Plant Archives Vol. 26, No. 1, 2026 pp. 1586-1594 e-ISSN:2581-6063 (online), ISSN:0972-5210

Plant Archives

Journal homepage: http://www.plantarchives.org

DOI Url : https://doi.org/10.51470/PLANTARCHIVES.2026.v26.no.1.217

PATHOGENIC VARIABILITY OF *FUSARIUM* ISOLATES INFECTING CEREALS AND THEIR ROLE IN CROP YIELD LOSSES: A REVIEW

Sulagna Sahoo[1]*, Amrita Dasgupta[1], Sangeeta Sahu[1], Ramesh kumar[1], Piyush Kumar[2] and Bhabani Shankar Rout[3]

[1]Department of Plant Pathology, Institute of Agricultural Sciences, SOA University Bhubaneswar, Odisha, India.
[2]Department of Plant Pathology, Sher-E-Kashmir University of Agricultural Sciences and Technology, Kashmir, India.
[3]Department of Plant pathology, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India.
*Corresponding author E-mail : sulagnasahoo516@gmail.com

(Date of Receiving-01-12-2025; Date of Revision-28-01-2026; Date of Acceptance-18-02-2026)

ABSTRACT

Fusarium is one of the most devastating pathogens of the major cereal crops, including wheat, rice, maize, barley, oats and sorghum, by declining global cereal production and affecting both the quality as well as quantity of the production. Fusarium head blight (FHB) is the most significant among the diseases caused by this fungus due to its pathogenicity, mycotoxin production, endemic nature and economic impact. The aggressive pathogenic rate of *Fusarium* arises due to its diverse species complexes, extensive genetic diversity, and distinct chemotypic profiles. Different *Fusarium* species complexes, such as the *Fusarium graminearum* species complex (FGSC), FASC and the *F. pseudograminearum* complex consist of multiple cryptic species that differ in host specificity, geographic distribution, toxin production and aggressiveness. Distinctness in trichothecene chemotypes, reportedly 15-ADON, 3-ADON, fumonisins, NIV and other toxins, affects disease development, disease severity, risks to food security and feed safety. The structure and dominance of *Fusarium* populations are framed across agro-ecological regions by the interaction of agronomic practices, host genotype, and environmental factors. Understanding the disease dynamics of *Fusarium* requires assessment of pathogenic variability through integrated biochemical, pathological, molecular, and cultural approaches. Such information is critical for the development of durable host resistance, appropriate epidemic forecasting and implementation of sustainable disease management strategies. Therefore, elucidating the process of pathogenic and toxigenic diversity of *Fusarium* is fundamental to eliminate crop loss as well as safeguarding cereal-based food security along with changing agricultural practices.

Key words : Cereal crops, Crop yield losses, Fusarium isolates, Pathogenic variability, Toxigenic diversity.

Introduction

The major cereal crops, including wheat, maize, barley, and oats, are at high risk from the most devastating diseases caused by *Fusarium* species (Parry *et al*., 1995; Dean *et al*., 2012). Among these diseases, serious economic damage is caused by Fusarium head blight (FHB), which is considered as a minor grain disease (McMullen *et al*., 1997; Goswami and Kistler, 2004). Severe FHB epidemics have led to large scale yield losses, quality deterioration, and socio-economic consequences in the major cereal growing countries (Goswami and Kistler, 2004; Parry *et al*., 1995). The rapid spread of FHB is emerging as a global concern that is influenced by the changing environment, changing cultural practices, as well as the highly virulent *Fusarium* population (McMullen *et al*., 1997; Dean *et al*., 2012). The duration and severity of the disease vary significantly with the pathogenic heterogeneity among the *Fusarium* populations affecting cereals (Aoki *et al*., 2012; O'Donnell *et al*., 2004). The diverse *Fusarium graminearum* species complex (FGSC) consists of several phenotypically different traits, showing distinct variations in virulence, aggressiveness, host specificity, and geographical distribution (Aoki *et al*., 2012). According to phylogenetic research, complex pathogen

communities are formed by the coexistence of different species of the pathogen, which causes hindrances in disease detection and management strategies (O'Donnell *et al*., 2004; Boutigny *et al*., 2011). Some population studies reveal that different FGSC members are found in several agro-ecological environments, leading to distinct evolutionary adaptation and structuring at the biogeographical level (Boutigny *et al*., 2011; Aoki *et al*., 2012). The pathogenic diversity of *Fusarium* is associated with the chemotypes of several mycotoxins, namely trichothecene profiles such as NIV, 3-ADON and 15-ADON (Foroud and Eudes, 2009). Different chemotypes of each mycotoxin regulates the amount of toxin production, environmental fitness, and aggressiveness of the pathogen by influencing the disease severity of major cereal crops (Foroud and Eudes, 2009; Dean *et al*., 2012). The aggressive chemotypes, especially those that produce 3-ADON, influence the transmission speed of the disease and increase its epidemic potential in certain areas (Kelley *et al*., 2020). The toxin-mediated pathogenicity adversely affects food safety, reduces grain production, and increases economic losses (Goswami and Kistler, 2004; Dean *et al*., 2012). Recent studies reveal the failure of resistant varieties against newly emerging *Fusarium* species, which highlights the need to integrate pathogenic and chemotypic variability for sustainable control practices (Dean *et al.,* 2012; Parry *et al*., 1995).

Hence, to understand the mechanisms of disease development and spread, a well-planned investigation is required that combines physiological, epidemiological, and molecular information (Kelley *et al*., 2020; O'Donnell *et al*., 2004). Therefore, assessing the pathogenic diversity of *Fusarium* isolates infecting major cereals is essential for reducing yield losses in world production, forecasting epidemic threats, and breeding resistant varieties (Aoki *et al*., 2012; McMullen *et al*., 1997).

Biology and Taxonomy of *Fusarium* spp. Infecting cereals

Species Complexes

Several *Fusarium* species complexes infecting different cereals show similarities in their morphological traits but differ in their genetic constitution (van der Lee *et al*., 2015). According to some molecular studies, the *Fusarium graminearum* species complex (FGSC) is the most important group associated with Fusarium head blight (FHB) in global cereal producing areas (Del Ponte *et al*., 2022). Multilocus sequence analysis is required to resolve the cryptic species of the FGSC, by the use of markers such as FGSC, TEF1-á has proven essential (Wang *et al*., 2011; Somma *et al*., 2014). The above markers are used to differentiate species that are genetically distinct but morphologically identical (O'Donnell *et al*., 2008). The *Fusarium avenaceum* species complex (FASC) is another complex that shows a considerable level of intraspecific variability and has low phylogenetic hierarchy (Kulik *et al*., 2011). According to multilocus phylogenetic studies, the *Fusarium pseudograminearum* complex associated with crown rot, which exists as a single distinct lineage (Scott & Chakraborty, 2006). Species-specific PCR assays are used for the identification of *Fusarium* species infecting cereals, which is further supported by molecular characterization (Edwards, 2012). Hence, molecular methods are used to determine findings related to genetically cohesive groups within species complexes (Bugingo *et al*., 2025).

Host Range

Cereal-infecting *Fusarium* species mainly infect oats, wheat, triticale, barley and maize, affecting a wide host range (van der Lee *et al*., 2015). Members of the FGSC cause Fusarium head blight in barley and wheat, particularly in temperate and subtropical regions. (Del Ponte *et al*., 2022). Although, FGSC members can infect both barley and oats, *Fusarium pseudograminearum* is the main causal agent of crown rot in dryland wheat systems (Scott and Chakraborty, 2006). Frequent secondary infections occur across a wide range of cereals, particularly under cool and humid conditions, caused by *Fusarium avenaceum* and *Fusarium poae* (Kulik *et al*., 2011; Edwards, 2012). According to Indian research, a wide variety of phytopathogenic and toxic Fusarium species are seen colonizing on some cereals like tiny millets, sorghum, and maize (Nagaraja *et al*., 2016). Ecological selection pressures, chemotype of variety, and genetic constitution all these factors contribute to Fusarium's adaptation across cereals growing areas (Wang *et al*., 2011; Armer *et al*., 2024).

Important Cereal-Pathogenic *Fusarium* Lineages

Several type of evolutionarily distinct lineages occur within *Fusarium* species due to their varying pathogenicity, distinct chemotypic profiles, and worldwide occurrence. There have been numerous records of the 3-ADON, 15-ADON and NIV chemotype lineages of FGSC in cereals and they exhibit variations in their aggressiveness as well as mycotoxin production (van der Lee *et al*., 2015; Del Ponte *et al*., 2022). *Fusarium graminearum* s.s. is one of the most aggressive lineage causing FHB in wheat and barley worldwide (Somma *et al*., 2014). Recently, diversification within the FGSC has been observed among identified species such as *Fusarium*

acaciae-mearnsii and lineages originating from Ethiopia (O'Donnell *et al*., 2008). *Fusarium pseudograminearum* exhibits genetically similar but extremely virulent clones which have adapted to drought stressed wheat by producing crown rot symptoms (Scott and Chakraborty, 2006). Multilocus phylogenetics has revealed several divergent lineages of *F. avenaceum* inside the FASC, suggesting a significant evolutionary potential for host adaptability (Kulik *et al*., 2011). According to some ongoing surveillance, it is further highlighted by recent analyses that show new lineages with broader host ranges as well as the potential for increasing pathogenicity across grains (Armer *et al*., 2024; Nagaraja *et al*., 2016).

Epidemiology and Disease Cycle of *Fusarium* in Cereal crops

The interaction among the pathogen, susceptibility of host, and environmental conditions determines the severity of Fusarium diseases in cereal crops (McMullen *et al.*, 1997). Airborne conidia or ascospores, dispersed by splashing of water onto vulnerable parts of cereal plants especially the florets during anthesis, initiate the infection process (Parry *et al*., 1995). With the onset of favourable conditions, the spores germinate on the flower surface, leading to hyphal penetration through the anthers, glumes, lemma, or palea, resulting in colonization of the spike (Alisaac and Mahlein, 2023). Following penetration, the pathogen propagates through the vascular tissues and the rachis, allowing for the quick spread of the fungus across several spikelets (Goswami and Kistler, 2004). Spore germination and early infection processes are considerably enhanced by favourable weather during flowering, especially moderate temperatures along with prolonged relative humidity (Paul *et al*., 2005). As the main overwintering structures and inoculum reservoirs, infected crop leftovers are the main source of survival for Fusarium species between cropping seasons (Sutton, 1982). On residual plant material, the fungus forms perithecia that produce ascospores, which are forcibly discharged and dispersed over long distances by wind, contributing to regional disease spread (Nganje, 2002). Conidia that develop on infected plant tissues or agricultural debris also function as secondary inoculum, primarily traveling shorter distances via rain splash (Dill-Macky and Jones, 2000).

The amount of inoculum available for future epidemics is greatly increased by cropping techniques including reduced tillage, maize–wheat rotation and retention of infected residues (Njeru *et al*., 2007). Conservation agriculture systems frequently exhibit higher inoculum pressure and a higher risk of Fusarium illness due to this residue-based survival strategy(Njeru *et al*., 2007). The timing, severity and dissemination of Fusarium outbreaks throughout cereal-growing regions are determined by environmental factors (Xu, 2003).Temperatures between 20 and 30°C and extended wetness are ideal for infection because they promote sporulation, spore dissemination, and colonization of spike tissues (Parry *et al*., 1995). Rainfall events that occur during anthesis lengthen the duration of spike wetness and promote inoculum distribution, which raises infection rates (Paul *et al*., 2005). Major epidemic outbreaks in North America, Europe, and Asia have been attributed to changes in temperature regimes, humidity, and precipitation patterns (McMullen *et al*., 1997). Regional epidemiological analysis show that disparities in disease severity and epidemic timing are caused by different climate conditions on different continents (Njeru *et al*., 2007). The disease cycle concludes when diseased grains become lightweight, shrivelled and frequently contaminated with mycotoxins, which reduce seed potentially and quality act as inoculum if used in planting (Goswami and Kistler, 2004). The recurrence and persistence of Fusarium diseases in worldwide grain production systems are supported by an integrated cycle of survival on residues, effective dispersal mechanisms, weather-driven infection, and ongoing pathogen build-up (Wegulo *et al*., 2015).

Concept of Pathogenic Variability in *Fusarium*

The variations in virulence, chemotype, host specificity, and aggressiveness across isolates within a species or species complex are referred to as pathogenic variability in Fusarium (Carter *et al*., 2002; Qu *et al*., 2008). Pathogen populations are modified throughout time by host driven, environmental, and genetic variables (Wang *et al*., 2011). Understanding this variability is essential for predicting disease outbreaks, improving resistance breeding, and implementing effective management strategies (Bai *et al*., 2018).

Role of Genetic Diversity

Genetic diversity is one of the important basis for assessing pathogenic variability in cereal infecting Fusarium species because diverse genomes encode different virulence factors, toxin pathways, and host-adaptation mechanisms (Wang *et al*., 2011). Novel multilocus haplotypes generated by the sexual recombination within *F. graminearum* species complex contribute to spontaneous evolutionary changes in aggressiveness (Wang *et al*., 2011; Oghenekaro *et al*., 2021). Numerous evolutionary lineages within FGSC have been identified with the help of population genetic analysis,

each of which is associated with the unique geographic locations and pathogenic profiles (Wang *et al*., 2011; Xi *et al*., 2021). Genetic differences in the trichothecene biosynthetic cluster cause chemotype differentiation, specifically the distribution of 3-ADON, 15-ADON, and NIV groups, which affect toxin accumulation and illness severity in cereals (Goswami *et al*., 2002; Vanheule *et al*., 2017). For an instance, as compared to 15-ADON isolates, 3-ADON isolates generally exhibit faster mycelial development and higher DON potential, which can lead to more severe field epidemics (Alexander *et al*., 2011; Oghenekaro *et al*., 2021). Additionally, molecular research has demonstrated that genetic subpopulations within a single species, like *F. poae*, varied considerably in the presence and expression patterns of toxin genes (Vanheule *et al*., 2017; Pasquali *et al*., 2014). These genomic differences explain why isolates vary widely in aggressiveness, with some causing extensive head blight or wilt, while others remain weak or non-pathogenic under similar conditions (Pasquali *et al*., 2014; Qu *et al*., 2008).

Influence of Environment on Pathogenic Variability

In cereal ecosystems, environmental variables directly influence the pathogenic behaviour, spread and dominance of Fusarium isolates (Xu and Nicholson, 2008; Qu *et al*., 2008). Infection development, sporulation and mycotoxin generation are all greatly increased by ecological factors such high humidity, dampness, and moderate to warm temperatures (Xu and Nicholson, 2008; Alexander *et al*., 2021). Regional chemotype prevalence is also influenced by environmental selection pressures, with warmer climates frequently preferring chemotypes that spread more quickly, such as 3-ADON (Oghenekaro *et al*., 2021; Pasquali *et al*., 2014). By increasing spore dissemination and encouraging spikelet colonization, rainfall and moisture during anthesis have a significant impact on the onset of disease (Xu and Nicholson, 2008; Qu *et al*., 2008). Different Fusarium populations can thrive and compete in microenvironments created by agronomic factors as conservation tillage, cereal-based crop rotations, and maize residue retention. Over time, this can result in changes in lineage dominance (Xu and Nicholson, 2008; Wang *et al*., 2011). The movement and persistence of airborne spores across growing regions are further influenced by crop density, wind patterns, and soil type (Alexander *et al*., 2021; Vanheule *et al*., 2017). Therefore, in addition to influencing disease incidence, environmental factors may cause evolutionary changes in pathogen populations, which can lead to the formation of Fusarium strains that are more toxic or aggressive (Qu *et al*., 2008; Oghenekaro *et al*., 2021).

Influence of Host Genotype

Host genotype exerts a significant role on the expression of pathogenic diversity because different cereal cultivars possess varying levels of genetic resistance and structural defences (Bai, 2018; Wu *et al*., 2022). Resistance QTLs such as Fhb1, Fhb2, and Fhb5 inhibit fungal spread within spikes and suppress DON accumulation, forcing Fusarium isolates to adapt or lose their ability to compete (Bai, 2018; Wu *et al*., 2022). Some Fusarium isolates show specialization exhibit specialization towards specific host genotypes, resulting in strong genotype × isolate interactions where aggressiveness varies from cultivar to cultivar (Carter *et al*., 2002; Xi *et al*., 2021). Strong selection pressure from resistant strains can cause Fusarium populations to shift toward isolates that can overcome resistance, hastening the evolution of virulence traits (Bai and Shaner, 2018; Qu *et al*., 2008). Certain isolates are more able to take advantage of weakened or stressed plants than others due to the host's nutritional status and physiological stage during infection (Wu *et al*., 2022; Xu and Nicholson, 2008). Therefore, interactions between pathogen diversity and host genetics are crucial in deciding which Fusarium lineages endure throughout grain production landscapes and influencing disease outcomes (Bai and Shaner, 2018; Qu *et al*., 2008).

Methods for Assessing Pathogenic Variability in *Fusarium*

Cultural and Morphological characterization

A fundamental basis for distinguishing the Fusarium isolates and identifying differences in colony characteristics, sporulation, pigmentation and growth rates is still coming under cultural and morphological evaluation (Leslie and Summerell, 2006). Comparison of macroconidia, microconidia, chlamydospores, and colony morphotypes, which frequently associated with species level variations and pathogenic potential, is only possible by standardized media like PDA, SNA and CLA (Leslie and Summerell, 2006). Particularly among the species complexes like *F. graminearum* and *F. oxysporum*, where minor phenotypic variations may indicate underlying genetic diversity, these morphological characteristics are helpful for first grouping of isolate prior to molecular confirmation (Velásquez-Zapata *et al*., 2022). In order to ensure that phenotypic variability is sufficiently recorded across populations, such traditional measures also enable the selection of representative isolates for the pathogenicity tests as well as further molecular investigations (Oliveira *et al*., 2021).

Pathogenicity Assays

Pathogenicity assays are crucial for linking phenotype to genetic diversity and for the measurement of virulence and variability across isolates under controlled conditions (Covarelli *et al*., 2013). Seedling assays, spray inoculation, spike inoculation tests, point inoculation, and colonized grain inoculum assays are successful examples of some common bioassays that enable evaluation of disease severity, lesion length and rate of infection development (Šarìvić *et al*., 2023). For fusarium head blight (FHB), spray and point inoculation techniques are effectively used because they accurately distinguish aggressiveness among isolates as well as the field relevant infection processes (Šaròviæ *et al*., 2023). For an efficient screening, seedling tests are helpful since they allow for accurate comparison for pathogenicity of isolates and detection of early host responses (Covarelli *et al*., 2013). Recent studies have also integrated metabolomic profiling during pathogenicity tests to link the specific toxins or metabolites with higher virulence levels among isolates of *F. graminearum* and *F. culmorum* (Khanal *et al*., 2024).

Physiological and Biochemical Markers

Physiological and biochemical traits provide a clearcut understanding for distinguishing pathogenic variants, especially when morphological features overlap with each other (Oliveira *et al*., 2022). Among the cereal-infecting Fusarium species, mycotoxin profiling specifically DON, 3-ADON, 15-ADON, and NIV production act as a major biochemical predictor of aggressiveness and lineage divergence (Khanal *et al*., 2024). Pectinases, cellulases and amylases are some great examples of enzymatic activity that can deviate greatly between isolates and lead to variations in host tissue colonization (Oliveira *et al*., 2022). For the better understanding of physiological diversity among pathogen populations, these biochemical markers are mostly utilized in combination with pathogenicity experiments to assess the correlations between metabolite production and disease severity (Khanal *et al*., 2024). Such integrated approaches help to identify pathotypes that indicate higher risk in cereal ecosystems due to enhanced metabolic efficiency and toxin biosynthesis (Velásquez-Zapata *et al*., 2022).

Molecular Markers for Genetic characterization

Molecular methods provide the accurate resolution for assessment of genetic variability in Fusarium populations (Oliveira *et al*., 2022). Molecular sequencing techniques such as ITS, TEF1-α and RPB2 are used to distinguish species and lineages within species complexes (Akgül *et al*., 2024). Fine scale diversity and virulence associated genetic clusters are revealed by advanced markers which includes whole-genome sequencing, SSRs, and SNPs (Murodova *et al*., 2024; Velásquez-Zapata *et al*., 2022).

Mycotoxin Production variability among *Fusarium* isolates

Fusarium species complex produces a wide range of mycotoxins, which include some important compounds such as deoxynivalenol (DON), nivalenol (NIV), zearalenone (ZEN), fumonisins (FB1, FB2), moniliformin (MON) and beauvericin (BEA). (Bennett and Klich, 2003; Desjardins, 2006). Variations in biosynthetic gene clusters (such as the TRI and FUM clusters), regulatory elements and the presence or absence of sequence polymorphisms of significant pathway genes among different isolates and species all contribute to variation in mycotoxin profiles (Stêpieñ *et al*., 2012; Alexander *et al*., 2009). Trichothecene chemotypes such as NIV, 15-ADON and 3-ADON are major instances of intraspecific toxigenic variability within the *Fusarium graminearum* species complex, where as the chemotype distributions differing geographically and temporally (Alexander *et al*., 2009). Other Fusarium species, such as *F. proliferatum, F. verticillioides,* and *F. equiseti*, produce non-trichothecene toxins such ZEN, fumonisins and MON which also exhibit variations among the isolate, in amount and co-occurrence patterns (Waœkiewicz and Stêpieñ, 2012).

Link with pathogenic fitness

Higher mycotoxin production has been mentioned in numerous studies to enhance the pathogenic fitness, where toxins function as virulence factors that leads to host colonization, the development of symptoms, or a competitive edge over other bacteria (Desjardins, 2006). For example, DON is involved in boosting spread of pathogen within wheat spikes by inhibiting host defences mechanism and enabling necrotrophic development and shifts to more aggressive 3-ADON populations have been linked with increased disease severity in the field (Ward *et al*., 2002). Similarly, increased stalk and ear colonization in particular genotypes of maize diseases is related with fumonisin production, which links toxin generation to fitness of isolates on that host (Marasas, 2001; Bryła *et al*., 2022). However, the relationship is not universally linear: some high toxin producing isolates are not necessarily the most aggressive under all condition since production of toxin can be context-dependent and differs by environmental variables and host genotype (Stêpieñ *et al*., 2012)

Table 1 : Pathogenic variability of Fusarium isolates infecting Major Cereal crops.

Cereal Crop	Major Fusarium Species involved	Key Pathogenic traits / Variability observed	Associated Mycotoxins/ Chemotypes	Representative References
Wheat	*F. graminearum*, *F. culmorum*, *F. avenaceum*, *F. poae*	High variation in aggressiveness; differences in spike infection efficiency; chemotype shifts (3-ADON vs. 15-ADON); lineages differ in DON production and spread.	DON, 3 ADON, 15-ADON, NIV	Bai *et al*. (2018); Vanheule *et al*. (2017)
Barley	*F. graminearum*, *F. culmorum*, *F. langsethiae*	Host-dependent aggressiveness; variation in pre-harvest colonization; differences in symptom expression and kernel discoloration.	T-2, HT-2, DON	Oghenekaro *et al*. (2021); Xu and Nicholson (2008)
Maize	*F. verticillioides*, *F. proliferatum*, *F. graminearum*, *F. boothii*	Strong genotype × environment interactions; fumonisin variability; distinct ear rot vs. stalk rot pathotypes.	Fumonisins (FB1, FB2), DON	Qu *et al.* (2008); Wang *et al*. (2011)
Oats	*F. poae*, *F. langsethiae*, *F. sporotrichioides*	Low symptom visibility but high toxin accumulation; significant differences in T-2/HT-2 production among isolates.	T-2, HT-2	Edwards (2012)
Rice	*F. asiaticum*, *F. fujikuroi*, *F. proliferatum*	Distinct host-adapted lineages; bakanae vs. grain infection types; variation in growth-promoting vs. pathogenic traits.	Fumonisins, moniliformin	Wulff *et al*. (2010)
Sorghum	*F. thapsinum*, *F. proliferatum*, *F. verticillioides*	Variation in stalk rot severity; chemotype and ear rot differences; environment-driven shifts in toxigenic profiles.	Fumonisins, moniliformin	Nor *et al*. (2011)

Risk to food safety

Variability in mycotoxin production poses significant food-safety challenges because mixed infections often lead to numerous toxins co-occurring in cereal grains, increasing toxicity risks for consumers and livestock (Bennett and Klich, 2003; Palumbo *et al*., 1999). ZEN has estrogenic effects, NIV and related trichothecenes have been linked to acute gastrointestinal toxicity, DON has been linked to vomiting, immunological dysregulation, and feed refusal and fumonisins have been linked to long-term conditions like neural tube defects and oesophageal cancer (Marasas, 2001). Changing climatic circumstances and alterations in chemotype dominance can modify regional toxin contamination patterns, making mycotoxin surveillance a dynamic and necessary component of cereal safety management (Desjardins, 2006). The most accurate evaluation of toxigenic risk at the field and storage levels is provided by integrated detection systems that combine toxin-gene screening with sophisticated analytical techniques like LC-MS/MS (Kulik, 2011).

Impact of Pathogenic Variability on Disease Severity and Crop Yield losses

Quantitative Yield impacts

According to some research findings on wheat FHB, depending on genotype and environment, most virulent *F. graminearum* lineages can reduce weight of grains by 15–60% (Wegulo *et al*., 2015). Similarly, under favourable conditions, maize yield losses associated with strains of *F. verticillioides* that produce fumonisin vary from 10 to 40%, revealing substantial genotype × isolate interactions (Shaikh, 2011). Additionally, extremely vigorous NIV-*producing F. asiaticum* isolates can cause a 30% yield decrease in rice and barley systems (Jang *et al*., 2019). In general, colonization rate, infection efficiency, and ensuing grain filling losses are all directly impacted by pathogenic diversity (Parry *et al*., 1995).

Influence on Grain quality

Variation in Fusarium pathogenicity directly contribute to decreasing grain quality due to variation in mycotoxin profiles produced by different haplotypes or chemotypes

(Goswami and Kistler, 2004). In comparison with ZEN-dominant strains, isolates that produce DON and NIV significantly lower the test weight, kernel soundness, and milling quality in wheat and barley (Pestka, 2010). Grain hardness as well as storability of maize are severely compromised by fumonisin-rich infections, leading to a degradation of up to 80% in commercial markets (Nelson *et al.*, 1993). Variation in pathogenic fitness greatly affected by seed germination rates, with aggressive isolates causing up to 50% reduction in viability (Desjardins, 2006). Grain contamination is therefore a major quality concern across all cereal growing regions due to isolate-specific variations in virulence and metabolite production (Ferrigo *et al.*, 2016).

Regional Case studies

Fusarium isolates that infect maize, rice, and small millets in India, especially eastern India, have been shown to exhibit pathogenic diversity with notable variations in toxin production and aggressiveness (Navale *et al*., 2023). According to surveys carried out in southern India, it has been revealed that rainfed maize is often infected by fumonisin-producing *Fusarium verticillioides*, which is associated with ear rot and significant yield losses during extremely humid Kharif seasons (Nayaka *et al*., 2010). Recent research indicates, *F. fujikuroi* species complex-induced rice spikelet rot disease results in pathotype variation associated with the interaction of environmental factors such as temperature as well as humidity, that affect sporulation and panicle infertility rates (Cao *et al*., 2025). The primary cause of seed and grain discolouration in cereal crops in Karnataka is Fusarium and other fungal diseases, which are also linked to decrease test weight, lower market value and lower germination percentage (Nagaraja *et al*., 2016). Regional temperature deviation, local cropping systems and host genotype selection are tend to be responsible for major pathogenic divergence and yield risk in India's cereal systems (Navale *et al*., 2023).

Conclusion

Fusarium has become one of the most devastating pathogens worldwide, especially for cereal crops, due to its wide host range, complex disease cycle and extensive pathogenic variability. Multiple species are associated with Fusarium species complexes; however, the *Fusarium graminearum* species complex shows pronounced differences in host adaptation, aggressiveness and mycotoxin production. These variations result in differential disease severity and significant financial losses in agricultural production. Highly virulent and toxin-producing species continue to emerge as a consequence of changing climatic conditions, evolving agronomic practices, and host genotype interactions. Pathogenic variability in Fusarium, particularly involving trichothecene and fumonisin chemotypes, leads to reductions in both the quality and quantity of agricultural produce, while enhancing the epidemic potential of the pathogen.

References

Akgül, D.S., Önder S., Savaş N.G., Yýldýz M., Bülbül İ. and Özarslandan M. (2024). Molecular identification and pathogenicity of *Fusarium* species associated with wood canker, root and basal rot in Turkish grapevine nurseries. *J. Fungi*, **10(7)**, 444

Alexander, N.J., McCormick S.P., Waalwijk C., van der Lee T. and Proctor R.H. (2011). The genetic basis for 3-ADON and 15-ADON trichothecene chemotypes in Fusarium. *Fungal Genetics and Biology*, **48(5)**, 485-495.

Alexander, N.J., Proctor R.H. and McCormick S.P. (2009). Genes, gene clusters, and biosynthesis of trichothecenes and fumonisins in Fusarium. *Toxin Reviews*, **28(2-3)**, 198-215.

Alisaac, E. and Mahlein A.K. (2023). Fusarium head blight on wheat: Biology, modern detection and diagnosis and integrated disease management. *Toxins*, **15(3)**, 192.

Aoki, T., Ward T.J., Kistler H.C. and O'donnell K. (2012). Systematics, phylogeny and trichothecene mycotoxin potential of Fusarium head blight cereal pathogens. *JSM Mycotoxins*, **62(2)**, 91-102.

Bai, G., Su Z. and Cai J. (2018). Wheat resistance to Fusarium head blight. *Canadian J. Plant Pathol.*, **40(3)**, 336–346. https://doi.org/10.1080/07060661.2018.1476411

Bennett, J.W. and Klich M. (2003). Mycotoxins. *Clin. Microbiol. Rev.*, **16(3)**, 497–516.

Boutigny, A.L., Ward T.J., Van Coller G.J., Flett B., Lamprecht S.C., O'Donnell K. and Viljoen A. (2011). Analysis of the *Fusarium graminearum* species complex from wheat, barley and maize in South Africa provides evidence of species-specific differences in host preference. *Fungal Genetics and Biology*, **48(9)**, 914-920.

Bryła, M., Pierzgalski A., Zapaœnik A., Uwineza P.A., Ksieniewicz-WoŸniak E., Modrzewska M. and Waœkiewicz A. (2022). Recent research on Fusarium mycotoxins in maize—A review. *Foods*, **11(21)**, 3465.

Bugingo, C., Infantino A., Okello P., Perez-Hernandez O., Petroviæ K., Turatsinze A.N. and Moparthi S. (2025). From morphology to multi-omics: A new age of Fusarium research. *Pathogens*, **14(8)**, 762.

Cao, Q., Wu J., Ma T., Mao C. and Zhang C. (2025). Characterization of Fusarium Diversity and Head Microbiota associated with Rice Spikelet Rot Disease. *Plants*, **14(10)**, 1531.

Carter, J.P., Rezanoor H.N., Holden D., Desjardins A.E., Plattner R.D. and Nicholson P. (2002). Variation in pathogenicity associated with the genetic diversity of *Fusarium graminearum*. *Europ. J. Plant Pathol.*, **108(6)**, 573-583.

Chandra Nayaka, S., Udaya Shankar A.C., Niranjana S.R., Wulff E.G., Mortensen C.N. and Prakash H.S. (2010). Detection and quantification of fumonisins from *Fusarium verticillioides* in maize grown in southern India. *World J. Microbiol. Biotechnol.*, **26(1)**, 71-78.

Covarelli, L., Gardiner D., Beccari G. and Nicholson P. (2013). Fusarium virulence assay on wheat and barley seedlings. *Bio-protocol*, **3(7)**, e446-e446.

Dean, R., Van Kan J.A., Pretorius Z.A., Hammond Kosack K.E., Di Pietro A., Spanu P.D. and Foster G.D. (2012). The Top 10 fungal pathogens in molecular plant pathology. *Molecular plant Pathology*, **13(4)**, 414-430.

Del Ponte, E.M., Moreira G.M., Ward T.J., O'Donnell K., Nicolli C.P., Machado F.J. and Lee T. (2022). *Fusarium graminearum* species complex: A bibliographic analysis and web-accessible database for global mapping of species and trichothecene toxin chemotypes. *Phytopathology®*, **112(4)**, 741-751.

Desjardins, A.E. (2006). *Fusarium mycotoxins: chemistry, genetics and biology* (pp. ix+-260).

Dill-Macky, R. and Jones R.K. (2000). The effect of previous crop residues and tillage on Fusarium head blight of wheat. *Plant Disease*, **84(1)**, 71-76.

Edwards, S.G., Imathiu S.M., Ray R.V., Back M. and Hare M.C. (2012). Molecular studies to identify the Fusarium species responsible for HT-2 and T-2 mycotoxins in UK oats. *Int. J. Food Microbiol.*, **156(2)**, 168-175.

Ferrigo, D., Raiola A. and Causin R. (2016). Fusarium toxins in cereals: Occurrence, legislation, factors promoting the appearance and their management. *Molecules*, **21(5)**, 627.

Foroud, N.A. and Eudes F. (2009). Trichothecenes in cereal grains. *Int. J. Mole. Sci.*, **10(1)**, 147-173.

Goswami, R.S. and Kistler H.C. (2004). Heading for disaster: *Fusarium graminearum* on cereal crops. *Mole. Plant Pathol.*, **5(6)**, 515-525.

Jang, J.Y., Baek S.G., Choi J.H., Kim S., Kim J., Kim D.W. and Lee T. (2019). Characterization of nivalenol-producing *Fusarium asiaticum* that causes cereal head blight in Korea. *Plant Pathol. J.*, **35(6)**, 543.

Khanal, R., Hudson K., Foster A., Wang X., Brauer E.K., Witte T.E. and Overy D.P. (2024). Pathogenicity and metabolomic characterization of *Fusarium graminearum* and Fusarium poae challenge in barley under controlled conditions. *J. Fungi*, **10(10)**, 670.

Kulik, T. (2011). Development of TaqMan assays for 3ADON, 15ADON and NIV Fusarium genotypes based on Tri12 gene. *Cereal Research Communications*, **39(2)**, 200-214.

Leslie, J.F. and Summerell B.A. (2008). *The Fusarium laboratory manual*. John Wiley & Sons.

Marasas, W.F. (2001). Discovery and occurrence of the fumonisins: a historical perspective. *Environ. Health Persp.*, **109(Suppl 2)**, 239.

McMullen, M., Bergstrom G., De Wolf E., Dill-Macky R., Hershman D., Shaner G. and Van Sanford D. (2012). A unified effort to fight an enemy of wheat and barley: Fusarium head blight. *Plant Disease*, **96(12)**, 1712-1728.

McMullen, M., Jones R. and Gallenberg D. (1997). Scab of wheat and barley: a re-emerging disease of devastating impact. *Plant Disease*, **81(12)**, 1340-1348.

Murodova, S.M., Bozorov T.A., Aytenov I.S., Ochilov B.O., Qulmamatova D.E., Salakhutdinov I.B. and Meliev S.K. (2024). Uncovering *Fusarium* species Associated with Fusarium Wilt in Chickpeas (*Cicer arietinum* L.) and the Identification of Significant Marker–Trait Associations for Resistance in the International Center for Agricultural Research in the Dry Areas' Chickpea Collection Using SSR Markers. *Agronomy*, **14(9)**, 1943.

Nagaraja, H., Chennappa G., Poorna Chandra Rao K., Mahadev Prasad G. and Sreenivasa M.Y. (2016). Diversity of toxic and phytopathogenic Fusarium species occurring on cereals grown in Karnataka state, India. *3 Biotech*, **6(1)**, 57.

Navale, V.D., Sawant A.M. and Vamkudoth K.R. (2023). Genetic diversity of toxigenic *Fusarium verticillioides* associated with maize grains, India. *Gen. Mole. Biol.*, **46(1)**, e20220073.

Nganje, W.E., Bangsund D.A., Leistritz F.L., Wilson W.W. and Tiapo N.M. (2002). National Fusarium Head Blight Forum Proceedings. *Michigan State University*, 275-281.

Njeru, N.K., Muthomi J.W., Mutegi C.K. and Wagacha J.M. (2015). Effect of cropping systems on accumulation of fusarium head blight of wheat inoculum in crop residues and soils.

Nor, N.M.M., Salleh B. and Leslie J.F. (2019). Fusarium species from sorghum in Thailand. *The Plant Pathol. J.*, **35(4)**, 301.

O'Donnell, K., Ward T.J., Geiser D.M., Kistler H.C. and Aoki T. (2004). Genealogical concordance between the mating type locus and seven other nuclear genes supports formal recognition of nine phylogenetically distinct species within the *Fusarium graminearum* clade. *Fungal Genetics and Biology*, **41(6)**, 600-623.

Oghenekaro, A.O., Oviedo-Ludena M.A., Serajazari M., Wang X., Henriquez M.A., Wenner N.G. and Fernando W.D. (2021). Population genetic structure and chemotype diversity of *Fusarium graminearum* populations from wheat in Canada and North Eastern United States. *Toxins*, **13(3)**, 180.

Oliveira, L.D.J.M.G.D., Rodrigues A.A.C., e Silva E.K.C., Oliveira A.C.S.D., Barros M.C., Fraga E.D.C. and Silva M.R.M. (2021). Morphological and phylogenetic characterization of 'Fusarium'Link. *Austr. J. Crop Sci.*, **15(12)**, 1406-1415.

Palumbo, R., Crisci A., Venâncio A., Cortiñas Abrahantes J., Dorne J.L., Battilani P. and Toscano P. (2020). Occurrence and co-occurrence of mycotoxins in cereal-based feed and food. *Microorganisms*, **8(1)**, 74.

Parry, D.W., Jenkinson P. and McLeod L. (1995). Fusarium ear blight (scab) in small grain cereals—a review. *Plant*

Pathology, **44(2)**, 207-238.

Pasquali, M. and Migheli Q. (2014). Genetic approaches to chemotype determination in type B-trichothecene producing Fusaria. *Int. J. Food Microbiol.*, **189**, 164-182.

Pestka, J.J. (2010). Deoxynivalenol: mechanisms of action, human exposure and toxicological relevance. *Arch. Toxicol.*, **84(9)**, 663-679.

Qu, B., Li H.P., Zhang J.B., Huang T., Carter J., Liao Y.C. and Nicholson P. (2008). Comparison of genetic diversity and pathogenicity of Fusarium head blight pathogens from China and Europe by SSCP and seedling assays on wheat. *Plant Pathology*, **57(4)**, 642-651.

Šarèević, H., Bukan M., Lovrić A. and Marièević M. (2023). Evaluation of inoculation methods for determination of winter wheat resistance to fusarium head blight. *Agronomy*, **13(4)**, 1175.

Scott, J.B. and Chakraborty S. (2006). Multilocus sequence analysis of *Fusarium pseudograminearum* reveals a single phylogenetic species. *Mycological Research*, **110(12)**, 1413-1425.

Shaikh, A. (2021). *Fusarium verticillioides* infection and fumonisin production during maize kernel maturation (*Doctoral dissertation*, Stellenbosch: Stellenbosch University).

Somma, S., Petruzzella A.L., Logrieco A.F., Meca G., Cacciola O.S. and Moretti A. (2014). Phylogenetic analyses of Fusarium graminearum strains from cereals in Italy, and characterisation of their molecular and chemical chemotypes. *Crop and Pasture Science*, **65(1)**, 52-60.

Stêpieñ, Ł., Gromadzka K. and Cheikowski J. (2012). Polymorphism of mycotoxin biosynthetic genes among *Fusarium equiseti* isolates from Italy and Poland. *J. Appl. Gen.*, **53(2)**, 227-236.

Sutton, J.C. (1982). Epidemiology of wheat head blight and maize ear rot caused by *Fusarium graminearum*. *Canadian J. Plant Pathol.*, **4(2)**, 195-209.

van der Lee, T., Zhang H., van Diepeningen A. and Waalwijk C. (2015). Biogeography of *Fusarium graminearum* species complex and chemotypes: A review. *Food Additives & Contaminants: Part A*, **32(4)**, 453-460.

Vanheule, A., De Boevre M., Moretti A., Scauflaire J., Munaut F., De Saeger S. and Audenaert K. (2017). Genetic divergence and chemotype diversity in the Fusarium head blight pathogen *Fusarium poae*. *Toxins*, **9(9)**, 255.

Velásquez-Zapata, V., Palacio-Rúa K., Cano L.E. and Gaviria-Rivera A. (2022). Assessment of genotyping markers in the molecular characterization of a population of clinical isolates of Fusarium in Colombia. *Biomédica*, **42(1)**, 18-30.

Wang, J.H., Ndoye M., Zhang J.B., Li H.P. and Liao Y.C. (2011). Population structure and genetic diversity of the *Fusarium graminearum* species complex. *Toxins*, **3(8)**, 1020-1037.

Waœkiewicz, A. and Stêpieñ Ł. (2012). Mycotoxins biosynthesized by plant-derived Fusarium isolates. *Arh Hig Rada Toksikol*, **63(4)**, 437-446.

Wegulo, S.N., Baenziger P.S., Nopsa J.H., Bockus W.W. and Hallen-Adams H. (2015). Management of Fusarium head blight of wheat and barley. *Crop Protection*, **73**, 100-107.

Wu, L., He X., He Y., Jiang P., Xu K., Zhang X. and Singh P.K. (2022). Genetic sources and loci for Fusarium head blight resistance in bread wheat. *Frontiers in Genetics*, **13**, 988264.

Wulff, E.G., Sørensen J.L., Lübeck M., Nielsen K.F., Thrane U. and Torp J. (2010). *Fusarium* spp. associated with rice Bakanae: ecology, genetic diversity, pathogenicity and toxigenicity. *Environ. Microbiol.*, **12(3)**, 649-657.

Xi, K., Shan L., Yang Y., Zhang G., Zhang J. and Guo W. (2021). Species diversity and chemotypes of *Fusarium* species associated with maize stalk rot in Yunnan province of southwest China. *Front. Microbiol.*, **12**, 652062.

Xu, X. (2003). Effects of environmental conditions on the development of Fusarium ear blight. *Europ. J. Plant Pathol.*, **109(7)**, 683-689.

Xu, X.M., Nicholson P., Thomsett M.A., Simpson D., Cooke B.M., Doohan F.M. and Edwards S.G. (2008). Relationship between the fungal complex causing Fusarium head blight of wheat and environmental conditions. *Phytopathology*, **98(1)**, 69-78.