, and SM biosynthesis. Deleting FpLaeB results in a marked reduction in DON levels (Wu et al. 2023a). *FpRCOI*, a chromatin regulator, modulates the trichothecene biosynthetic gene (*TRI*) gene cluster through a histone-modifying complex, controlling toxin production at the epigenetic level (Li et al. 2024b). Intriguingly, knockout of the bZIP transcription factor Fpkapc enhances *TRI* gene expression without significantly altering DON titers, suggesting the existence of compensatory regulatory mechanisms (Zhao et al. 2022a). Genome-wide association studies have revealed that variations in mycotoxin chemotypes (*e.g.*, 3AcDON vs. 15AcDON) are driven by SNPs in *SM* gene clusters, implying adaptive evolution in response to diverse host environments (Li et al. 2024d).

Beyond DON, non-*TRI*-associated SMs also contribute to virulence. Genomic surveys predict at least 16 NRPS and 14 PKS gene clusters in *F. pseudograminearum*, indicating

a broad potential for secondary metabolite biosynthesis (Hansen et al. 2015). For example, the lipopeptide W493B, synthesized by *NRPS32*, may modulate host interactions despite not directly influencing virulence (Sørensen et al. 2014). The transcription elongation factor FpRtfA modulates PKS gene expression and is required for full virulence (Wu et al. 2023b). Similarly, the velvet family protein FpVelB coordinates the expression of *TRI* and *PKS11*, thereby integrating DON and polyketide pathways (Wu et al. 2024). The *FpPPR1* gene, encoding a pentatricopeptide repeat protein, regulates expression of *PKS12* and associated yellow pigmentation, linking visible traits with metabolic functions (Wang et al. 2021).

6.4 Co-evolution of pathogenicity and virulence factors

F. pseudograminearum orchestrates infection through a temporally synchronized deployment of pathogenicity and virulence factors. Transcriptional networks integrate growth and metabolism to coordinate these processes during host colonization.

During the initial infection stage, the pathogen secretes cell wall-degrading enzymes, along with DON to breach plant defenses (Beccari et al. 2011). In the mid to late stages, extensive mycelial proliferation and increased SM production accelerate host tissue degradation and systemic invasion (Mudge et al. 2006).

This multi-phase strategy reflects a sophisticated adaptation that balances resource acquisition with evasion of host immunity. Targeting pivotal regulatory nodes, such as chromatin-based control of *TRI* cluster activity, may offer novel routes for controlling infection (Table 1).

7 Host defense mechanisms in wheat and counter-defense mechanisms in *F. Pseudograminearum*

Wheat deploys layered defenses against FCR involving temporal and spatial modulation of gene networks and metabolic pathways. Upon pathogen detection, resistant cultivars rapidly activate a suite of defense-related genes. Comparative transcriptomics between the resistant cultivar Sunco and the susceptible cultivar Kennedy revealed differential activation of genes associated with antimicrobial responses, stress adaptation, and signal transduction (Desmond et al. 2005). This differential expression, particularly at early stages of infection, is critical for effective resistance. This early recognition is physically reinforced at the cell wall interface; for instance, the receptor-like kinase TaRLK-6A interacts with the co-receptor TaSERK1 to amplify pathogen perception, while TaWAK-5D600

directly fortifies the cell wall barrier by activating downstream defense genes (Qi et al. 2023; Zhou et al. 2025).

Subsequently, a cascade of hormonal signaling networks orchestrates systemic resistance, with jasmonic acid (JA), salicylic acid (SA), cytokinin (CK), abscisic acid (ABA), and auxin (IAA) serving as key hubs in this process (Bari and Jones 2009). Proteomic analyses further confirm the central role of these hormones in this signaling phase. For instance, the JA biosynthesis gene TaOPR3 exemplifies the dual role of hormonal pathways in promoting both immunity and growth (Gao et al. 2023). The delicate balance among these hormones is critical to their function: exogenous application of methyl jasmonate enhances resistance by amplifying JA signaling, while treatment with the cytokinin 6-benzylaminopurine (BAP) compromises defense by disrupting hormonal homeostasis, underscoring the spatial and temporal sensitivity of this balance (Ahmed et al. 2014). Additionally, auxin (IAA) levels are tightly regulated through the tryptophan pathway, a crucial metabolic bridge linking defense responses with stress adaptation; intermediates of this pathway, including melatonin and indoleacetaldehyde, modulate IAA levels (Arnao and Hernández-Ruiz 2006; Sun et al. 2021), and aldehyde dehydrogenase catalyzes the conversion of indoleacetaldehyde to IAA—moderate IAA levels reinforce cell wall integrity, while excessive accumulation weakens host defenses (Abu-Zaitoon et al. 2019). The transcription factor TaWRKY24 precisely modulates this hormonal interface by limiting IAA overproduction while simultaneously enhancing SA-mediated responses, thereby contributing to broad-spectrum resistance (Xu et al. 2024).

Downstream of these signaling events, a coordinated arsenal of metabolic and detoxification enzymes is deployed. Genes encoding transporters and detoxification enzymes, such as ATP-binding cassette (ABC) transporters and UDP-glucosyltransferases, act in concert to mitigate fungal spread (Powell et al. 2017).

F. pseudograminearum evades plant defenses by subtelomeric chromosome rearrangements that alters gene clusters that detoxify the plant toxin benzoxazolin-2-one (BOA). Infection significantly induces wheat production of antimicrobial benzoxazinoids such as BOA and 6-methoxy-benzoxazolin-2-one (MBOA), both of which inhibit fungal growth under in planta and in vitro conditions (Duan et al. 2022). However, *F. pseudograminearum* neutralizes these compounds via the *FDB1* (γ -lactamase) and *FDB2* (N-malonyltransferase) gene clusters, located at subtelomeric regions of its genome (Kettle 2016). These degradation pathways underscore the pathogen's capacity to adapt to host chemical defenses, exemplifying the dynamic molecular "arms race" between wheat and *F. pseudograminearum* (Fig. 2).

Table 1 Summary of key virulence factors associated with infection and pathogenicity in *Fusarium pseudograminearum*

Functional category	Factor name	Molecular/structural feature	Core Function in Infection and Pathogenicity	References
Transcriptional, Epigenetic and Global Regulators	FpCzf14	Cys2 Zinc-Finger (CZF) protein	Essential for conidiation, a core process for fungal propagation and infection initiation	Chen et al. (2020b)
	FpCzf7	Cys2 Zinc-Finger (CZF) protein	Regulates deoxynivalenol (DON) biosynthesis, a key virulence determinant	Zhao et al. (2022b)
	Fp487	Zn ₂ Cys ₆ -type transcription factor	Balances fungal vegetative growth and pathogenicity	Yang et al. (2024b)
	FpAPSES1	APSES-family transcription factor	Modulates vegetative growth, conidiation, and virulence; expression peaks during host colonization	Zhao et al. (2021)
	FpAPSES4	APSES-family transcription factor	Modulates vegetative growth, conidiation, and virulence; expression peaks during host colonization	Zhao et al. (2021)
	FpbHLH9	Basic helix-loop-helix (bHLH) transcription factor	Critical for fungal infectivity; gene deletion significantly reduces pathogenicity in susceptible wheat	Chen et al. (2019b)
	Fpkapc	Basic leucine zipper (bZIP) transcription factor	Regulates carbon metabolism, mitosis, ion transport, and stress responses; knockout enhances trichothecene biosynthetic (TRI) gene expression with no significant changes in DON titer	Zhao et al. (2022a)
	FpAda1	Basic leucine zipper (bZIP) transcription factor	Governs hyphal extension, conidiation, pathogenicity, and cell cycle progression	Chen et al. (2020a)
	FpRttA	Transcription elongation factor	Modulates polyketide synthase (PKS) gene expression; required for full fungal virulence	Wu et al. (2023b)
	FpLaeB	Global transcriptional regulator	Orchestrates cell wall remodeling, membrane integrity, carbohydrate metabolism, and secondary metabolite (SM) biosynthesis; deletion results in marked reduction of DON accumulation	Wu et al. (2023a)
	FpRCO1	Chromatin regulator	Modulates the TRI gene cluster via histone-modifying complex, controls mycotoxin production at the epigenetic level	Li et al. (2024b)
	FpVelB	Velvet family protein	Coordinates expression of the TRI cluster and PKS11, integrates DON and polyketide biosynthetic pathways	Wu et al. (2024)
	FpPPR1	Pentatricopeptide repeat (PPR) protein	Regulates PKS12 expression and associated yellow pigmentation, links phenotypic traits with metabolic functions	Wang et al. (2021)

Table 1 (continued)

Functional category	Factor name	Molecular/structural feature	Core Function in Infection and Pathogenicity	References
Functional Proteins and Core Cellular Pathway Components	FpPEX5 and 7	Peroxisome biosynthetic receptor protein	Mediates fatty acid metabolism and secondary metabolite transport, thereby influencing fungal growth and virulence	Bi et al. (2024)
	FpLhs1	Endoplasmic reticulum (ER) luminal heat shock protein 70 (Hsp70)	Vital for mycelial development and conidiation	Chen et al. (2019a)
	FpHsp104	Heat shock protein	Supports fungal thermotolerance, vegetative growth, and virulence	Xia et al. (2021)
	FpBIR1	Apoptosis inhibitor	Regulates spore germination and early host infection, along with the pro-apoptotic factor FpNUC1	Chen et al. (2021)
	FpNUC1	Pro-apoptotic factor	Regulates spore germination and early host infection, along with the anti-apoptotic factor FpBIR1	Chen et al. (2021)
	FpAtg3	Autophagy-related protein	Governs fungal growth and stress resilience; interacts with hyphal fusion proteins FpHam-2/3/4	Dong et al. (2024)
	FpHam-2,3,4	Hyphal fusion-related protein	Modulates conidiation and virulence; functionally linked to the autophagy pathway	Chen et al. (2024)
Secondary Metabolite Virulence Factors and Biosynthesis Components	DON (3ADON, 15ADON)	Trichothecene mycotoxin	Core virulence factor for FCR; acts as a mobile virulence factor to facilitate systemic fungal colonization via vascular tissues	Khudhair et al. (2019), Mudge et al. (2006), Tunali et al. (2012)
	PKs	Polyketide secondary metabolites	Virulence factors that intensify disease progression	Hansen et al. (2015)
	NRPs	Non-ribosomal peptide secondary metabolites	Virulence factors that intensify disease progression	Hansen et al. (2015)
	W493B	Lipopeptide (non-ribosomal peptide)	Modulates host-fungus interactions, despite no direct effect on virulence	Sørensen et al. (2014)
	<i>Tri5</i>	Key enzyme in DON biosynthetic pathway	Core enzyme for DON biosynthesis; targeted deletion inhibits hyphal spread through stem tissues, confirming its essential role in systemic infection	Mudge et al. (2006)
	<i>NRPS32</i>	Non-ribosomal peptide synthetase	Catalyzes biosynthesis of the lipopeptide W493B, modulates host-fungus interactions	Sørensen et al. (2014)
	Summary of major transcription factors, functional proteins, and secondary metabolites associated with fungal growth, stress adaptation, host colonization, and virulence regulation during infection			

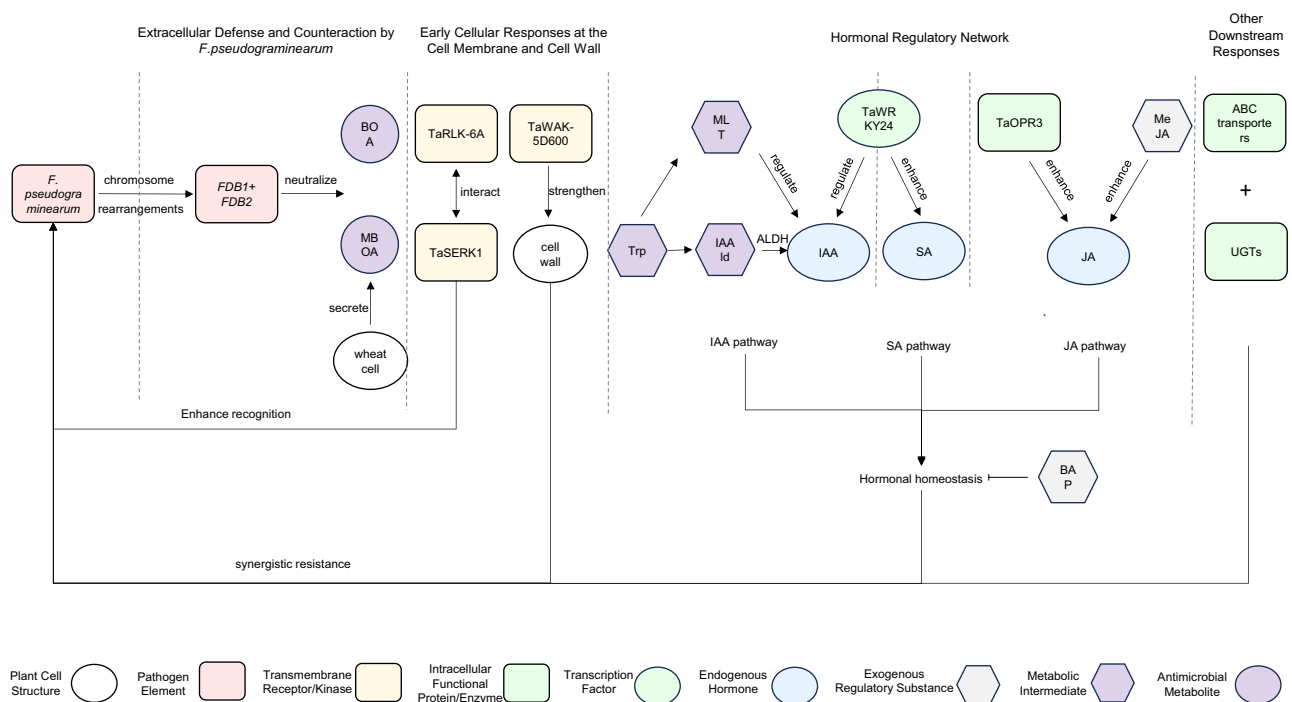


Fig. 2 Schematic diagram of the multi-layered defense mechanism of wheat against *F.pseudograminearum* infection. This schematic systematically depicts the complete process of wheat defense response to *F. pseudograminearum* infection, which is organized into four sequential, functionally linked core modules labeled a to d: a, early cellular recognition and cell wall barrier reinforcement at the cell wall-cell membrane interface; b, intracellular hormonal regulatory network and key pathway crosstalk; c, execution of downstream defense responses and secretion of antimicrobial compounds; d, counter-defense and immune evasion mechanisms of *F. pseudograminearum*. ABA Absciscic acid, ABC transporters ATP-binding cassette transporters, ALDH Aldehyde Dehydrogenase, BAP

6-benzylaminopurine (exogenous synthetic cytokinin analog), BOA Benzoxazolin-2-one, CK Cytokinin, FDB1 Fungal Detoxification 1 (γ -lactamase), FDB2 Fungal Detoxification 2 (N-malonyltransferase), IAA Indole-3-acetic acid, IAAld Indoleacetaldehyde, JA Jasmonic acid, MBOA 6-methoxy-benzoxazolin-2-one, MeJA Methyl jasmonate (exogenous jasmonic acid analog), MLT Melatonin, SA Salicylic acid, TaOPR3 *Triticum aestivum* 12-Oxophytodienoate Reductase 3, TaRLK-6A *Triticum aestivum* Receptor-Like Kinase 6A, TaSERK1 *Triticum aestivum* Somatic Embryogenesis Receptor Kinase 1, TaWAK-5D600 *Triticum aestivum* Wall-Associated Kinase 5D600, TaWRKY24 *Triticum aestivum* WRKY Transcription Factor 24, Trp Tryptophan, UGTs UDP-glucosyltransferases

8 Progress in research on prevention and control technologies

8.1 Stubble and tillage management

Optimizing soil management and adopting strategic crop rotation are fundamental to managing FCR. *F. pseudograminearum* can persist in crop residues for extended periods, making ecological field management essential (Summerell et al. 1990). A well-designed crop rotation disrupts the pathogen life cycle and improves soil health.

FCR severity increases with continuous wheat cultivation, particularly in maize–wheat rotations, such as in northern China (Xu et al. 2018). Introducing non-host crops, such as sorghum and oats, effectively reduces pathogen loads (Burgess 2005). Rotating *Brassica* crops, including rapeseed and mustard, with durum wheat not only lowers disease incidence but also boosts yield in northern NSW. The dense canopy of these crops accelerates residue

decomposition (Kirkegaard et al. 2004). Legumes, such as chickpeas and lupins, also have disease-suppressive effects (Felton et al. 1998; Theron et al. 2023). Interestingly, alternating summer watermelon with winter wheat significantly reduced FCR, although the underlying mechanisms remain unclear (Li et al. 2022). These findings underscore the value of crop diversification in disrupting pathogen cycles.

Stubble management is equally critical for disease suppression (Dodman and Wildermuth 1987). Conservation tillage methods, such as straw incorporation and no-till seeding, preserve soil integrity but can promote pathogen accumulation (Cook et al. 1980; Shrestha et al. 2021). However, combining no-till systems with non-host rotations helps retain soil structure while suppressing FCR (Theron et al. 2023). Traditional deep plowing remains effective, burying infected residues and interrupting fungal survival (Paulitz et al. 2002).

Environmental conditions also influence disease dynamics. Warm, humid weather favors infection (Buster et al.

2022), while drought stress enhances pathogen survival (Liu and Liu 2016). This paradox highlights the importance of precise water management. Elevated atmospheric CO₂ can increase biomass and residue, potentially facilitating pathogen spread (Melloy et al. 2010). Excessive nitrogen accelerates growth but elevates disease risk (Paulitz 2006), whereas zinc supplementation mitigates pathogen invasion (Grewal et al. 1996).

Specific agronomic interventions directly reduce disease pressure. Delayed sowing minimizes overlap between the wheat's susceptible stage and peak pathogen activity (Shah et al. 2018). Precision irrigation—especially during key stages like greening, jointing, and grain filling—supports plant development while limiting pathogen advantage (Luck et al. 2011). Collectively, these practices create a resilient cropping system by enhancing soil quality, regulating microclimates, and fortifying host resistance.

Advances in molecular biology hold promise for next-generation tools that enable early detection of FCR and precision interventions (Nkongolo and Narendrula-Kotha 2020).

8.2 Integration strategies

Due to the lack of fully resistant wheat varieties, chemical control remains primary for FCR management. Current approaches include seed treatment, foliar fungicide application during green-up, or both. Seed dressings formulated with combinations such as tebuconazole, pyraclostrobin, difenoconazole-fludioxonil, and azoxystrobin-fludioxonil have proven effective in suppressing early-stage infection (Akgül and Erkilic 2016). A novel seed treatment, trifluoropyridinamine, has recently shown potent antifungal activity by inhibiting mitochondrial function, thereby suppressing respiration. (Wang et al. 2023). During crop development, foliar applications of pyraclostrobin, prothioconazole, tebuconazole, and phenamacril reduce FCR severity (Akgül and Erkilic 2016; Li et al. 2023c). When applying fungicides, focusing on root zones rather than solely foliar surfaces enhances efficacy and limits the spread. (Lamichhane et al. 2020).

However, overreliance on fungicides risks resistance development and environmental contamination. Continuous monitoring of pathogen populations is therefore critical, as the widespread and repeated use of fungicides over the years has been the primary driver for the emergence of resistant isolates. Indeed, resistance has been reported against multiple classes of fungicides, with notable cases of reduced sensitivity to triazoles, as well as to other chemistries like fludioxonil (Li et al. 2024a, 2023d; Zhou et al. 2022). To mitigate this, integrated strategies are imperative. These include adhering to recommended application regimes, limiting sprays per season, rotating or mixing fungicides with different modes of action, and leveraging disease forecasting models to optimize spray timing and necessity. Such measures are essential to reduce selection pressure and

delay the onset of resistance, thereby preserving the efficacy of chemical controls. This is particularly concerning when wheat straw is used as livestock feed, where toxin-blocking fungicides are preferable to prevent secondary exposure of humans through the food chain and potential occupational exposure for farmers handling the feed.

As sustainable agriculture gains momentum, biological control is receiving increasing attention for its ecological compatibility. Given FCR's soil-borne nature, managing microbial communities presents a promising solution. *Burkholderia cepacia*, a soil bacterium, was among the first species shown to suppress FCR effectively (Huang and Wong 1998). Additional key biocontrol agents include *Bacillus* and *Trichoderma* strains, which reduce FCR severity by inducing systemic acquired resistance (SAR) in wheat, leading to upregulated expression of pathogenesis-related (PR) proteins, such as peroxidases and endochitinases, that strengthen plant cell walls and degrade fungal structures (Moya-Elizondo and Jacobsen 2016). In field trials, *Bacillus subtilis* YB-05 combined with shenzinomycin achieved a 57.8% reduction in disease incidence (Zhang et al. 2018a). In vitro, *Trichoderma atroviride* HB20111 and *T. harzianum* TW21990 showed 78.81% inhibition (Chen et al. 2022), and co-inoculation with *T. gamsii* A5MH and *T. harzianum* Tr906 significantly suppressed *F. pseudograminearum* colonization (Stummer et al. 2022).

Biocontrol fungi act via diverse mechanisms. *Trichoderma longibrachiatum* (TG1) induces plant immunity by activating defense-related genes, including those encoding pathogenesis-related proteins such as *PR2* and *PR1-2* and chitinase class I (*CHIA1*), and through mycoparasitic activity involving upregulation of cell wall-degrading enzymes like β -1,6-glucan synthase and chitinase that directly target the pathogen. (Boamah et al. 2021), while *Bacillus velezensis* YB-185 directly disrupts fungal cell structures by downregulating genes involved in glucan and fatty acid synthesis while inducing cell apoptosis and oxidative stress, thereby impeding infection. (Zhang et al. 2022a). The arbuscular mycorrhizal fungus *Rhizophagus intraradices* competitively excludes the pathogen by reducing its population density by 75.7% and disease severity by 39%, while enhancing antioxidant responses and preserving membrane integrity. (Spagnoletti et al. 2021). Similarly, *Chaetomium globosum* 12XP1-2-3 inhibits pathogen growth both by hyphal contact—competing with *F. pseudograminearum* for ecological niches and nutrients—and by releasing unidentified bioactive inhibitory compounds (Feng et al. 2023).

Endophytic microbes further expand biocontrol potential. *Streptomyces hygroscopicus* strains MH71 and MH243, isolated from wheat roots, completely halted pathogen proliferation, though the specific mechanisms underlying this biocontrol effect remain unclear (O'Sullivan et al. 2021). *Alaromyces muroii* TM28, sourced from *Panax quinquefolius* roots, compromised pathogen membrane integrity, halting its growth. RNA-seq further revealed that TM28 downregulates genes involved

in fatty acid and steroid synthesis, although the specific active metabolites responsible require validation. (Yang et al. 2024a). *Talaromyces trachyspermus*, isolated from *Cornus officinalis*, also showed strong antifungal activity (Zhao et al. 2022d). Additionally, *Piriformospora indica* enhances wheat resistance by stimulating the phenylpropanoid pathway (Li et al. 2023a). Genetically engineered strains, such as Flg22-secreting plant growth-promoting rhizobacteria (PGPRs), confer dual benefits: pathogen suppression and root growth promotion (Li et al. 2024e). Although numerous biocontrol agents, such as *Trichoderma viride*, have been reported to exhibit antagonistic effects against FCR, their specific secondary metabolites and signaling pathways often remain insufficiently characterized. Elucidating these core active substances and mechanisms is crucial for advancing the targeted application and genetic enhancement of biocontrol strains.

To optimize FCR control, integrated strategies combining chemical and biological tools are essential. While chemical treatments offer rapid action, they risk resistance (Kim and Hwang 2007). Proactive resistance management, therefore, forms the bedrock of durable chemical control. In contrast, biological control supports long-term resilience by fostering beneficial plant–microbe interactions. Future efforts should focus on synergistic integration, such as developing fungicides compatible with Biological Control Agents (BCAs) or using gene editing to enhance microbial viability across soil types (Ons et al. 2020; Qiu et al. 2019). Such advancements will facilitate the development of more sustainable and robust disease management systems.

8.3 Breeding for FCR resistance

Establishing a reliable and efficient evaluation system for FCR resistance is essential for identifying resistant wheat cultivars. Currently, there is no globally standardized inoculation protocol; instead, assessments are primarily conducted at the seedling stage under controlled indoor conditions or at the adult stage in the field. Among seedling-stage methods, hydroponic and soil-based systems are widely used. The hydroponic spore immersion technique is especially popular for large-scale screening due to its simplicity and reproducibility (Nicol et al. 2001). Stem browning severity is typically assessed using a 0–5 scale developed by Li et al. (2008), which is adapted from the original scoring system by Wildermuth and McNamara (Li et al. 2008; Wildermuth and McNamara 1994). In soil-based systems, inoculation with natural media is favored for its consistency in large-scale experiments (Erginbas-Orakci et al. 2016; Poole et al. 2012). However, discrepancies can arise between seedling and adult-stage resistance due to extended evaluation periods and genotype-by-environment interactions (Erginbas-Orakci et al. 2016; Wildermuth and McNamara 1994).

Standard metrics for evaluating FCR resistance include the crown rot score (Poole et al. 2012), the crown rot severity index

(Mitter et al. 2006), and the leaf sheath sum (Wildermuth and McNamara 1994), with the crown rot score being the most prevalent (Erginbas-Orakci et al. 2016). Among these, the crown rot score—particularly the 0–5 scale by Li et al. (2008)—is the most widely adopted in both academic research and breeding programs (Li et al. 2008; Wildermuth and McNamara 1994). For adult plants, resistance is evaluated in greenhouses or field plots, typically using natural medium inoculation (Smiley et al. 2005b; Wallwork et al. 2004). Greenhouse assessments follow a 0–7 scale (Smiley et al. 2005b), while field evaluations occur at two growth stages: from 21 to 35 days post-emergence and again at plant maturity, with the latter involving a 0–4 scale to score main stem symptoms (Smiley et al. 2005b; Wildermuth and McNamara 1994; Yang et al. 2015).

Given the scarcity of genetic resistance resources in wheat, breeding for FCR resistance is a priority. Large-scale germplasm screenings have identified only a few moderately resistant lines. In an early study, Wildermuth et al. screened 400 wheat lines and identified Gluyas Early, Mexico 234, and Reliance as moderately resistant at the adult stage (Wildermuth and Purss 1971). In China's Huang-Huai region, where *F. pseudograminearum* is the dominant pathogen, Yang et al. reported no resistant genotypes at the seedling stage among 88 lines. Still, they observed moderate adult-stage resistance in cultivars such as Zhongyu 8 and Zhoumai 27 (Yang et al. 2015). Similarly, in another study involving 234 wheat lines, Yang et al. again found no seedling-stage resistance, underscoring the limited availability of effective germplasm (Yang et al. 2019).

To broaden the source of resistance, researchers have turned to mutagenesis and interspecific hybridization. A team identified a stable, moderately resistant mutant, *fcrZ22*, from an EMS-mutagenized population of Zhoumai 22 (Li et al. 2024c). In a notable advance, Wang et al. (2020) transferred the *Fhb7* gene—a glutathione transferase gene derived from the wild wheat relative *Thinopyrum elongatum*—into cultivated wheat. This gene confers resistance by detoxifying trichothecene mycotoxins, key virulence factors produced by *Fusarium* pathogens (Wang et al. 2020). The resulting near-isogenic lines (NILs) exhibited combined resistance to both *Fusarium* head blight (FHB) and FCR. This milestone represents a promising strategy for pyramiding multi-disease resistance traits in breeding programs.

FCR resistance in wheat is a quantitatively inherited trait (Poole et al. 2012). Identifying major resistance loci is critical for accelerating molecular breeding. The first FCR resistance QTL was identified near the *Rht1* dwarfing gene on chromosome 4B using the Kukri×Janz doubled haploid population (Wallwork et al. 2004). Since then, QTLs associated with FCR resistance have been mapped to all 21 wheat chromosomes, with significant loci concentrated on 1B, 2B, 3B, 4B, 6A, and 6B. Among these, loci on 3B, 4B, and 5D have shown the most substantial phenotypic effects. For instance, *Qcrs.cpi-3B* (also known as *Qcrs.wsu-3BL*) has been consistently detected across diverse

populations and environments, highlighting its central role in FCR resistance (Ma et al. 2014, 2012a; Zheng et al. 2015).

Population-based mapping studies have further validated these loci (Collard et al. 2006; Guo et al. 2022; Zheng et al. 2014; Zhou et al. 2021). Poole et al. (2012) identified QTLs on 2B, 3B, 4B, 4D, and 7A using Sunco/Macon and Sunco/Otis recombinant inbred line (RIL) populations, with 3B showing the highest phenotypic contribution (Poole et al. 2012). Similarly, Pariyar et al. used a 90 K SNP array on 161 wheat lines to identify 15 resistance QTLs, including a major locus on 3B linked to *Fhb1*, a gene conferring FHB resistance (Pariyar et al. 2020). Yang et al. (2021) mapped an FCR resistance QTL on 4B using a set of 435 introgression lines and a doubled-haploid (DH) population, ultimately cloning the regulatory gene *TaDIR*, which negatively regulates resistance by affecting lignin biosynthesis (Yang et al. 2021). By contrast, Jin et al. (2020a, b) conducted a genome-wide association study (GWAS) on 358 accessions using a 55 K SNP array and identified a major locus on chromosome 5DL containing five environment-stable SNPs, illustrating the background dependency of resistance expression (Jin et al. 2020b). In addition to chromosomes 3B, 4B, and 5D, other chromosomes also appear to harbor important QTL loci. For example, Martin et al. (2015) identified 13 FCR resistance QTL in four resistant sources, among which the 1AS, 1BS, and 4BS loci contributed by 2–49, and the 2BS locus contributed by Sunco, were stably expressed at both seedling and adult plant stages, whereas the 1DL locus in 2–49 and the 3BL locus in IRN497 were only detected at the seedling stage (Martin et al. 2015). Collard et al. (2005, 2006) consistently mapped major QTL on chromosomes 1D and 1A in the resistant source 2–49, explaining 21% and 9% of the phenotypic variation, respectively (Collard et al. 2005, 2006). Zheng et al. (2014) identified a major QTL, *Qcrs.cpi-2D*, located on chromosome 2D in the commercial variety EGA Wylie, which explained 20.2% of the phenotypic variation (Zheng et al. 2014). Bovill et al. (2010), through QTL pyramiding studies, detected a 3B locus from W21MMT70 explaining 40.5% of the phenotypic variation and a novel 7A locus from 2–49 in the 2–49/W21MMT70 population (Bovill et al. 2010); after pyramiding, the disease severity of the best line was only 3.8% of the susceptible control. Subsequent studies have further discovered and validated FCR resistance-related loci. Many researchers have also identified or validated FCR resistance-related loci through different research approaches (Bovill et al. 2006; Erginbas-Orakci et al. 2018; Guo et al. 2022; Hou et al. 2023; Li et al. 2023b; Malosetti et al. 2021; Rahman et al. 2020). Most recently, Zhang et al. (2024) identified 57 FCR resistance-associated SNPs via GWAS and BSA, mainly on chromosomes 2A and 7A. A SNP cluster on 2A (730–750 Mb) may carry novel resistance genes. These SNPs are promising for breeding but need functional validation. (Zhang et al. 2024) (Table 2).

Some data were extracted from Li et al. and Ma et al. (Li et al. 2023b; Ma et al. 2025). The physical locations of the loci were

identified using the JBrowse reference genome on the wheat multi-Omics website (<http://202.194.139.32/>). Stable QTLs with large effect or genes are labeled with asterisk and QTLs related to adult resistance was in bold.

Together, these findings reveal a complex polygenic regulatory landscape underlying FCR resistance in wheat. The continued identification and functional validation of key loci will provide a robust foundation for marker-assisted selection and the development of resistant cultivars (Fig. 3). Despite this progress, translating these findings into breeding applications faces several practical challenges. The polygenic nature of resistance and its environmental interactions complicate the development of standardized phenotyping systems, necessitating optimized greenhouse and field evaluation protocols that balance screening efficiency with reliable adult-plant resistance assessment. The limited availability of elite germplasm with stable and durable resistance requires expanded screening of diverse genetic resources including wild relatives and mutagenized populations, along with systematic evaluation of existing resistance sources across different genetic backgrounds and environments (Zhang et al. 2017). Strategic pyramiding of complementary resistance genes without compromising agronomic performance remains essential, demanding integrated molecular breeding approaches to develop elite lines combining robust resistance with favorable yield-related traits.

A multifaceted approach includes agronomic practices such as crop rotation with non-hosts, residue management via deep tillage or conservation agriculture, optimized water and nutrient regimes, and adjusted sowing dates to interrupt disease cycles. Chemical and biological control strategies integrate fungicide seed treatments, precision spraying, and application of endophytic or antagonistic microbes. Concurrently, breeding programs focus on standardized resistance screening, QTL mapping, and molecular marker-assisted selection to develop FCR-resistant cultivars. The synergistic integration of these components enables sustainable disease suppression.

9 Future research directions and prospects

To proactively manage the escalating threat of FCR under climate change, we propose deploying an integrated global real-time monitoring network combining remote sensing, IoT, and pathogen genomics for early detection and predictive modeling. Simultaneously, accelerating the development of durable resistant varieties through standardized screening and advanced gene editing (e.g., Prime Editing), coupled with optimizing sustainable field management using microbiome engineering and synergistic biocontrols, is essential. Furthermore, deciphering pathogenicity mechanisms through structural biology and epigenomics will enable the design of next-generation targeted interventions such as small-molecule

Table 2 QTL conferring FCR resistance that are stable or repeatedly detected in different studies

Chromosome	Quantitative trait loci (QTL)	Associated markers	Physical position (Mb)	References
1A	<i>Qfcr.sicau.1A-1</i>	<i>wsnp_Ku_c183_358844</i>	8.33–14.33	Hou et al. (2023), Rahman et al. (2020)
1A	<i>1AQcr-Xwmc120</i>	<i>20Xwmc120-Xwmc312</i>	344.70	Collard et al. (2005), Guo et al. (2022)
1B	<i>Qcr-Ajfx-88612017</i>	<i>Ajfx-88612017</i>	5.85	Jin et al. (2020a), Su et al. (2025)
1B	<i>*Qfcr.sicau.1B-4</i>	<i>SNP1868</i>	641.36–645.13	Hou et al. (2023)
1D	<i>Qcr-Ajfx-92108178</i>	<i>Ajfx-92108178, BobWhite_c1715_887</i>	6.33–9.88	Jin et al. (2020a), Rahman et al. (2021)
1D	<i>Qcr.usq-1D.1-Xwmc216</i>	<i>Xwmc216, ChrID-416032418</i>	364.03	Bovill et al. (2010), Collard et al. (2005, 2006), Rahman et al. (2021)
2B	<i>*TaCWI-B1-TraesCS2B02G311900</i>	<i>Xgwm630, Xcfa22278, primer P9, Chr2B-518870327</i>	442.79–446.08	Bovill et al. (2006), Lv et al. (2023), Martin et al. (2015), Zhou et al. (2021)
2D	<i>Qers.cpi-2D-1131013F0</i>	<i>1131013F0, BS00062567_51, D_cont-fig17313_245</i>	1.13–13.78	Pariyar et al. (2020), Rahman et al. (2021)
2D	<i>Qcr-Xgwm484</i>	<i>Xgwm484, wPt-669517</i>	37.05–48.17	Erginbas-Orakci et al. (2018), Martin et al. (2015)
3A	<i>Qcr-BobWhite_c30009_285</i>	<i>wsnp_Ra_c16278_24893033, BobWhite_c30009_285, Excalibur_rep_c68583_689</i>	68.30–79.45	Pariyar et al. (2020), Rahman et al. (2021)
3A	<i>Qcr-CAP8_c1393_327</i>	<i>CAP8_c1393_327, Ku_c1638_648</i>	557.42–565.78	Pariyar et al. (2020), Rahman et al. (2021)
3A	<i>Qcr-BS00036168_51_3A</i>	<i>Excalibur_c78654_184, BS00036168_51_3A</i>	687.92–688.62	Malosetti et al. (2021), Rahman et al. (2021)
3B	<i>*Qcr-CAP12_rep_c3868_270</i>	<i>CAP12_rep_c3868_270, Xgwm389</i>	0.81–0.99	Ma et al. (2012b), Pariyar et al. (2020)
3B	<i>Qfcr.sicau.3B-1</i>	<i>SNP7310, AX-110372139</i>	23.11–39.46	Erginbas-Orakci et al. (2018), Hou et al. (2023), Pu et al. (2020)
3B	<i>Qfcr.sicau.3B-4</i>	<i>SNP7936, BS0007299451, BS0007902951, IACX11310</i>	582.01–586.51	Rahman et al. (2020)
3B	<i>Qers.cpi-3B, Qers.wau-3BL</i>	<i>Xgwm181, wPt-1834, Xwmc236, Xgwm299, Xgwm247, wPt-0365RCR, RAC875_c17373_848</i>	-830.40	Erginbas-Orakci et al. (2018), Ma et al. (2010), Malosetti et al. (2021), Martin et al. (2015), Poole et al. (2012), Rahman et al. (2020), Zhou et al. (2021)
3D	<i>*TaAACT1-TraesCS3D02G005500.1</i>	<i>Qfcr.sicau.3D-1, SNP8533</i>	2.15–5.30	Hou et al. (2023), Xiong et al. (2023)
4A	<i>QFCR-S.sau-4AS</i>	<i>BS00035307_51, JWA1900</i>	3.38–8.20	Guo et al. (2022), Rahman et al. (2020)
4B	<i>Qers.cpi-4B.2</i>	<i>11084721F10, 10936161F10, wsnp_Ku_c12399_20037334</i>	1.11–1.26	Pariyar et al. (2020), Zheng et al. (2014)
4B	<i>Qcr.usq-4B.1</i>	<i>Xhare193, wPt-4535</i>	24.92–26.12	Bovill et al. (2010), Martin et al. (2015)
4B	<i>Qers.cpi-4B.1</i>	<i>100004319FN0, wPt-5334</i>	101.69	Ma et al. (2010), Zheng et al. (2014)
4B	<i>Kukri_c15910_159</i>	<i>Kukri_c15910_159, wPt-732448</i>	527.01–535.07	Martin et al. (2015), Rahman et al. (2021)
4B	<i>*Qcr.usq-4B.1-Xgwm251</i>	<i>Xgwm251, GENE-2422_208, BobWhite_c11005236</i>	568.56–579.39	Bovill et al. (2010), Collard et al. (2005, 2006), Martin et al. (2015), Rahman et al. (2021), Wallwork et al. (2004)
4B	<i>Qfcr.sicau.4B-2</i>	<i>SNP10193, IACX6482, RAC875_rep_c72961_977</i>	617.00–629.22	Hou et al. (2023), Pariyar et al. (2020), Rahman et al. (2021)
4B	<i>*TaDIR-B1-TraesCS4B02G385500</i>	<i>TraesCS4B02G385500, SNP10261, Kukri_c21787_283, AX-110977572, AX-111079978, BS00034147_51</i>	652.91–664.68	Hou et al. (2023), Rahman et al. (2021), Yang et al. (2021)
5A	<i>Qcr-Kukri_c33022_198</i>	<i>Kukri_c33022_198, wsnp_Ex_c8424_14192191_5A</i>	589.22–595.15	Malosetti et al. (2021), Rahman et al. (2021)
5A	<i>Qfcr.sicau.5A-1</i>	<i>SNP11200, wsnp_BG607308A_TA_2_1</i>	617.35–620.15	Hou et al. (2023), Rahman et al. (2021)
5D	<i>Qers.cpi-5D</i>	<i>1215315FN0, 1237596FN0, Xcfa189</i>	1.22–8.75	Bovill et al. (2006), Zheng et al. (2017, 2014)

Table 2 (continued)

Chromosome	Quantitative trait loci (QTL)	Associated markers	Physical position (Mb)	References
5D	* <i>Qcr-5DL-Ajfx-110484766</i>	<i>Ajfx-110557977</i> , <i>Ajfx-110079634</i>	79.57	Jin et al. (2020a), Pariyar et al. (2020)
5D	* <i>TaWAK-5D600-TraesCS5D02G268600</i>	<i>TraesCS5D02G268600</i>	371.84	Qi et al. (2023)
5D	<i>Qcr-D_contig79410_237</i>	<i>Excilbur_c2795_1518</i> , <i>D_contig79410_237</i>	542.66–546.92	Pariyar et al. (2020), Rahman et al. (2021)
6A	<i>Qcr-IAAV5346</i>	<i>IAAV5346</i> , <i>RAC875_c17297_341</i> , <i>BS00065852_51</i>	63.30–74.00	Pariyar et al. (2020), Rahman et al. (2021)
6A	* <i>TaRLK-TraesCS6A02G222000</i>	<i>TraesCS6A02G222000</i>	414.86	Qi et al. (2021)
6A	* <i>Qcr-6AL-AX-111106634</i>	<i>AX-111106634</i>	490.49–497.46	Yang et al. (2019)
6B	<i>Qcr-AX-108823788</i>	<i>Jagger_c555_287_6B</i> , <i>AX-108823788</i>	191.53–191.99	Malosetti et al. (2021), Pu et al. (2020)
6B	<i>Qcr-Xbare178</i>	<i>Xbare24-Xbare178</i> , <i>RAC875_c60007_199</i>	656.19–664.63	Ma et al. (2012b), Rahman et al. (2020)
6B	<i>Qcr-Kukri_c27662_675</i>	<i>Kukri_c27662_675</i> , <i>BobWhite_c19298_97</i>	705.38	Pariyar et al. (2020), Rahman et al. (2021)
6D	* <i>TaWAK-6D-TraesCS6D02G210200</i>	<i>TraesCS6D02G210200</i>	296.82	Qi et al. (2021)
7A	<i>Qcr-usq-7A.1</i>	<i>Tdurum_contig54860_441_7A</i> , <i>wPt-4748</i> , <i>Bobwhite_c33300_159</i>	17.33–20.66	Bovill et al. (2010), Malosetti et al. (2021), Rahman et al. (2020)
7A	* <i>TaCRK-7A-TraesCS7A02G105100</i>	<i>TraesCS7A02G105100.1</i>	63.78	Wu et al. (2021)
7B	<i>Qcr-Kukri_c28260_2017</i>	<i>Kukri_c28160_2017</i> , <i>RAC875_c4876_2096_7B</i>	716.96–734.27	Malosetti et al. (2021), Rahman et al. (2021)

virulence blockers. Ultimately, realizing this anticipatory approach requires sustained global interdisciplinary collaboration and data sharing to rapidly bridge research gaps and deploy convergent innovations across detection, breeding, ecology, and molecular design.

The management of FCR is becoming increasingly complex due to the accelerating impacts of climate change. Elevated atmospheric CO₂ levels enhance plant biomass accumulation, inadvertently intensifying FCR severity by fostering the growth of the stubble-borne pathogen *F. pseudograminearum* (Melloy et al. 2010). In parallel, rising temperatures, decreased rainfall, and increasingly uneven soil moisture in key wheat-producing regions, such as Australia, further elevate FCR risks by promoting pathogen proliferation and disease transmission (Hochman et al. 2017). Addressing these challenges demands an integrated, multidisciplinary approach that combines advances in detection, breeding, biotechnology, and disease ecology.

Recent advances in detection technologies have significantly improved early diagnosis capabilities. Thermal imaging, along with multispectral and hyperspectral imaging, has enabled successful early-stage detection of FCR by detecting subtle physiological changes, such as increased temperature caused by FCR infection across multiple wheat plants before visible symptoms appear (Buster et al. 2023). These technologies lay the groundwork for a real-time early warning system that integrates satellite remote sensing, Internet of Things (IoT) sensors, and pathogen genomic data. Drones are also proving effective in precision disease management (Sun et al. 2024), particularly in environments where traditional sprayers are impractical (Köycü et al. 2024). Equipped with spectral imaging systems, drones can identify early visual symptoms, such as basal stem browning, and apply targeted treatments with centimeter-level accuracy using Geographic Information Systems (GIS)-based prescription maps. This precision approach significantly reduces input costs, such as pesticides and fertilizers, while minimizing environmental impacts. However, its cost-effectiveness depends on factors such as crop value, scale of deployment, and disease severity (Guarino et al. 2002).

However, building a global real-time monitoring network entails considerable challenges, notably the substantial financial investment required for infrastructure, the necessity of tailoring systems to diverse agronomic conditions through regional calibration, and the complex task of establishing fair data governance protocols for handling sensitive pathogen genomic information (Dhavale et al. 2025). A phased approach offers a pragmatic path forward. Initial efforts could concentrate on creating regional observatories within major wheat producing zones to refine monitoring protocols suited

to local contexts while keeping expenses manageable. Subsequent expansion would then focus on developing shared data platforms capable of integrating these regional networks, ultimately leading to a globally coordinated surveillance system that can predict pathogen threats and inform timely interventions (Miller et al. 2009).

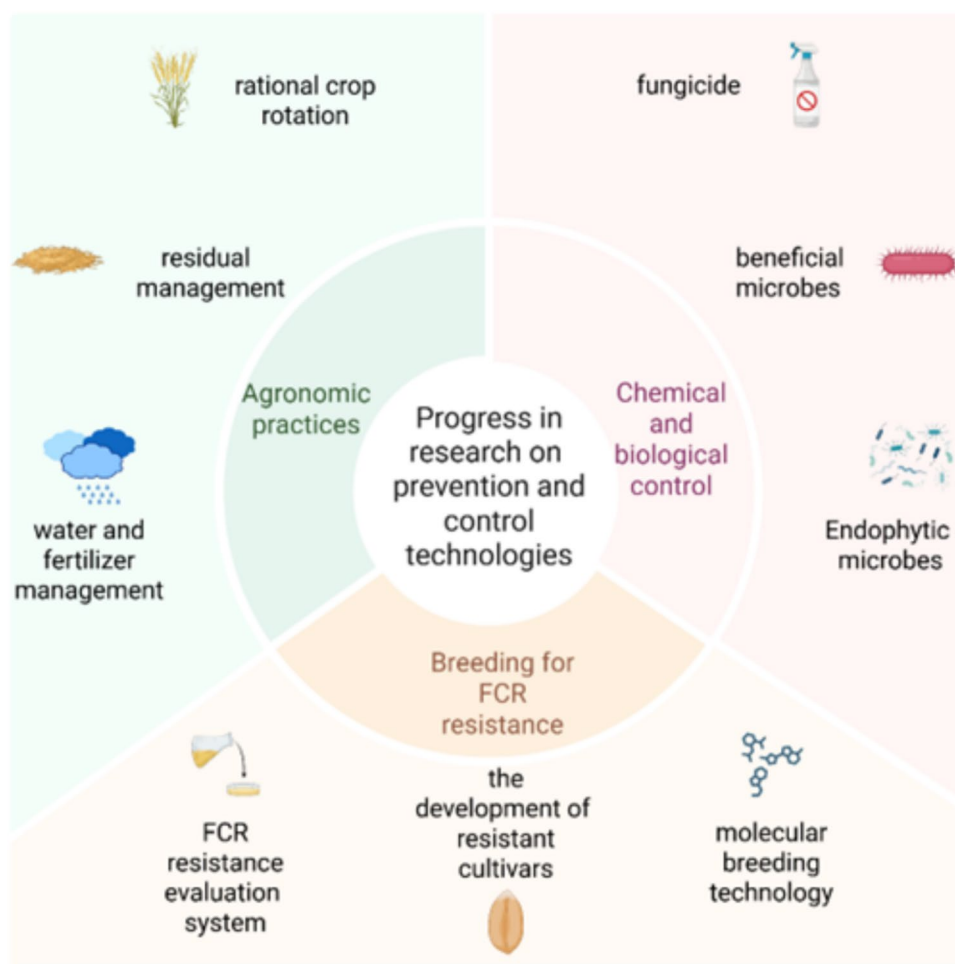
Breeding for FCR-resistant cultivars remains a cornerstone of long-term disease control. Despite ongoing evaluation efforts, the lack of standardized, high-throughput inoculation protocols, stemming from challenges like inconsistent inoculant distribution, complex interactions with other pathogens, and variable weather and environmental conditions, has hindered efficient large-scale resistance screening (Mitter et al. 2006). Developing globally harmonized and reproducible inoculation systems will be essential to overcoming this bottleneck. Furthermore, although QTL mapping has yielded promising candidates, few of these loci have been effectively deployed in breeding programs to date. To accelerate gene discovery, integrating genome-wide association studies (GWAS) with weighted gene co-expression network analysis (WGCNA) offers a powerful approach,

leveraging complementary strengths to pinpoint candidate genes more effectively and elucidate their functional relationships, particularly with the availability of high-resolution wheat pan-genome resources, thereby enhancing the statistical power and biological interpretability of findings (Lübberstedt 2017; Wei et al. 2022). Single-cell sequencing technologies can further refine this approach by identifying cell-type-specific resistance pathways, such as those in bundle sheath cells (Zheng et al. 2023).

The emergence of Prime Editing enables precise, single-base modifications in susceptibility genes, allowing the removal of virulence targets without disrupting the broader genome (Dong and Fan 2024). However, the sustainable deployment of resistant varieties also requires robust pathogen surveillance. A global monitoring network capable of tracking the evolutionary dynamics of virulence genes, paired with population genomics, will be critical for anticipating pathogen adaptation (Gardy and Loman 2018; Jørgensen et al. 2014).

The growing resistance of *F. pseudograminearum* to chemical treatments further underscores the urgency of biological control strategies (Li et al. 2023c). Recent studies on rhizosphere microbiome engineering have

Fig. 3 Integrated strategies for FCR management



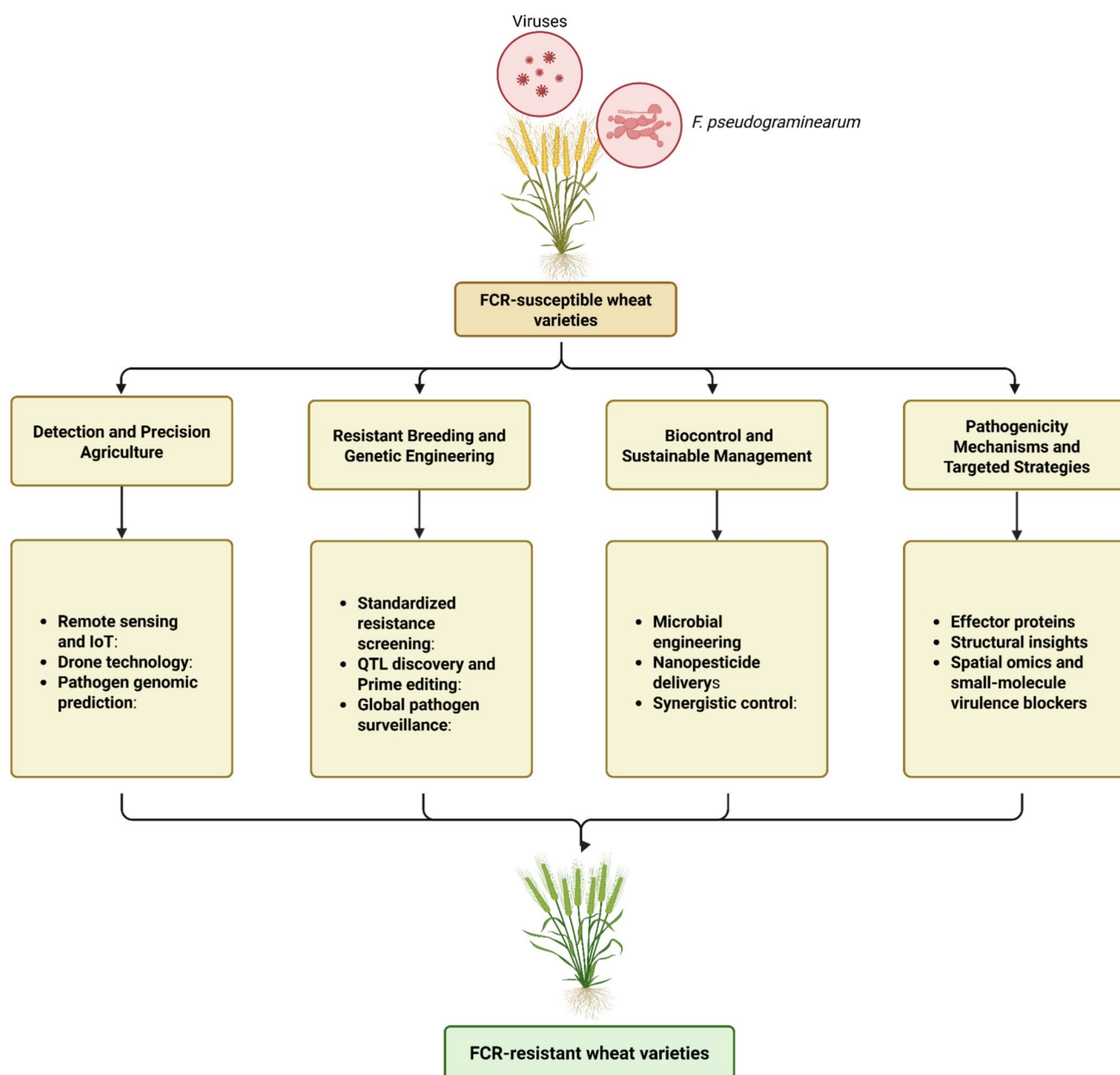


Fig. 4 Emerging technologies and future directions in FCR control

identified microbial antagonists with the potential to suppress FCR pathogens through competitive exclusion and metabolite production (Deroo et al. 2022). Combining nanoparticle delivery systems with metagenomic screening has enhanced the precision and efficacy of biopesticide applications (Jun and Zheng 2019; Ray et al. 2023). Additionally, integrating chemical and biological agents into synergistic formulations reduces chemical load while maintaining control efficacy (Reddy and Chowdary 2021).

Understanding the molecular mechanisms of *F. pseudograminearum* infection remains a high priority.

Comparative genomics, drawing on parallels with *F. graminearum*, enables the identification of conserved effector families (Alouane et al. 2021). These can be validated using wheat protoplast transient expression systems to assess their immunosuppressive capacity (Chen et al. 2023). Structural studies via cryo-electron microscopy may reveal the interaction dynamics between effector proteins and host NLR immune receptors, guiding the rational design of synthetic resistance genes (Wang and Chai 2020). Epigenomic tools such as ChIP-seq, enables the exploration of histone modifications across different stages of infection (Atanasoff-Kardjalieff and

Studt 2022), while targeting key enzymatic regulators of toxin biosynthesis may open avenues for chemical control. For instance, suberoylanilide hydroxamic acid (SAHA), a histone deacetylase inhibitor, can suppress fungal virulence gene expression without harming host plants (Hong-Geller and Micheva-Viteva 2013). Spatial transcriptomics and metabolomics are emerging as indispensable tools for mapping gene expression and metabolite flux in infected tissues, thereby identifying critical infection zones and enabling real-time tracking of pathogen progression (Liu et al. 2025; Wang et al. 2024).

Looking forward, the phased construction of a real-time monitoring infrastructure that fuses aerial and terrestrial data streams will be essential. By integrating field-based sensing technologies with predictive models of pathogen evolution, researchers can transition from reactive to anticipatory disease management (Jin et al. 2022; Zhang et al. 2018b). A multilayered ecological approach that integrates resistance gene editing, microbiome optimization, and targeted chemical application will form the basis of next-generation FCR control strategies. Ultimately, advancing this vision will require sustained interdisciplinary collaboration and a global commitment to data sharing to bridge gaps across research domains and accelerate the deployment of innovative technologies (Langridge et al. 2022) (Fig. 4).

Next-generation strategies incorporate precision agriculture—utilizing drones, remote sensing, and IoT systems for real-time disease detection—coupled with molecular breeding innovations, including QTL discovery, prime editing, and harmonized resistance evaluation. Sustainable disease management is further reinforced by microbial community engineering, nanopesticide delivery platforms, and effector-targeted small-molecule inhibitors. Spatial omics and global pathogen surveillance systems contribute to predictive epidemiological modeling. Cross-disciplinary collaboration is key to advancing durable resistance and integrated FCR management.

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Data Availability No datasets were generated or analysed during the current study.

Declarations Conflict of interest

All authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript. No funding sources or institutional affiliations have created any potential conflicts related to the design, literature collation, analysis, or publication of this review.

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References

- Abdallah-Nekache N, Laraba I, Ducos C, Barreau C, Bouznad Z, et al. Occurrence of *Fusarium* head blight and *Fusarium* crown rot in Algerian wheat: identification of associated species and assessment of aggressiveness. *Eur J Plant Pathol*. 2019;154:499–512.
- Abu-Zaitoon YM, Al Tawaha AR, Alnaimat SM, Al-Rawashdeh IM, Abu-Zaiton A, et al. Investigation of the potential role of aldehyde oxidase in indole-3-acetic acid synthesis of developing rice grains. *Plant Cell Biotechnol Mol Biol*. 2019;20:6–13.
- Ahmed ST, Islam MM, Mun H-S, Sim H-J, Kim Y-J, et al. Effects of *Bacillus amyloliquefaciens* as a probiotic strain on growth performance, cecal microflora, and fecal noxious gas emissions of broiler chickens. *Poult Sci*. 2014;93:1963–71.
- Akgül DS, Erkilic A. Effect of wheat cultivars, fertilizers, and fungicides on *Fusarium* foot rot disease of wheat. *Turk J Agric for*. 2016;40:101–8.
- Akinsanmi OA, Mitter V, Simpfendorfer S, Backhouse D, Chakraborty S. Identity and pathogenicity of *Fusarium* spp. isolated from wheat fields in Queensland and northern New South Wales. *Aust J Agric Res*. 2004;55:97–107.
- Alkadri D, Nipoti P, Döll K, Karlovsky P, Prodi A, et al. Study of fungal colonization of wheat kernels in Syria with a focus on *Fusarium* species. *Int J Mol Sci*. 2013;14:5938–51.
- Alouane T, Rimbert H, Bormann J, González-Montiel GA, Loesgen S, et al. Comparative genomics of eight *Fusarium graminearum* strains with contrasting aggressiveness reveals an expanded open pangenome and extended effector content signatures. *Int J Mol Sci*. 2021;22:6257.
- Aoki T, O'Donnell K. Morphological and molecular characterization of *Fusarium pseudograminearum* sp. nov., formerly recognized as the Group 1 population of *F. graminearum*. *Mycologia*. 1999;91:597–609.
- Arnao MB, Hernández-Ruiz J. The physiological function of melatonin in plants. *Plant Signal Behav*. 2006;1:89–95.
- Atanasoff-Kardjaleff AK, Studt L. Secondary metabolite gene regulation in mycotoxigenic *Fusarium* species: a focus on chromatin. *Toxins Basel*. 2022;14:96.
- Bari R, Jones JD. Role of plant hormones in plant defence responses. *Plant Mol Biol*. 2009;69:473–88.

- Beccari G, Covarelli L, Nicholson P. Infection processes and soft wheat response to root rot and crown rot caused by *Fusarium culmorum*. Plant Pathol. 2011;60:671–84.
- Bi Z, Wang T, Wang X, Xu H, Wu Y, et al. *FpPEX5* and *FpPEX7* are involved in the growth, reproduction, DON toxin production, and pathogenicity in *Fusarium pseudograminearum*. Int J Biol Macromol. 2024;270:132227.
- Blum A, Benfield AH, Sørensen JL, Nielsen MR, Bachleitner S, et al. Regulation of a novel fusarium cytokinin in *Fusarium pseudograminearum*. Fungal Biol. 2019;123:255–66.
- Boamah S, Zhang S, Xu B, Li T, Calderón-Urrea A. *Trichoderma longibrachiatum* (TG1) enhances wheat seedlings tolerance to salt stress and resistance to *Fusarium pseudograminearum*. Front Plant Sci. 2021;12:741231.
- Bovill W, Ma W, Ritter K, Collard B, Davis M, et al. Identification of novel QTL for resistance to crown rot in the doubled haploid wheat population ‘W21MMT70’×‘Mendos.’ Plant Breeding. 2006;125:538–43.
- Bovill W, Horne M, Herde D, Davis M, Wildermuth G, et al. Pyramiding QTL increases seedling resistance to crown rot (*Fusarium pseudograminearum*) of wheat (*Triticum aestivum*). Theor Appl Genet. 2010;121:127–36.
- Bragard C, Baptista P, Chatzivassiliou E, Di Serio F, Gonthier P, et al. Pest categorisation of *Fusarium pseudograminearum*. EFSA J. 2022;20:e07399.
- Burgess L. Intermediate hosts and the management of crown rot and head blight. In: Annual report of GRDC strategic initiative on crown rot, common root rot and Fusarium head blight, pp. 34–36, 2005.
- Buster M, Simpfendorfer S, Guppy C, Sissons M, Flavel RJ. Fusarium crown rot reduces water use and causes yield penalties in wheat under adequate and above average water availability. Agronomy. 2022;12:2616.
- Buster M, Simpfendorfer S, Guppy C, Sissons M, Tighe M, et al. Remote detection of Fusarium crown rot in broadacre bread wheat and durum wheat through use of aerial imagery. Crop Pasture Sci. 2023;75.
- Castañares E, Wehrhahne L, Stenglein SA. *Fusarium pseudograminearum* associated with barley kernels in Argentina. Plant Dis. 2012;96:763–763.
- Chakraborty S, Liu C, Mitter V, Scott J, Akinsanmi O, et al. Pathogen population structure and epidemiology are keys to wheat crown rot and *Fusarium* head blight management. Australas Plant Pathol. 2006;35:643–55.
- Champeil A, Doré T, Fourbet J-F. *Fusarium* head blight: epidemiological origin of the effects of cultural practices on head blight attacks and the production of mycotoxins by *Fusarium* in wheat grains. Plant Sci. 2004;166:1389–415.
- Chen L, Geng X, Ma Y, Zhao J, Chen W, et al. The ER luminal *Hsp70* protein *FpLhs1* is important for conidiation and plant infection in *Fusarium pseudograminearum*. Front Microbiol. 2019a;10:1401.
- Chen L, Geng X, Ma Y, Zhao J, Li T, et al. Identification of basic helix-loop-helix transcription factors reveals candidate genes involved in pathogenicity of *Fusarium pseudograminearum*. Can J Plant Pathol. 2019b;41:200–8.
- Chen L, Ma Y, Zhao J, Geng X, Chen W, et al. The *bZIP* transcription factor *FpAdal1* is essential for fungal growth and conidiation in *Fusarium pseudograminearum*. Curr Genet. 2020a;66:507–15.
- Chen L, Zhao J, Xia H, Ma Y, Liu Y, et al. *FpCzf14* is a putative C₂H₂ transcription factor regulating conidiation in *Fusarium pseudograminearum*. Phytopathology Research. 2020b;2:1–10.
- Chen L, Ma Y, Peng M, Chen W, Xia H, et al. Analysis of apoptosis-related genes reveals that apoptosis functions in conidiation and pathogenesis of *Fusarium pseudograminearum*. Msphere. 2021;6:10.1128/msphere. 01140-01120.
- Chen K, Chen J, Pi X, Huang L-J, Li N. Isolation, purification, and application of protoplasts and transient expression systems in plants. Int J Mol Sci. 2023;24:16892.
- Chen L, Shan Y, Dong Z, Zhang Y, Peng M, et al. A potential hyphal fusion protein complex with an important role in development and virulence interacts with autophagy-related proteins in *Fusarium pseudograminearum*. J Integr Agric. 2024;23:4093–106.
- Chen K, Sui LN, Yang K, Zhao ZJ, Hu JD, et al. Co-cultivation of two *Trichoderma* strains enhanced control efficiency against wheat crown rot at seedling stage. 2022.
- Collard B, Grams R, Bovill W, Percy C, Jolley R, et al. Development of molecular markers for crown rot resistance in wheat: mapping of QTLs for seedling resistance in a ‘2-49’×‘Janz’ population. Plant Breeding. 2005;124:532–7.
- Collard B, Jolley R, Bovill W, Grams R, Wildermuth G, et al. Confirmation of QTL mapping and marker validation for partial seedling resistance to crown rot in wheat line ‘2-49.’ Aust J Agric Res. 2006;57:967–73.
- Cook RJ. *Fusarium* foot rot of wheat and its control in the Pacific Northwest. Plant Dis. 1980;64:1061–6.
- Cook R, Sitton J, Waldher J. Evidence for *Pythium* as a pathogen of direct-drilled wheat in the Pacific Northwest. Plant Dis. 1980;64:61–61.
- Deroo W, De Troyer L, Dumoulin F, De Saeger S, De Boevre M, et al. A novel in planta enrichment method employing *Fusarium graminearum*-infected wheat spikes to select for competitive bio-control bacteria. Toxins Basel. 2022;14:222.
- Desmond OJ, Edgar CI, Manners JM, Maclean DJ, Schenk PM, et al. Methyl jasmonate induced gene expression in wheat delays symptom development by the crown rot pathogen *Fusarium pseudograminearum*. Physiol Mol Plant Pathol. 2005;67:171–9.
- Dhavalé MC, Aiwale HN, Sarnobat DH. Modern diagnostic techniques in plant pathology. Plant Health Dis Manag Modern Approach 184; 2025.
- Dodman R, Wildermuth G. Inoculation methods for assessing resistance in wheat to crown rot caused by *Fusarium graminearum* Group 1. Aust J Agric Res. 1987;38:473–86.
- Dong G, Fan Z. CRISPR/Cas-mediated germplasm improvement and new strategies for crop protection. Crop Health. 2024;2:2.
- Dong Z, Ding T, Shan Y, Li H, Chen L, et al. Autophagy-related gene *FpAtg3* involves in growth and pathogenicity of *Fusarium pseudograminearum*. Sci Agric Sin. 2024;57:1080–90.
- Duan S, Jin J, Gao Y, Jin C, Mu J, et al. Integrated transcriptome and metabolite profiling highlights the role of benzoxazinoids in wheat resistance against *Fusarium* crown rot. Crop J. 2022;10:407–17.
- Erginbas-Orakci G, Poole G, Nicol JM, Paulitz T, Dababat AA, et al. Assessment of inoculation methods to identify resistance to *Fusarium* crown rot in wheat. J Plant Dis Prot. 2016;123:19–27.
- Erginbas-Orakci G, Sehgal D, Sohail Q, Ogbonnaya F, Dreisigacker S, et al. Identification of novel quantitative trait loci linked to crown rot resistance in spring wheat. Int J Mol Sci. 2018;19:2666.
- Fan X, Yan Z, Yang M, Waalwijk C, van der Lee T, et al. Contamination and translocation of deoxynivalenol and its derivatives associated with *Fusarium* crown rot of wheat in Northern China. Plant Dis. 2021;105:3397–406.
- Felton W, Marcellos H, Alston C, Martin R, Backhouse D, et al. Chickpea in wheat-based cropping systems of northern New South Wales. II. Influence on biomass, grain yield, and crown rot in the following wheat crop. Aust J Agric Res. 1998;49:401–8.
- Feng C, Li L, Zhang J, Wang J, Song Y, et al. Mechanism of *Chaetomium globosum* in promoting growth and preventing disease and advance of its application and research. Chin J Biol Control. 2023;39:961.

- Francis RG, Burgess L. Characteristics of two populations of *Fusarium roseum* 'Graminearum' in eastern Australia. *Trans Br Mycol Soc.* 1977;68:421–7.
- Gao Y, Tian X, Wang W, Xu X, Su Y, et al. Changes in concentrations and transcripts of plant hormones in wheat seedling roots in response to *Fusarium* crown rot. *Crop J.* 2023;11:1441–50.
- Gardiner DM, Benfield AH, Stiller J, Stephen S, Aitken K, et al. A high-resolution genetic map of the cereal crown rot pathogen *Fusarium pseudograminearum* provides a near-complete genome assembly. *Mol Plant Pathol.* 2018;19:217–26.
- Gardy JL, Loman NJ. Towards a genomics-informed, real-time, global pathogen surveillance system. *Nat Rev Genet.* 2018;19:9–20.
- Gargouri S, Mtat I, Kammoun LG, Zid M, Hajlaoui MR. Molecular genetic diversity in populations of *Fusarium pseudograminearum* from Tunisia. *J Phytopathol.* 2011;159:306–13.
- Grewal HS, Graham RD, Rengel Z. Genotypic variation in zinc efficiency and resistance to crown rot disease (*Fusarium graminearum* Schw. Group 1) in wheat. *Plant Soil.* 1996;186:219–26.
- Guarino L, Jarvis A, Hijmans R, Maxted N. Geographic information systems (GIS) and the conservation and use of plant genetic resources. In: *Managing plant genetic diversity. Proceedings of an international conference, Kuala Lumpur, Malaysia, 12–16 June 2000.* Wallingford: CABI publishing; 2002, pp. 387–404.
- Guo C, Cheng D, Qi J, Liu X, Liu H, et al. QTL mapping for resistance to *Fusarium* crown rot in common wheat at seedling stage. *Shandong Agric Sci.* 2022;54:1–7.
- Hameed M, Rana R, Ali Z. Identification and characterization of a novel Iraqi isolate of *Fusarium pseudograminearum* causing crown rot in wheat. *Genet Mol Res.* 2012;11:1341–8.
- Hansen FT, Gardiner DM, Lysøe E, Fuertes PR, Tudzynski B, et al. An update to polyketide synthase and non-ribosomal synthetase genes and nomenclature in *Fusarium*. *Fungal Genet Biol.* 2015;75:20–9.
- Hochman Z, Gobbett DL, Horan H. Climate trends account for stalled wheat yields in Australia since 1990. *Glob Change Biol.* 2017;23:2071–81.
- Hong-Geller E, Micheva-Viteva S. Small molecule screens to identify inhibitors of. *Drug Discov.* 2013;157.
- Hou S, Lin Y, Yu S, Yan N, Chen H, et al. Genome-wide association analysis of *Fusarium* crown rot resistance in Chinese wheat landraces. *Theor Appl Genet.* 2023;136:101.
- Huang Y, Wong P. Effect of *Burkholderia* (*Pseudomonas*) *cepacia* and soil type on the control of crown rot in wheat. *Plant Soil.* 1998;203:103–8.
- Ji L, Kong L, Li Q, Wang L, Chen D, et al. First report of *Fusarium pseudograminearum* causing *Fusarium* head blight of wheat in Hebei Province, China. *Plant Dis.* 2016a;100:220–220.
- Ji L, Li Q, Wang L, Wang Y, Kong L. Warning on the outbreaks of wheat disease and its controlling. *China Plant Prot.* 2016b;36:39–42.
- Jin J, Duan S, Qi Y, Yan S, Li W, et al. Identification of a novel genomic region associated with resistance to *Fusarium* crown rot in wheat. *Theor Appl Genet.* 2020a;133:2063–73.
- Jin J, Qi Y, Wang L, Wang F, Yan C, et al. Evaluation of Chinese wheat germplasm resources for crown rot resistance. *Plant Genet Resour.* 2020b;21:308–13.
- Jin X, Yang W, Doonan JH, Atzberger C. *Crop phenotyping studies with application to crop monitoring*; Elsevier: New York; 2022, pp. 1221–1223.
- Jørgensen LN, Hovmøller MS, Hansen JG, Lassen P, Clark B, et al. IPM strategies and their dilemmas including an introduction to www.eurowheat.org. *J Integr Agric.* 2014;13:265–81.
- Jun X, Zheng Y. Progress of the discovery, application, and control technologies of chemical pesticides in China. *J Integr Agric.* 2019;18:840–53.
- Kammoun LG, Gargouri S, Hajlaoui MR, Marrakchi M. Occurrence and distribution of *Microdochium* and *Fusarium* species isolated from durum wheat in northern Tunisia and detection of mycotoxins in naturally infested grain. *J Phytopathol.* 2009;157:546–51.
- Kazan K, Gardiner DM. *Fusarium* crown rot caused by *Fusarium pseudograminearum* in cereal crops: recent progress and future prospects. *Mol Plant Pathol.* 2018;19:1547–62.
- Kettle AJ. Degradation of the benzoxazolinone class of phytoalexins by *Fusarium pseudograminearum*, a pathogenic fungus of wheat. In: *The University of Queensland*; 2016.
- Khudhair M, Thatcher L, Gardiner D, Kazan K, Roper M, et al. Comparative analysis of genetic structures and aggressiveness of *Fusarium pseudograminearum* populations from two surveys undertaken in 2008 and 2015 at two sites in the wheat belt of Western Australia. *Plant Pathol.* 2019;68:1337–49.
- Khudhair M, Obanor F, Kazan K, Gardiner D, Aitken E, et al. Genetic diversity of Australian *Fusarium pseudograminearum* populations causing crown rot in wheat. *Eur J Plant Pathol.* 2021;159:741–53.
- Kim BS, Hwang BK. Microbial fungicides in the control of plant diseases. *J Phytopathol.* 2007;155:641–53.
- Kirkegaard J, Simpfendorfer S, Holland J, Bambach R, Moore K, et al. Effect of previous crops on crown rot and yield of durum and bread wheat in northern NSW. *Aust J Agric Res.* 2004;55:321–34.
- Klein T, Burgess L, Ellison F. The incidence and spatial patterns of wheat plants infected by *Fusarium graminearum* Group 1 and the effect of crown rot on yield. *Aust J Agric Res.* 1991;42:399–407.
- Knight NL, Sutherland MW. Histopathological assessment of *Fusarium pseudograminearum* colonization of cereal culms during crown rot infections. *Plant Dis.* 2016;100:252–9.
- Knight NL, Macdonald B, Sutherland MW. Colonization of durum wheat (*Triticum turgidum* L. var. *durum*) culms exhibiting premature senescence (dead heads) associated with *Fusarium pseudograminearum* crown rot. *Plant Dis.* 2017;101:1788–94.
- Köycü ND, Özyurt HB, Çelen İH. Comparison of aerial and ground spraying applications in controlling *Fusarium* crown rot in wheat. *Int J Agric Biol Eng.* 2024;17:73–80.
- Laday M, Bagi F, Mesterhazy A, Szecsi A. Isozyme evidence for two groups of *Fusarium graminearum*. *Mycol Res.* 2000;104:788–93.
- Lamichhane JR, You MP, Laudinet V, Barbet MJ, Aubertot J-N. Revisiting sustainability of fungicide seed treatments for field crops. *Plant Dis.* 2020;104:610–23.
- Lamprecht S, Marasas W, Hardy M, Calitz F. Effect of crop rotation on crown rot and the incidence of *Fusarium pseudograminearum* in wheat in the Western Cape, South Africa. *Australas Plant Pathol.* 2006;35:419–26.
- Langridge P, Alaux M, Almeida NF, Ammar K, Baum M, et al. Meeting the challenges facing wheat production: The strategic research agenda of the Global Wheat Initiative. *Agronomy.* 2022;12:2767.
- Li X, Liu C, Chakraborty S, Manners JM, Kazan K. A simple method for the assessment of crown rot disease severity in wheat seedlings inoculated with *Fusarium pseudograminearum*. *J Phytopathol.* 2008;156:751–4.
- Li H, Yuan H, Fu B, Xing X, Sun B, et al. First report of *Fusarium pseudograminearum* causing crown rot of wheat in Henan, China. *Plant Dis.* 2012;96:1065–1065.
- Li Y, Li G, Huang Z, Miao J, Liu X. Research progress on the occurrence, damage and prevention of *Fusarium* crown rot caused by *Fusarium pseudograminearum*. *Chin J Pestic Sci.* 2022;24:949–61.
- Li L, Hao R, Yang X, Feng Y, Bi Z. *Piriformospora indica* increases resistance to *Fusarium pseudograminearum* in wheat by inducing phenylpropanoid pathway. *Int J Mol Sci.* 2023a;24:8797.

- Li Q, Guo Z, Hao X, Tang J, Gao Y, et al. Advances in identification methods for resistance to wheat crown rot. *Triticeae Crops*. 2023b;43:591–9.
- Li Y, Tang Y, Xue Z, Wang Y, Shi Y, et al. Resistance risk and resistance-related point mutation in *SdhB* and *SdhC1* of cyclobutirfluram in *Fusarium pseudograminearum*. *J Agric Food Chem*. 2023c;71:1886–95.
- Li Y, Wang Y, Li X, Fan H, Gao X, et al. Resistant risk and resistance-related point mutation in *SdhC1* of pydiflumetofen in *Fusarium pseudograminearum*. *Pest Manag Sci*. 2023d;79:4197–207.
- Li G, Ru B, Zhang L, Li Y, Gao X, et al. Mefentrifluconazole-resistant risk and resistance-related point mutation in *FpCYP51B* of *Fusarium pseudograminearum*. *J Agric Food Chem*. 2024a;72:1516–26.
- Li H, Zhang Y, Qin C, Wang Z, Hao L, et al. Identification and characterization of *FpRco1* in regulating vegetative growth and pathogenicity based on T-DNA insertion in *Fusarium pseudograminearum*. *J Integr Agric*. 2024b;23:3055–65.
- Li Q, Hao X, Guo Z, Qu K, Gao M, et al. Screening and resistance locus identification of the mutant *ferZ22* resistant to crown rot caused by *Fusarium pseudograminearum*. *Plant Dis*. 2024c;108:426–33.
- Li W, Cao S, Sun H, Yang X, Xu L, et al. Genome analyses reveal the secondary metabolites that potentially influence the geographical distribution of *Fusarium pseudograminearum* populations. *Plant Dis*. 2024d;108:1812–9.
- Li Y, Li Y, Wang Y, Yang Y, Qi M, et al. *Flg22*-facilitated PGPR colonization in root tips and control of root rot. *Mol Plant Pathol*. 2024e;25:e70026.
- Li W. Dynamic changes of wheat crown rot pathogens and its integrated control Taian: Shandong Agricultural University; 2021.
- Liu X, Liu C. Effects of drought-stress on *Fusarium* crown rot development in barley. *PLoS ONE*. 2016;11:e0167304.
- Liu Z, You J, Zhao P, Wang X, Sun S, et al. Metabolomics profiling and advanced methodologies for wheat stress research. *Metabolites*. 2025;15:123.
- Lübbertstedt T. Genomics-assisted breeding—a revolutionary strategy for crop improvement. *J Integr Agric*. 2017;16:2674–85.
- Luck J, Spackman M, Freeman A, Tre, bicki P, Griffiths W, et al. Climate change and diseases of food crops. *Plant Pathol*. 2011;60:113–21.
- Lv G, Zhang Y, Ma L, Yan X, Yuan M, et al. A cell wall invertase modulates resistance to *Fusarium* crown rot and sharp eyespot in common wheat. *J Integr Plant Biol*. 2023;65:1814–25.
- Ma J, Li H, Zhang C, Yang X, Liu Y, et al. Identification and validation of a major QTL conferring crown rot resistance in hexaploid wheat. *Theor Appl Genet*. 2010;120:1119–28.
- Ma J, Yan G, Liu C. Development of near-isogenic lines for a major QTL on 3BL conferring *Fusarium* crown rot resistance in hexaploid wheat. *Euphytica*. 2012a;183:147–52.
- Ma J, Zhang C, Liu Y, Yan G, Liu C. Enhancing *Fusarium* crown rot resistance of durum wheat by introgressing chromosome segments from hexaploid wheat. *Euphytica*. 2012b;186:67–73.
- Ma J, Stiller J, Zhao Q, Feng Q, Cavanagh C, et al. Transcriptome and allele specificity associated with a 3BL locus for *Fusarium* crown rot resistance in bread wheat. *PLoS ONE*. 2014;9:e113309.
- Ma J, Chen F, Yin G, Hu H, Wei X, et al. Research status and prospects of genetic breeding for resistance to *Fusarium* crown rot in wheat. *Acta Agron Sin*. 2025;51:2559–69.
- Malosetti M, Zwep LB, Forrest K, van Eeuwijk FA, Dieters M. Lessons from a GWAS study of a wheat pre-breeding program: pyramiding resistance alleles to *Fusarium* crown rot. *Theor Appl Genet*. 2021;134:897–908.
- Martin A, Bovill W, Percy C, Herde D, Fletcher S, et al. Markers for seedling and adult plant crown rot resistance in four partially resistant bread wheat sources. *Theor Appl Genet*. 2015;128:377–85.
- Matny ON. *Fusarium* head blight and crown rot on wheat and barley: losses and health risks. *Adv Plants Agric Res*. 2015;2:00039.
- Melloy P, Hollaway G, Luck J, Norton R, Aitken E, et al. Production and fitness of *Fusarium pseudograminearum* inoculum at elevated carbon dioxide in FACE. *Glob Change Biol*. 2010;16:3363–73.
- Miller SA, Beed FD, Harmon CL. Plant disease diagnostic capabilities and networks. *Annu Rev Phytopathol*. 2009;47:15–38.
- Mitter V, Zhang M, Liu C, Ghosh R, Ghosh M, et al. A high-throughput glasshouse bioassay to detect crown rot resistance in wheat germplasm. *Plant Pathol*. 2006;55:433–41.
- Moya-Elizondo EA, Jacobsen BJ. Integrated management of *Fusarium* crown rot of wheat using fungicide seed treatment, cultivar resistance, and induction of systemic acquired resistance (SAR). *Biol Control*. 2016;92:153–63.
- Mudge AM, Dill-Macky R, Dong Y, Gardiner DM, White RG, et al. A role for the mycotoxin deoxynivalenol in stem colonisation during crown rot disease of wheat caused by *Fusarium graminearum* and *Fusarium pseudograminearum*. *Physiol Mol Plant Pathol*. 2006;69:73–85.
- Murray GM, Brennan JP. Estimating disease losses to the Australian wheat industry. *Australas Plant Pathol*. 2009;38:558–70.
- Nicol J, Rivoal R, Trethowan R, Van Ginkel M, Mergoum M, et al. CIMMYT's approach to identify and use resistance to nematodes and soil-borne fungi, in developing superior wheat germplasm. In: *Wheat in a Global Environment: Proceedings of the 6th International Wheat Conference*, 5–9 June 2000, Budapest, Hungary: Springer; 2001, pp. 381–389.
- Nkongolo K, Narendrula-Kotha R. Advances in monitoring soil microbial community dynamic and function. *J Appl Genet*. 2020;61:249–63.
- O'Sullivan CA, Roper MM, Myers CA, Thatcher LF. Developing actinobacterial endophytes as biocontrol products for *Fusarium pseudograminearum* in wheat. *Front Bioeng Biotechnol*. 2021;9:691770.
- Ons L, Bylemans D, Thevissen K, Cammue BP. Combining biocontrol agents with chemical fungicides for integrated plant fungal disease control. *Microorganisms*. 2020;8:1930.
- Pariyar SR, Erginbas-Orakci G, Dadshani S, Chijioke OB, Léon J, et al. Dissecting the genetic complexity of *Fusarium* crown rot resistance in wheat. *Sci Rep*. 2020;10:3200.
- Paulitz TC. Low input no-till cereal production in the Pacific Northwest of the US: the challenges of root diseases. *Eur J Plant Pathol*. 2006;115:271–81.
- Paulitz TC, Smiley RW, Cook RJ. Insights into the prevalence and management of soilborne cereal pathogens under direct seeding in the Pacific Northwest, USA. *Can J Plant Pathol*. 2002;24:416–28.
- Pestka J. Toxicological mechanisms and potential health effects of deoxynivalenol and nivalenol. *World Mycotoxin J*. 2010;3:323–47.
- Poole G, Smiley R, Paulitz T, Walker C, Carter A, et al. Identification of quantitative trait loci (QTL) for resistance to *Fusarium* crown rot (*Fusarium pseudograminearum*) in multiple assay environments in the Pacific Northwestern US. *Theor Appl Genet*. 2012;125:91–107.
- Poole GJ, Smiley RW, Walker C, Huggins D, Rupp R, et al. Effect of climate on the distribution of *Fusarium* spp. causing crown rot of wheat in the Pacific Northwest of the United States. *Phytopathology*. 2013;103:1130–40.
- Powell JJ, Carere J, Fitzgerald TL, Stiller J, Covarelli L, et al. The *Fusarium* crown rot pathogen *Fusarium pseudograminearum* triggers a suite of transcriptional and metabolic

- changes in bread wheat (*Triticum aestivum* L.). *Ann Bot.* 2017;119:853–67.
- Pu L, Ren H, Ou Y, Ren H, Zeng Q, et al. Screening of germplasms resistant to crown rot and fusarium head blight and the associated SNPs in wheat. *Triticeae Crops.* 2020;40:780–8.
- Qi H, Guo F, Lv L, Zhu X, Zhang L, et al. The wheat wall-associated receptor-like kinase *TaWAK-6D* mediates broad resistance to two fungal pathogens *Fusarium pseudograminearum* and *Rhizoctonia cerealis*. *Front Plant Sci.* 2021;12:758196.
- Qi H, Zhu X, Shen W, Zhang Z. A novel wall-associated kinase *TaWAK-5D600* positively participates in defense against sharp eyespot and fusarium crown rot in wheat. *Int J Mol Sci.* 2023;24:5060.
- Qiu Z, Egidi E, Liu H, Kaur S, Singh BK. New frontiers in agriculture productivity: optimised microbial inoculants and in situ microbiome engineering. *Biotechnol Adv.* 2019;37:107371.
- Rahman M, Davies P, Bansal U, Pasam R, Hayden M, et al. Marker-assisted recurrent selection improves the crown rot resistance of bread wheat. *Mol Breeding.* 2020;40:28.
- Rahman MM, Davies P, Bansal U, Pasam R, Hayden M, et al. Relationship between resistance and tolerance of crown rot in bread wheat. *Field Crop Res.* 2021;265:108106.
- Ray MK, Mishra AK, Mohanta YK, Mahanta S, Chakrabartty I, et al. Nanotechnology as a promising tool against phytopathogens: A futuristic approach to agriculture. *Agriculture.* 2023;13:1856.
- Reddy DS, Chowdary NM. Botanical biopesticide combination concept—a viable option for pest management in organic farming. *Egyptian Journal of Biological Pest Control.* 2021;31:1–10.
- Saad A, Christopher J, Martin A, McDonald S, Percy C. *Fusarium pseudograminearum* and *F. culmorum* affect the root system architecture of bread wheat. *Crop J.* 2023;11:316–21.
- Scott JB, Chakraborty S. Multilocus sequence analysis of *Fusarium pseudograminearum* reveals a single phylogenetic species. *Mycol Res.* 2006;110:1413–25.
- Shah L, Ali A, Yahya M, Zhu Y, Wang S, et al. Integrated control of fusarium head blight and deoxynivalenol mycotoxin in wheat. *Plant Pathol.* 2018;67:532–48.
- Shikur Gebremariam E, Sharma-Poudyal D, Paulitz T, Erginbas-Orakci G, Karakaya A, et al. Identity and pathogenicity of *Fusarium* species associated with crown rot on wheat (*Triticum* spp.) in Turkey. *Eur J Plant Pathol.* 2018;150:387–99.
- Shrestha S, Poudel RS, Zhong S. Identification of fungal species associated with crown and root rots of wheat and evaluation of plant reactions to the pathogens in North Dakota. *Plant Dis.* 2021;105:3564–72.
- Smiley RW, Gourlie JA, Easley SA, Patterson L-M. Pathogenicity of fungi associated with the wheat crown rot complex in Oregon and Washington. *Plant Dis.* 2005a;89:949–57.
- Smiley RW, Gourlie JA, Easley SA, Patterson L-M, Whittaker RG. Crop damage estimates for crown rot of wheat and barley in the Pacific Northwest. *Plant Dis.* 2005b;89:595–604.
- Sørensen JL, Sondergaard TE, Covarelli L, Fuertes PR, Hansen FT, et al. Identification of the biosynthetic gene clusters for the lipopeptides fusaristatin A and *W493 B* in *Fusarium graminearum* and *F. pseudograminearum*. *J Nat Prod.* 2014;77:2619–25.
- Spagnoletti FN, Carmona M, Balestrasse K, Chiochio V, Giacometti R, et al. The arbuscular mycorrhizal fungus *Rhizophagus intraradices* reduces the root rot caused by *Fusarium pseudograminearum* in wheat. *Rhizosphere.* 2021;19:100369.
- Stummer BE, Zhang X, Yang H, Harvey PR. Co-inoculation of *Trichoderma gamsii* A5MH and *Trichoderma harzianum* Tr906 in wheat suppresses in planta abundance of the crown rot pathogen *Fusarium pseudograminearum* and impacts the rhizosphere soil fungal microbiome. *Biol Control.* 2022;165:104809.
- Su Y, Xu X, Wang Y, Wang T, Yu J, et al. Identification of genetic loci and candidate genes underlying *Fusarium* crown rot resistance in wheat. *Theor Appl Genet.* 2025;138:23.
- Summerell B, Burgess L, Klein T, Pattison A. Stubble management and the site of penetration of wheat by *Fusarium graminearum* group 1. *Phytopathology.* 1990;80:877–9.
- Sun C, Liu L, Wang L, Li B, Jin C, et al. Melatonin: a master regulator of plant development and stress responses. *J Integr Plant Biol.* 2021;63:126–45.
- Sun H, Cai S, Deng Y, Cao S, Yang X, et al. Efficacy of cyclobutylfluram in controlling *Fusarium* crown rot of wheat and resistance risk of three *Fusarium* species to cyclobutylfluram. *Pestic Biochem Physiol.* 2024;198:105723.
- Theron JS, van Coller GJ, Rose LJ, Labuschagne J, Swanepoel PA. The effect of crop rotation and tillage practice on *Fusarium* crown rot and agronomic parameters of wheat in South Africa. *Crop Prot.* 2023;166:106175.
- Tunali B, Obanor F, Erginbaş G, Westcott RA, Nicol J, et al. Fitness of three *Fusarium* pathogens of wheat. *FEMS Microbiol Ecol.* 2012;81:596–609.
- Ueno Y, Nakajima M, Sakai K, Ishii K, Sato N, et al. Comparative toxicology of trichothec mycotoxins: inhibition of protein synthesis in animal cells. *J Biochem Tokyo.* 1973;74:285–96.
- Wallwork H, Butt M, Cheong J, Williams K. Resistance to crown rot in wheat identified through an improved method for screening adult plants. *Australas Plant Pathol.* 2004;33:1–7.
- Wang J, Chai J. Structural insights into the plant immune receptors PRRs and NLRs. *Plant Physiol.* 2020;182:1566–81.
- Wang H, Sun S, Ge W, Zhao L, Hou B, et al. Horizontal gene transfer of *Fhb7* from fungus underlies *Fusarium* head blight resistance in wheat. *Science.* 2020;368:eaba5435.
- Wang L, Xie S, Zhang Y, Kang R, Zhang M, et al. The *FpPPRI* gene encodes a pentatricopeptide repeat protein that is essential for asexual development, sporulation, and pathogenesis in *Fusarium pseudograminearum*. *Front Genet.* 2021;11:535622.
- Wang X, Jiao Y, Zhao Y, Hui X, Xu L, et al. Field efficacy of different seed coating agents on wheat crown rot. *Journal of Agriculture.* 2023;13:7.
- Wang J, Fu Y, Guo X, Cheng M, Li H, et al. Spatial transcriptomics uncover coordinated cellular responses to heat stress in developing wheat grains. 2024.
- Wei J, Fang Y, Jiang H, Wu X, Zuo J, et al. Combining QTL mapping and gene co-expression network analysis for prediction of candidate genes and molecular network related to yield in wheat. *BMC Plant Biol.* 2022;22:288.
- Wildermuth G, McNamara R. Testing wheat seedlings for resistance to crown rot caused by *Fusarium graminearum* Group 1. *Plant Dis.* 1994;78:949–53.
- Wildermuth G, Purss G. Further sources of field resistance to crown rot (*Gibberella zeae*) of cereals in Queensland. *Aust J Exp Agric.* 1971;11:455–9.
- Wu T, Guo F, Xu G, Yu J, Zhang L, et al. The receptor-like kinase *TaCRK-7A* inhibits *Fusarium pseudograminearum* growth and mediates resistance to *Fusarium* crown rot in wheat. *Biology (Basel).* 2021;10:1122.
- Wu Y, Wang Y, Han S, Li Q, Kong L. The global regulator FpLaeB is required for the regulation of growth, development, and virulence in *Fusarium pseudograminearum*. *Front Plant Sci.* 2023a;14:1132507.
- Wu Y, Wang Y, Han S, Li Q, Kong L. RNA-Pol II transcription elongation factor FpRtfA is required for virulence and secondary metabolism in *Fusarium pseudograminearum*. *Sustainability.* 2023b;15:11401.
- Wu Y, Han S, Wang Y, Li Q, Kong L. Velvet family protein *FpVelB* affects virulence in association with secondary metabolism in *Fusarium pseudograminearum*. *Cells (Basel).* 2024;13:950.

- Xia H, Chen L, Fan Z, Peng M, Zhao J, et al. Heat stress tolerance gene FpHsp104 affects conidiation and pathogenicity of *Fusarium pseudograminearum*. *Front Microbiol*. 2021;12:695535.
- Xie S, Li H, Zhang W, Wang L, Kang R, et al. Optimum culture conditions in the lab for sexual stage formation of ascospore isolates from perithecia collected in field for *Fusarium pseudograminearum*. *Acta Phytopathol Sin*. 2022;52:77–85.
- Xiong F, Zhu X, Luo C, Liu Z, Zhang Z. The cytosolic acetoacetyl-CoA thiolase *TaAACT1* is required for defense against *Fusarium pseudograminearum* in wheat. *Int J Mol Sci*. 2023;24:6165.
- Xu F, Song Y, Zhou Y, Zhang H, Wang J, et al. Occurrence dynamic and characteristics of *Fusarium* root and crown rot wheat in Henan Province during 2013–2016. *Plant Prot*. 2016;42:126–32.
- Xu F, Yang G, Wang J, Song Y, Liu L, et al. Spatial distribution of root and crown rot fungi associated with winter wheat in the North China Plain and its relationship with climate variables. *Front Microbiol*. 2018;9:1054.
- Xu X, Yu T, Wei J, Ma X, Liu Y, et al. *TaWRKY24* integrates the tryptophan metabolism pathways to participate in defense against *Fusarium* crown rot in wheat. *Plant J*. 2024;120:1764–85.
- Xu C, Guo M, Han X, Ren C, Liu C, et al. Fungal pathogen diversity and fungicide resistance assessment in *Fusarium* crown rot of wheat in the Huanghuai Region of China. *J Agric Food Chem*. 2025;73:2299–311.
- Yan L, Mao Z, Yang M, Feng J, Chen W, et al. Development and application of a TaqMan Probe-based qPCR detection assay for detecting *Fusarium pseudograminearum*. *Phytopathology* 2026
- Yang Y, He X, Hu Y, Hou Y, Niu Y, et al. Resistance of wheat cultivars in Huang-Huai region of China to crown rot caused by *Fusarium pseudograminearum*. *Triticeae Crops*. 2015;35:339–45.
- Yang X, Pan Y, Singh P, He X, Ren Y, et al. Investigation and genome-wide association study for *Fusarium* crown rot resistance in Chinese common wheat. *BMC Plant Biol*. 2019;19:1–14.
- Yang X, Zhong S, Zhang Q, Ren Y, Sun C, et al. A loss-of-function of the dirigent gene *TaDIR-B1* improves resistance to *Fusarium* crown rot in wheat. *Plant Biotechnol J*. 2021;19:866.
- Yang H, Cui S, Wei Y, Li H, Hu J, et al. Antagonistic effects of *Talaromyces muroii* TM28 against *Fusarium* crown rot of wheat caused by *Fusarium pseudograminearum*. *Front Microbiol*. 2024a;14:1292885.
- Yang X, Cao S, Sun H, Deng Y, Zhang X, et al. The critical roles of the *Zn2Cys6* transcription factor Fp487 in the development and virulence of *Fusarium pseudograminearum*: a potential target for *Fusarium* crown rot control. *Microbiol Res*. 2024b;285:127784.
- Zhang H, Mittal N, Leamy L, Barazani O, Song B. Back into the wild—apply untapped genetic diversity of wild relatives for crop improvement. *Evol Appl*. 2017;10:5–24.
- Zhang J, Tang M, Xia M, Lin Q, Sun R, et al. Combination of *Bacillus subtilis* YB-05 and Shenqinmycin for integrated control of wheat crown rot. *Chin J Biol Control*. 2018a;34:866.
- Zhang Y, Chen D, Wang S, Tian L. A promising trend for field information collection: an air-ground multi-sensor monitoring system. *Inf Process Agric*. 2018b;5:224–33.
- Zhang J, Tang M, Xia M, Lin Q, Sun R, et al. Response of *Fusarium pseudograminearum* to biocontrol agent *Bacillus velezensis* YB-185 by phenotypic and transcriptome analysis. *J Fungi (Basel)*. 2022a;8:763.
- Zhang N, Yuan S, Zhang Q, Liu W, Zhou Y, et al. Screening fungicides for controlling wheat crown rot caused by *Fusarium pseudograminearum* across Hebei province in China. *Agriculture (Basel)*. 2022b;12:1643.
- Zhang L, Yan X, Yuan M, Jian J, Wei J, et al. The resistance investigation and mapping analysis of *Fusarium* crown rot for wheat accessions. *Plant Genet Resour*. 2024;20:184–92.
- Zhao J, Peng M, Chen W, Xing X, Shan Y, et al. Transcriptome analysis and functional validation identify a putative bZIP transcription factor, *Fpkapc*, that regulates development, stress responses, and virulence in *Fusarium pseudograminearum*. *Phytopathology*. 2022a;112:1299–309.
- Zhao L, Lv G, He Y, Feng H, Peng H. Occurrence status and integrated control measures of wheat crown rot in Henan Province. *China Plant Prot*. 2022c;42:49–51.
- Zhao X, Hou D, Xu J, Wang K, Hu Z. Antagonistic activity of fungal strains against *Fusarium* crown rot. *Plants*. 2022d;11:255.
- Zhao J, Xia H, Peng M, Fan Z, Yin Y, et al. Identification and functional analysis of transcription factors *FpAPSES* in *Fusarium pseudograminearum*. 2021.
- Zhao J, Peng M, Zhang S, Shan Y, Xing X, et al. C₂H₂ zinc finger transcription factor FpCzf7 is involved in growth and pathogenicity of *Fusarium pseudograminearum*. *Biotechnol Bull*. 2022b; 216–224.
- Zheng Z, Kilian A, Yan G, Liu C. QTL conferring *Fusarium* crown rot resistance in the elite bread wheat variety EGA Wylie. *PLoS ONE*. 2014;9:e96011.
- Zheng Z, Ma J, Stiller J, Zhao Q, Feng Q, et al. Fine mapping of a large-effect QTL conferring *Fusarium* crown rot resistance on the long arm of chromosome 3B in hexaploid wheat. *BMC Genomics*. 2015;16:1–7.
- Zheng Z, Gao S, Zhou M, Yan G, Liu C. Enhancing *Fusarium* crown rot resistance by pyramiding large-effect QTL in common wheat (*Triticum aestivum* L.). *Mol Breed*. 2017;37:107.
- Zheng D, Xu J, Lu Y, Chen H, Chu Q, et al. Recent progresses in plant single-cell transcriptomics. *Crop Design*. 2023;2:100041.
- Zhou M, Zhang P, Yang X, Chen D, Yao J. Analysis of QTLs for the resistance to crown rot in wheat. *Triticeae Crops*. 2021;41:538–43.
- Zhou F, Cui Y, Wang B, Zhou Y, Li S, et al. Baseline sensitivity and potential resistance mechanisms for *Fusarium pseudograminearum* to fludioxonil. *Plant Dis*. 2022;106:2138–44.
- Zhou H, Cao W, Li Y, Zhang Q, Liu F, et al. Research progress on the analysis of resistance genes and mechanisms of wheat *Fusarium* crown rot. *Front Plant Sci*. 2025;16:1603842.

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