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Host Metabolites in *Asparagus–Fusarium* Interaction: Mechanisms and Regulation

Elsie Ayamoh Enow | Monika Urbaniak | Łukasz Stępień

Department of Plant–Pathogen Interaction, Institute of Plant Genetics, Polish Academy of Sciences, Poznań, Poland

Correspondence: Łukasz Stępień (lste@igr.poznan.pl)

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ABSTRACT

Asparagus (*Asparagus officinalis*) is an important perennial vegetable crop cultivated worldwide. Its productivity and field longevity are often threatened by soilborne pathogens such as *Fusarium* spp., which cause wilt and rot diseases. These pathogens severely reduce yield and negatively impact the economic viability of asparagus cultivation. However, the interaction between asparagus and *Fusarium* involves a complex interplay of processes, including pathogen recognition, activation of defence signalling pathways and disease susceptibility. These processes are regulated by specific metabolites that may serve as defence signals or even facilitate the infection. Despite the progress in understanding the mechanisms in plants like *Arabidopsis thaliana* and cereal crops, knowledge about the asparagus–*Fusarium* pathosystem remains limited. Specifically, the mechanism by which host metabolites regulate the asparagus–*Fusarium* interaction still needs to be fully exploited. This article explores the defensive responses of asparagus plants towards infection by *Fusarium* spp. and delves into the regulatory mechanisms of host metabolites in the asparagus–*Fusarium* interaction. Understanding the dynamics of these interactions is essential for devising strategies to manage *Fusarium*-related diseases effectively and to develop disease-resistant asparagus varieties.

1 | Introduction

The genus *Asparagus* includes multiple species that are rich in nutrients and bioactive compounds, particularly antioxidants. *Asparagus officinalis* is highly susceptible to a complex of soilborne *Fusarium* fungi. Globally, members of this genus pose a significant threat to asparagus cultivation, causing rot, wilt and seedling blight diseases (Andrzejak and Janowska 2021; Brizuela et al. 2020). *Fusarium oxysporum* f. sp. *asparagi* (Foa) and *Fusarium proliferatum* predominate in causing asparagus wilt and rot diseases (Borrego-Benjumea et al. 2015; Nahiyan et al. 2011; Stępień et al. 2016; Waśkiewicz et al. 2013b). These pathogens are ubiquitous in asparagus fields, infesting crowns, roots and spears. While *F. oxysporum* typically affects young roots, *F. proliferatum* is found in nearly all parts of the plant (Andrzejak and Janowska 2021). In infected fields, they reduce yield, plant vigour and damage the whole plant, leading to the asparagus decline syndrome (Elmer 2015). In both in vivo and in vitro infection studies, the interaction between *Asparagus* and *Fusarium*, particularly with *F. oxysporum*, has been extensively examined (Djalali Farahani-Kofoet et al. 2020; He et al. 2002; Jacobi et al. 2023). However, developing effective, long-term solutions for managing *Fusarium* diseases remains challenging due to the pathogen's genetic diversity, the lack of genetic uniformity among asparagus cultivars and the persistent nature of the pathogen in soil and plant debris (Brizuela et al. 2020; Mercati et al. 2015).

Suggested cultural, chemical and biological control methods against *Fusarium* have produced mostly contradictory or inconclusive results (Ahmad and Matsubara 2020; Borrego-Benjumea et al. 2014; Brizuela et al. 2020; de la Lastra et al. 2021a;

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Elmer 2015; Haque and Matsubara 2018; Matsubara et al. 2014; Okada and Matsubara 2012; Surono and Narisawa 2018), especially in field trials (Jacobi et al. 2023). Most currently available asparagus cultivars have low resistance to *Fusarium* (Djalali Farahani-Kofoet et al. 2020), highlighting the urgent need to breed new resistant cultivars. While some wild relatives, such as *A. densiflorus* and *A. aethiopicus*, exhibit resistance to *F. oxysporum* infection (He et al. 2002; Jacobi et al. 2023; Kathe et al. 2019), strong crossing barriers hinder the transfer of this resistance into commercial asparagus cultivars (Plath et al. 2018).

The interaction between asparagus and *Fusarium* involves a multifaceted network of complexes that are only partially understood. During the infection, *Fusarium* spp. secrete a large array of lytic enzymes, effector proteins and/or toxic metabolites (mycotoxins) to degrade host immune defence and enhance pathogenesis (Gálvez and Palmero 2022; Ma et al. 2013; Perincherry et al. 2019). These *Fusarium* pathogen-associated molecular patterns (PAMPs) are usually recognised by receptors on the host membrane. This recognition initiates a cascade of reactions in the host, leading to cell wall fortification, reactive oxygen species (ROS) production, synthesis of antimicrobial metabolites, phytohormones and pathogenesis-related (PR) protein expression. Later responses include programmed cell death (PCD) through the hypersensitive response (HR) to limit pathogen spread (Chrpová et al. 2021; He et al. 2001; Jacobi et al. 2023). Plant secondary metabolites (SMs) with antifungal properties, produced in response to infection, include phenolic compounds, phenylpropanoids and polyamines (Elhamouly et al. 2022; Lalak-Kańczugowska et al. 2023; Perincherry et al. 2021a). These metabolites, synthesised via the phenylpropanoid pathway, have been shown to directly inhibit fungal growth, host colonisation, disrupt fungal cell structures, induce the expression of defence-related genes and enhance systemic acquired resistance (SAR) (Elhamouly et al. 2022; Perincherry et al. 2021a). Previous research showed that asparagus spear extracts inhibited the growth and mycotoxin production by *F. proliferatum* in vitro (Witaszak et al. 2020).

Fusarium spp. can counteract these host defences by producing toxic molecules to detoxify or neutralise host metabolic reactions, fostering successful colonisation. Moreover, environmental factors, such as pH, may affect the pathogenicity of *Fusarium* and the expression of virulence genes, such as HSP70 and SOD proteins (Lalak-Kańczugowska et al. 2023; Li et al. 2017). Notably, the integration of 'omics' data—genomics, transcriptomics, proteomics and metabolomics—has advanced the understanding of the intricate and dynamic molecular interplay underlying *Fusarium* pathogenicity and plant immunity (Deng et al. 2024). Proteomic approaches have provided insights into the secretome changes and infection mechanisms of various fungal pathogens (Lakshman et al. 2016; Li et al. 2012, 2019; Meijer et al. 2014), while metabolome and transcriptome analyses have revealed the accumulation of flavonoids and saponin defence-related metabolites in resistant wild *A. kiusianus* (Abdelrahman et al. 2017). Despite this significant progress, proper understanding of the regulatory networks underlying asparagus's metabolic responses to *Fusarium* infection remains largely elusive. Deeper insights into these pathways are essential for developing new strategies to enhance disease resistance in asparagus and manage *Fusarium*-related diseases effectively.

This review highlights the defence responses of asparagus and host metabolites that may play a role in protecting asparagus plants against *Fusarium* infection. Identifying key metabolites and understanding their regulatory mechanisms can guide the breeding of more resilient asparagus cultivars and inform future efforts to mitigate the impact of *Fusarium*-related diseases on asparagus cultivation.

2 | *Fusarium* Pathogens of Asparagus

Fusarium species infecting asparagus plants are diverse, and within each species, different strains might exhibit varying degrees of virulence, aggressiveness and host specificity across crops. Pathogenic species associated with asparagus include *F. acuminatum*, *F. avenaceum*, *F. culmorum*, *F. compactum*, *F. equiseti*, *F. ershadii*, *F. fujikuroi*, *F. heterosporum*, *F. oxysporum* f. sp. *asparagi* (Foa), *F. proliferatum*, *F. redolens*, *F. solani*, *F. sporotrichioides* and *F. tricinctum* (Andrzejak and Janowska 2021; Borrego-Benjumea et al. 2014; Brizuela et al. 2020; de la Lastra et al. 2021b; Elena 2007; Papizadeh et al. 2018). These pathogens cause diseases such as crown and root rot, stem rot, wilt and seedling blight, which result in reduced plant lifespan, lower quality and diminished yields (Andrzejak and Janowska 2021; Brizuela et al. 2020; de la Lastra et al. 2021b). According to Noperi-Mosqueda et al. (2020), *Fusarium* infections can significantly shorten the longevity of asparagus plants within 5–6 years, with fields typically showing reduced plant vigour, poor spear quality, lower yields and premature death (Noperi-Mosqueda et al. 2020). The presence of *F. proliferatum* in post-harvest asparagus severely affects spear quality, disrupting sugar and respiratory metabolism (Zhang et al. 2022). Among the two recognised predominant species, Foa is highly specific to asparagus, whereas *F. proliferatum* is more opportunistic and can infect various crops. Other species, such as *F. culmorum* (Elena 2007) and *F. solani*, are less common or aggressive (Nahiyan et al. 2011; Brizuela et al. 2020). While *F. oxysporum* is a strong soil inhabitant, *F. proliferatum* can also be airborne (Elena 2007).

2.1 | *Fusarium* Infection of Asparagus and Pathogenesis

The infection process is complex and unfolds in several stages, each marked by specific pathogen activities and plant responses. This process begins with the germination of *Fusarium* spores, or conidia, in the soil. These spores are highly adaptable and can remain dormant for extended periods until environmental conditions become favourable. Upon contact with asparagus roots, the long-lived chlamydospores (surviving up to 15 years) begin to germinate under warm, humid conditions (25°C–30°C), forming hyphae (Brizuela et al. 2021). Fungal hyphae adhere to the root surface by utilising compounds found in the plant cutin layers, such as dihydroxy-C16 and trihydroxy-C18, which guide the fungus towards its specific host and induce cutinase activity (Husaini et al. 2018). With the aid of excreted enzymes, the hyphae penetrate the root through natural openings or wounds (Omar et al. 1999). This enzymatic breakdown of the plant cell wall weakens the plant's defence and facilitates entry into the root epidermis and outer layers.

The fungus spreads into the cortex and invades the xylem vessels (Jangir et al. 2021). Once inside the xylem, the pathogen blocks movement of water and nutrients by producing toxins and polysaccharides in the vascular system. Colonisation of the vascular tissue triggers both local and systemic plant defence responses (Berrocal-Lobo and Molina 2008). Eventually, infected plants show symptoms such as yellowing of shoots, wilting, stunted growth, brown discolouration of vascular tissue and crown rot. In severe cases, the pathogen kills the plant by the end of its life cycle (Brizuela et al. 2020), and infected spears develop brown discolouration and rot (Elena 2007). According to Djalali Farahani-Kofoet et al. (2023), brown lesions appeared on asparagus plants following *F. oxysporum* infection by the fifth day in vitro and the seventh day in sand culture. Similarly, Omar et al. (1999) reported necrotic symptoms on asparagus tissue culture plantlets 4 days after inoculation with *F. proliferatum*. Similarly, Omar et al. (1999) observed necrotic signs on asparagus tissue culture plantlets 4 days after inoculation with *F. proliferatum*. The infection process alters the morphology of asparagus roots, reducing their ability to store carbohydrates (Brizuela et al. 2020; Corpas-Hervias et al. 2006; Djalali Farahani-Kofoet et al. 2020), which further contributes to the decline in plant health and productivity. As the plant deteriorates, *F. oxysporum* ensures its persistence through the production of chlamydospores and propagates via macroconidia and microconidia. Notably, *F. proliferatum* does not produce chlamydospores but can still spread via conidia (Brizuela et al. 2020). While the infection process generally follows a similar pattern (spore attachment, germination, root penetration, tissue colonisation and symptom development), variability can arise at any stage of the process depending on the *Fusarium* species involved (Ekwomadu and Mwanza 2023). The pathogenicity of *Fusarium* spp. on asparagus is shaped by genetic variability among species, which impacts their virulence factors such as mycotoxin production (e.g., fumonisins, fusarin C by *F. proliferatum*), enzymatic activity, host specificity and adaptation to environmental conditions. The highly specialised Foa invades the xylem, causing vascular wilt and occasionally root and crown rot. In contrast, *F. proliferatum* typically induces localised infections in the crown, stems and, rarely, on root tissues across a broader host range (Brizuela et al. 2020; Elena 2007; Sonoda et al. 2002). Other species, like *F. solani*, may lead to root rot with occasional vascular wilting (Brizuela et al. 2020; Elena 2007). *F. redolens* f. sp. *asparagi* causes symptoms similar to those of Foa and *F. proliferatum* (Baayen et al. 2000). Moreover, *F. redolens* shares common morphological features, such as spore types and pathogenicity with Foa (Brizuela et al. 2020). Younger plants and susceptible cultivars are more prone to severe infection, especially from *F. proliferatum*, than mature or resistant varieties (Elena 2007).

2.2 | *Fusarium* Virulence Factors

Hemibiotrophic fungi, like *Fusarium* species, possess a variety of pathogenicity factors that enable them to infect asparagus. They secrete a diverse range of carbohydrate-active CAZymes (Zhao et al. 2013), enabling them to break plant cell wall polymers and facilitating their entry into plant tissues (Ekwomadu and Mwanza 2023). *Fusarium* cell wall-degrading enzymes (CWDEs) include cellulases, hemicellulases, pectinases, lipases and xylanases, which target cellulose, hemicellulose, pectin, lipids and xylose, respectively (Djalali Farahani-Kofoet et al. 2020; Kubicek et al. 2014; Perincherry et al. 2021b). These enzymes not only enhance the infection process but also allow the pathogen to exploit the carbon sources from the plant (Djalali Farahani-Kofoet et al. 2020; Ruiz et al. 2016).

In addition to these enzymes, *Fusarium*-secreted mycotoxins play a critical role in disease progression. These mycotoxins disrupt signalling pathways in *A. officinalis*, suppressing the production of defence-related compounds and promoting fungal growth and development (Stępień et al. 2016). For example, *F. oxysporum* secretes mycotoxins that can alter the permeability of plant cell membranes, reduce ROS levels, disrupt ATP synthesis and inhibit plant growth (Shao et al. 2020). Mycotoxins identified in infected asparagus include fumonisins, fusaric acid (FA), fusarins, moniliformin (MON), beauvericin (BEA) and enniatins (ENN), which are produced by *F. proliferatum*, *F. oxysporum* and other *Fusarium* species (Stępień et al. 2016; Waśkiewicz and Stępień 2012; Waśkiewicz et al. 2013a; Weber et al. 2006). The study by Stępień et al. (2016) further revealed significant accumulation of fumonisins B_1–B_3 and BEA in *Fusarium*-infected asparagus spears. Their findings established *F. sporotrichioides* and *F. equiseti* as effective producers of HT-2 toxin and zearalenone mycotoxin, respectively, in asparagus spears (Stępień et al. 2016). Further evidence suggests a possible correlation between toxigenicity and the pathogen's aggressiveness (Kosová et al. 2009), which may explain why fumonisin-producing *F. proliferatum* is more aggressive on asparagus than *F. oxysporum* and *F. solani* (Brizuela et al. 2020; Djalali Farahani-Kofoet et al. 2020).

Fumonisins represent a group of polyketide mycotoxins primarily synthesised by *F. proliferatum*. The B series analogues of fumonisins, including FB_1, FB_2 and FB_3, are among the most hazardous, with FB_1 exhibiting the highest level of toxicity (Awuchi et al. 2021). FB_1 triggers cell death through excessive ROS and nitric oxide production (NO), accumulation of phytoalexins and overexpression of PR genes (Berkey et al. 2012; Iqbal et al. 2021). The excessive ROS/NO production facilitates the pathogen's evasion of plant immunity, leading to oxidative stress within the plant cell and causing damage to cellular components. This, in turn, renders the host more susceptible to infection and further disease progression. Moreover, FB_1 functions as a specific effector that can activate SAR and accumulation of key plant hormones such as salicylic acid (Iqbal et al. 2021; Waśkiewicz et al. 2013b).

FA can reprogramme the host's metabolic pathways, leading to premature senescence and intensifying infection severity (Dong et al. 2014). It promotes pathogenesis in the later stages of infection (Selim and El-Gammel 2015) and induces metal and oxidative stresses within plant cells. This increases ROS production, which can trigger cell death and cell structural disruption. FA also diminishes the activity of antioxidant enzymes, reduces chlorophyll content and inhibits photosynthetic efficiency in plants (López-Díaz et al. 2018; Selim and El-Gammal 2015; Singh and Upadhyay 2014; Singh et al. 2017). As reported by Singh et al. (2017), FA elevates the levels of proteolytic enzymes in tomato plants, contributing to tissue damage and disease progression (Singh et al. 2017). FA accumulation in plant tissues

disrupts water and nutrient uptake, leading to wilting and stunted growth (Iqbal et al. 2023). In saffron, moderate concentrations of FA (50–100 μM) induce apoptosis, while higher concentrations (>200 μM) lead to necrotic symptoms (Luo et al. 2023; Samadi and Shahsavan-Behboodi 2006). Presently, not much work has been conducted to identify the potential threat of fusarin beyond maize.

MON is a mycotoxin that exists as sodium or potassium salts of cyclobutane 1-hydroxycyclobut-3,4-dione, often found in grain products (Waśkiewicz et al. 2013a). In garlic, MON produced by *F. oxysporum* inhibits plant growth and photosynthesis and has been reported to play a potential role in the pathogenesis of *F. proliferatum* (Gasser et al. 2023). Both *F. oxysporum* and *F. proliferatum* are also known to produce fusarins and MON in infected asparagus spears (Stępień et al. 2016; Weber et al. 2006).

BEA and ENNs—cyclic oligopeptides known for their ionophoric properties (Urbaniak et al. 2020)—disrupt membrane permeability and exhibit significant cytotoxic effects, including the induction of chromosomal aberrations, DNA fragmentation, apoptosis and cell death (Mallebrera et al. 2018). These cyclic peptide toxins (BEA and ENNs produced by *F. oxysporum*) were reported, in addition to MON and FA, at relatively low concentrations in infected asparagus tissues (Stępień et al. 2016).

Effector proteins play a crucial role in the virulence strategies of fungal pathogens (Ekwomadu and Mwanza 2023). *Fusarium* species secrete small, cysteine-rich proteins during host colonisation to manipulate host physiology, suppress immune responses and promote infection. Effector proteins such as the Secreted in Xylem (SIX) proteins are encoded on pathogenicity chromosomes, which are often highly dynamic and horizontally transferable (Jangir et al. 2021). Transcription factors (TFs) such as SGE1 (*SIX* gene expression 1), FTF1 (*Fusarium* TF 1) and FTF2 (*Fusarium* TF 2) regulate the expression of these effectors, demonstrating the sophisticated genetic control underlying pathogenicity infection (Jangir et al. 2021; Thatcher et al. 2012). SIX proteins have been implicated in the virulence of several species such as *F. oxysporum*, *F. proliferatum*, *F. graminearum*, *F. verticillioides* and *F. virguliforme* (Gao et al. 2018; Maldonado et al. 2018), interacting with hosts like tomato (Maldonado et al. 2018) and wheat (Lu and Edwards 2016). However, their role in asparagus infection remains understudied. Also, the precise regulatory mechanisms linking *FTF1* and *FTF2* with *SGE1* and *SIX* genes need to be elucidated. Additionally, *Fusarium* species rely on activating general pathogenicity factors to achieve full virulence (Ma et al. 2013). The complex network, including cyclic adenosine monophosphate (cAMP), mitogen-activated protein kinase (MAPK), Ras (retrovirus-associated DNA sequences) proteins, the velvet complex and guanine nucleotide-binding (G) proteins, regulates gene expression related to *Fusarium* development and virulence (Ma et al. 2013; Stępień and Lalak-Kańczugowska 2021). These signalling pathways enable *Fusarium* to adapt to host environments and enhance pathogenicity. Notably, the pathogenic potential of *Fusarium* can be influenced by the interplay between species-specific traits, environmental conditions (like soil pH, temperature and moisture) and the physiological state of the asparagus plants. Generally, different asparagus varieties may exhibit varying resistance levels, and even closely related *Fusarium* species can display significant metabolic differences that can affect their infection strategies and disease severity (Niehaus et al. 2016). These differences can lead to variations in disease progression, with some strains being more aggressive and others showing lower virulence.

3 | Asparagus Defence Responses to *Fusarium* Infection

To mount an effective defence against *Fusarium* infection, asparagus, like other plants, must recognise the pathogen during the early stages of invasion and activate its innate immune responses (Figure 1). This detection is mediated through membrane-bound receptors called pattern recognition receptors (PRRs), which probably recognise PAMPs such as chitin, glucan fragments and pathogen-derived effectors. The recognition of PAMPs by receptors such as receptor-like kinases (RLKs) and receptor-like proteins (RLPs) activates the plant's basal immune defence, leading to downstream innate immune responses such as pattern-triggered immunity, PTI (Jones and Dangl 2006). Additionally, when specific pathogen-derived effectors are detected by nucleotide-binding site-leucine-rich repeat (NBS-LRR) proteins, a second layer of immunity known as effector-triggered immunity (ETI) is activated (Jones and Dangl 2006). These defence layers initiate downstream signalling cascades involving MAPKs and calcium-dependent protein kinases (CDPKs), which serve to amplify the defence signal within the plant cell and lead to transcriptional reprogramming. Key TFs such as WRKY and MYB (myeloblastosis) are then activated, stimulating the production of defence-related compounds. These compounds include ROS, SMs, phytohormones, hydrolytic enzymes and cell wall-strengthening molecules. This complex network of signalling ultimately leads to the HR, a localised cell death mechanism that prevents the spread of the pathogen and contributes to disease resistance (He et al. 2001; Jacobi et al. 2023).

A key aspect of the asparagus active defence against *Fusarium* involves the production of ROS and reactive nitrogen species (RNS) at the infection site. These molecules serve both as signalling molecules and cell-damaging agents that limit pathogen advancement in host tissues. The respiratory burst oxidase homologue (RBOH) gene family encodes NADPH oxidases, which generate ROS molecules, such as hydrogen peroxide (H_2O_2) and superoxide anions (O_2^-). In resistant species, ROS production is swift and localised to infection sites, where it can directly damage *Fusarium* cells and signal downstream immune responses. Studies on asparagus wild species, such as *A. densiflorus* and *A. aethiopicus*, demonstrated elevated ROS levels after *Fusarium* infection (He et al. 2001; Jacobi et al. 2023). These species also exhibited rapid accumulation of ROS-scavenging enzymes, such as ascorbate peroxidase (APX) (Djalali Farahani-Kofoet et al. 2023) and catalase (CAT), which help to maintain ROS homeostasis and enhance resistance. The oxidative burst is also counterbalanced by other antioxidant enzymes such as superoxide dismutase (SOD), whose genes are upregulated to manage O_2^- levels and mitigate potential damage to host cells (Liu and Matsubara 2016). However, only a limited number of studies have been carried out to explore the defensive mechanism of asparagus towards *Fusarium* infection (Table 1).

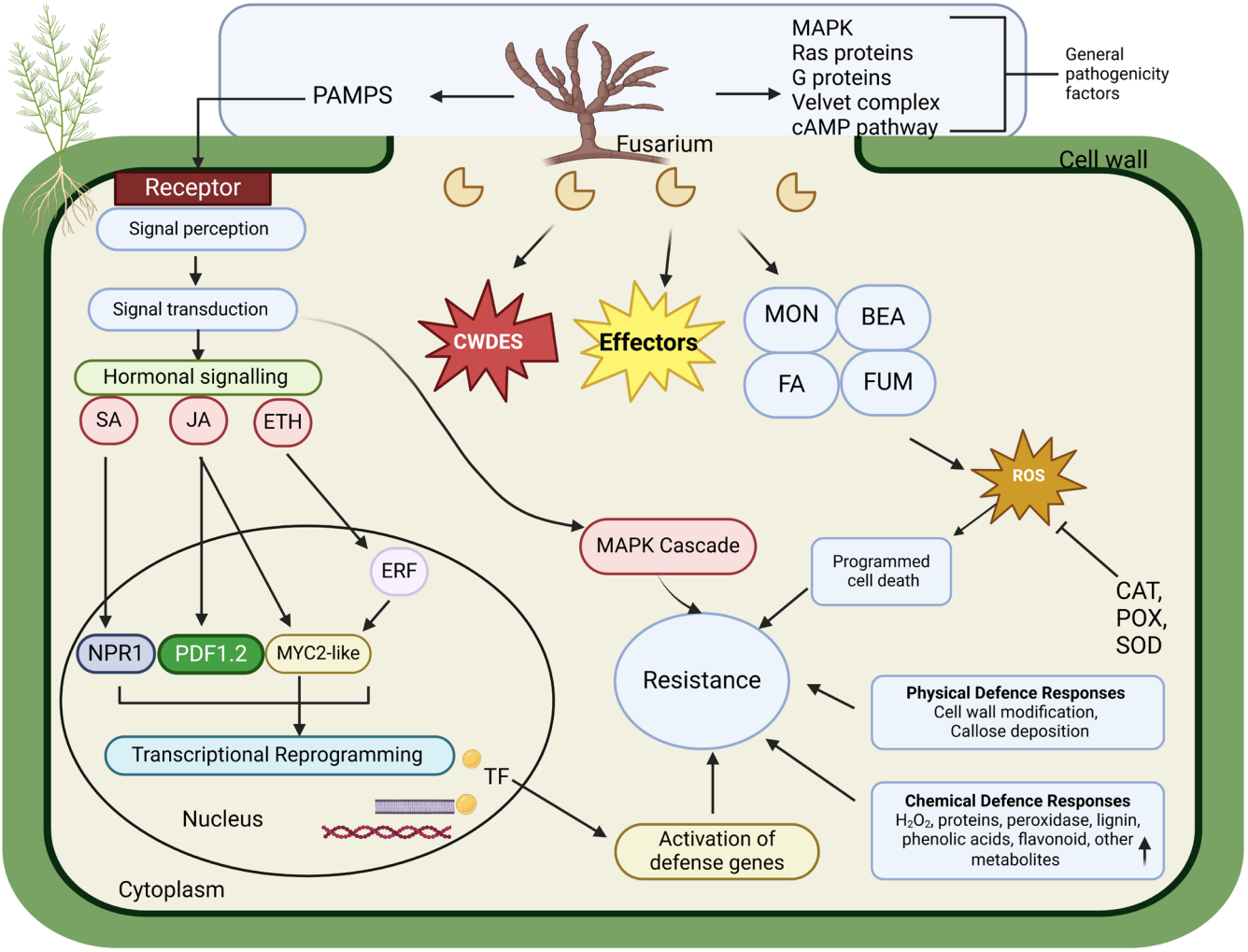


FIGURE 1 | An overview of the physiological, biochemical and molecular defence responses occurring in plants towards *Fusarium* infection. BEA, beauvericin; CWDEs, cell wall-degrading enzymes; ERF, ethylene response factor; ETH, ethylene; FA, fusaric acid; FUM, fumonisins; MAPK, mitogen-activated protein kinase; MON, moniliformin; MYC2, myelocytomatosis 2; NPR1, Non-expressor of Pathogenesis-Related Gene 1; PDF1.2, Plant Defensin 1.2.

Asparagus plants synthesise PR proteins, which are defence-related enzymes such as peroxidase (POX), polyphenol oxidase (PPO), phenylalanine ammonia-lyase (PAL), chitinases and β-1,3-glucanases that are produced in response to pathogenic invasion (Abdelrahman et al. 2017). POX and PPO contribute to defence by producing toxic intermediates that impede fungal growth (Xu et al. 2021). PAL is involved in the phenylpropanoid pathway, catalysing the production of lignin and other phenolic compounds that reinforce cell walls (He and Wolyn 2005). The PR gene family is highly induced following *Fusarium* infection, playing roles in cell wall degradation, signal transduction and the synthesis of antimicrobial compounds (Xu et al. 2021). PR1 is often associated with, and is frequently used as a marker for, SAR, a long-term immune memory that primes the plant against future infections. Djalali Farahani-Kofoet et al. (2020) observed higher expression of PR1 in *A. densiflorus* and *A. aethiopicus* following infection with *F. redolens*, *F. oxysporum* and *F. proliferatum*. Additionally, *PR1*, *POX* and *PAL* genes were highly expressed in asparagus cultivars such as Rapsody, Xenolim, Vitalim and Grolim, 5 days post-infection with Foa in vitro (Djalali Farahani-Kofoet et al. 2023). However, in some plants, like saffron, these enzymes help resist infection at the early stages, but as the infection progresses, the resistance becomes difficult to maintain, and this may also contribute to host susceptibility (Luo et al. 2023). Additionally, asparagus plants respond to *Fusarium* spp. by activating the phenylpropanoid pathway. This pathway leads to the synthesis of important defence metabolites like lignin, lignans, flavonoids, salicylic acid (SA) and other phenylpropane compounds (Boba et al. 2017; He et al. 2001; He and Wolyn 2005). Lignin strengthens the plant's middle lamella and secondary cell walls, forming a physical barrier that prevents pathogen penetration (Li et al. 2023b; Sattler and Funnell-Harris 2013) and resists cell wall degradation by pathogen-secreted CWDEs (He et al. 2001). Comparative studies demonstrated that resistant species such as *A. densiflorus* showed higher levels of lignification and PAL activity upon infection with Foa compared to the more susceptible *A. officinalis* (He et al. 2001). Hormonal responses also play a role in asparagus defence. SA not only promotes local resistance but also activates SAR and PR gene expression (He and Wolyn 2005). The exogenous application of SA has been shown to reduce *Fusarium* symptoms in asparagus, inducing SAR

TABLE 1 | Response of *Asparagus* species to various *Fusarium* spp.

Plant type	*Fusarium* species	Inoculum density	Effects on plants, defence response	References
A. officinalis	*F. oxysporum* f. sp. *asparagi*	10^2, 10^4, 10^6 mL^{-1}, 6–8 × 10^6 conidia mL^{-1}	Root browning, growth depression, susceptibility necrotic lesions	Djalali Farahani-Kofoet et al. (2020), He et al. (2001), Jacobi et al. (2023), Kathe et al. (2019)
A. densiflorus	*F. oxysporum* f. sp. *asparagi*	10^2, 10^4, 10^6 spores mL^{-1}	Hypersensitive cell death, peroxidase, phenylalanine ammonia-lyase	He et al. (2001)
A. officinalis 'Backlim' and 'Rapsody'	*F. oxysporum* f. sp. *asparagi*, *F. proliferatum*, *F. redolens* Fred1, Fred2	6–8 × 10^6 conidia mL^{-1}, in vitro, in vivo	Reduced total length of fibrous and storage roots, reduced total root length, volume, and surface area, significant induction of pathogenesis-related gene expression, fungal DNA in stem (strongest effect with Fred1)	Djalali Farahani-Kofoet et al. (2020)
A. densiflorus	*F. oxysporum* (Foxy I–Foxy III)	10^4, 10^5, 10^6 conidia mL^{-1} in vitro	No disease symptoms	Kathe et al. (2019)
A. ramosissimus, *A. amarus*, *A. prostratus*, *A. verticillatus*, *A. maritimus*, wild relatives	*F. oxysporum* (Foxy I–Foxy III)	10^4, 10^5, 10^6 conidia mL^{-1} in vitro	Less infestation for all cultivars besides *A. maritimus*	Kathe et al. (2019)
A. densiflorus, *A. aethiopicus*, *A. officinalis* 'Thielim'	*F. oxysporum* Foxy II and Foxy III	10^6 conidia mL^{-1} in vitro	No browning of roots or growth depression, rapid accumulation of H_2O_2 in wild species	Jacobi et al. (2023)
A. officinalis	Nonpathogenic *F. oxysporum*	10^2, 10^4, 10^6 spores mL^{-1}	Hypersensitive response, induced systemic resistance, activation of peroxidase, phenylalanine ammonia-lyase and lignin deposition	He et al. (2002)
A. officinalis	*F. oxysporum* f. sp. *asparagi*, *F. proliferatum*		Increased salicylic acid concentration in roots and stems, peroxidase, phenylalanine ammonia-lyase and lignin	Waśkiewicz et al. (2013b)

TABLE 2 | Defence-related genes in asparagus responsive to *Fusarium* infection.

Gene	Properties	Function	*Fusarium* infection	References
POX	Peroxidase 1-like	Lignin-forming	Induced	Djalali Farahani-Kofoet et al. (2020, 2023), He et al. (2001), Waśkiewicz et al. (2013b)
PAL	Phenylalanine ammonia-lyase	Antifungal	Induced	Djalali Farahani-Kofoet et al. (2020, 2023), He et al. (2001), Waśkiewicz et al. (2013b)
PR1	Unknown	Antifungal	Induced	Djalali Farahani-Kofoet et al. (2020)
PR9	Peroxidase	Lignin-forming	Unchanged or reduced	Djalali Farahani-Kofoet et al. (2020)
NPRI	Non-expressor of pathogen-response 1	Salicylic acid induction	Induced	He and Wolyn (2005)

and enhancing expression of PR genes, such as *PR1* (He and Wolyn 2005). Research also highlights that defence timing and intensity impact resistance levels. Jacobi et al. (2023) observed that *A. densiflorus* initiated defence mechanisms within 8–24 h post-infection, whereas *A. officinalis* responded more slowly, with visible lesions appearing only after 7–14 days. This delay in response may contribute to the susceptibility of *A. officinalis*. Furthermore, inoculating asparagus seedlings with nonpathogenic *F. oxysporum* strains has been found to boost resistance. Liu and Matsubara (2016) showed that such treatments increased SOD activity, radical scavenging, polyphenol levels and ascorbic acid, thereby reducing disease severity upon exposure to pathogenic Foa. In addition to biochemical defences, asparagus plants possess morphological traits such as thick cuticles, rigid cell walls and tightly regulated stomatal apertures, which could provide passive resistance against pathogen penetration, complementing molecular defences. Nonetheless, aggressive *Fusarium* species may still overcome these defences, especially in stressed plants.

The genetic framework underlying the asparagus defence response towards *Fusarium* pathogenicity factors and signal transduction relies on the coordinated activation of multiple genes and biochemical pathways (Figure 1). This defence primarily operates through a gene-for-gene mechanism, where plant resistance (R) genes recognise specific avirulence (avr) genes in the pathogen and activate downstream signalling cascades to trigger defence responses. The most common R genes possess nucleotide-binding site and leucine-rich repeat (NBS-LRR) domains (Tailor and Bhatla 2024). Besides asparagus, several R genes or NLRs with identified functions have been isolated from *Arabidopsis*, tomato and other host plants (Blake et al. 2003; Jangir et al. 2021). Advanced techniques like genome-wide association studies (GWAS) and transcriptomics can reveal candidate genes involved in fungal resistance (Abdelrahman et al. 2017, 2020). Some studies have identified genes in asparagus potentially linked to *Fusarium* defence (Table 2), but these genes still need further functional validation. Nonetheless, ongoing research should identify PRRs, specific *Fusarium*-derived PAMPs recognised by asparagus PRRs, isolate-specific NB-containing R genes and elucidate unique immune pathways activated in response to *Fusarium*. This will offer opportunities for breeding more disease-resistant asparagus varieties and improving plant immunity.

4 | Role of Host Metabolites in Plant–Pathogen Interactions

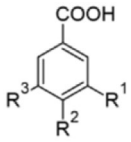
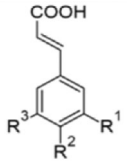
Primary metabolites such as carbohydrates, amino acids, organic acids and fatty acids play a crucial role in plant growth and development. During pathogenic infection, these metabolites not only serve as energy sources but also act as essential building blocks for various cellular processes and signals in defence mechanisms (Rojas et al. 2014). Previously, Kasote et al. (2020) reported increased free amino acid content (histidine, citrulline, valine and phenylalanine) in the watermelon plant defence response against *Fusarium* infection. Notably, in response to pathogens, plants produce SMs via primary metabolic pathways as a defensive strategy. These SMs can eliminate pathogens, inhibit mycotoxin production and limit the spread of fungi. In asparagus, the most bioactive SMs include phenolic acids, flavonoids, alkaloids, tannins, stilbenes, hydroxycinnamic acids and saponins (Fuentes-Alventosa et al. 2013; Guo et al. 2020; Hamdi et al. 2024; Jiménez-Sánchez et al. 2016; Lee et al. 2014; Li et al. 2023a). The abundance and composition of these metabolites can vary based on factors such as asparagus genotype, developmental stage and environmental conditions (Hamdi et al. 2020; Jaramillo-Carmona et al. 2017).

Phenolic compounds or polyphenols represent the largest group of antioxidant phytochemicals that exhibit a range of defence responses against pathogens. Their accumulation inhibits fungal growth and reproduction while reducing *Fusarium* mycotoxin accumulation in plant tissues (Nakabayashi and Saito 2015). Phenolics, such as suberin and lignin, can be bound to the host cell wall, reinforcing its structure and forming defensive barriers.

4.1 | Phenolic Acids

Phenolic acids derived from cinnamic acid or benzoic acid (Table 3) are known for their strong antioxidant properties. Common phenolic acids in asparagus extracts include ferulic, chlorogenic, caffeic, coumaric, sinapic and 4-hydroxybenzoic acids (Ayhan and Rosenberg 2021; Hamdi et al. 2024; Jiménez-Sánchez et al. 2016). Increased concentrations of these metabolites result in toxicity to pathogens, inhibition of fungal enzymes, detoxification of mycotoxins and prevention of further

TABLE 3 | Structures of some phenolic acids.

Phenolic acid	Side group		
	R^1	R^2	R^3
Hydroxybenzoic acid	 hydroxybenzoic		
Benzoic	H	H	H
p-Hydroxybenzoic	H	OH	H
Gallic	OH	OH	OH
Protocatechuic	OH	OH	H
Vanillic	OCH_3	OH	H
Hydroxycinnamic acid	 hydroxycinnamic		
Cinnamic	H	H	H
p-Coumaric	H	OH	H
Caffeic	OH	OH	H
Ferulic	OCH_3	OH	H
Sinapic	OCH_3	OH	OCH_3

Note: Adapted from Šamec et al. (2021).

infection (Wu et al. 2014). For example, ferulic acid significantly suppressed the expression of *FUM* genes necessary for fumonisin biosynthesis in *F. proliferatum*, while caffeic and chlorogenic acids inhibited its growth and suppressed FB_1 biosynthesis (Lalak-Kańczugowska et al. 2023). Another study showed that hydroxycinnamic acids can inhibit fungal growth and enniatin production in *F. avenaceum* (Gautier et al. 2020). While phenolic compounds generally exhibit antifungal properties, some can act as stimulants or inhibitors of *Fusarium* growth (Chrpová et al. 2021; Schöneberg et al. 2018). For instance, ferulic acid, 4-hydroxybenzoic acid and phthalic acid from watermelon roots encouraged spore germination and proliferation of *F. oxysporum* f. sp. *niveum* (Fon; Hao et al. 2010), whereas others, like *p*-coumaric acid, hindered Fon development (Hao et al. 2010). Interestingly, *p*-coumaric acid can also facilitate the production of harmful toxins such as T-2 and HT-2 by *F. langsethiae* and *F. sporotrichioides* (Ferruz et al. 2016). In contrast, cinnamic acid effectively suppressed Fon biomass, mycotoxin production and PR enzymes in Lanzhou lily plants (Wu et al. 2014). However, the combined action of caffeic acid and Foa facilitated asparagus infection, leading to decreased seedling growth and increased root rot incidence (Liu and Matsubara 2018). In different plant–pathogen systems, *p*-hydroxybenzoic acid and benzoic acid were implicated in promoting *F. oxysporum* wilt infection in strawberry (Qi et al. 2015) and faba bean (Hu et al. 2023). Similarly, cinnamic acid promoted the infectivity and pathogenicity of *F. oxysporum* f. sp. *fabae* in beans (Guo et al. 2020; Yang et al. 2022). This dual behaviour highlights the potential for these compounds to either promote resistance or susceptibility of various crops to *Fusarium* infections. Generally, the efficacy of phenolic acids against *Fusarium* spp. can vary depending on their concentration and specific composition. Liu and Matsubara (2018) observed that 0.1% of caffeic acid and ferulic acid effectively suppressed Foa as opposed to 0.01% of the same metabolites. Also, higher concentrations of ferulic acid significantly inhibited the growth of *F. graminearum*, *F. langsethiae* and *F. poae* (Schöneberg et al. 2018). Ferrochio et al. (2013) observed that higher concentrations of ferulic acid (≥20 mM) greatly reduced mycelial growth and fumonisin production. Lower ferulic acid (1–10 mM) content enhanced fumonisin production (Ferrochio et al. 2013) but reduced *F. proliferatum* growth by 30%, as opposed to the slight stimulation of mycelium growth by *p*-hydroxybenzoic acid and vanillic acid (Coma et al. 2011). Ferulic acid in 1 mM concentration significantly decreased *F. langsethiae* biomass but increased T-2 toxin production after 10 days of incubation (Ferruz et al. 2016). 1 mM of benzoic acid and *trans*-cinnamic acid inhibited the mycelial growth of *Fusarium oxysporum* f. sp. *cubense*, Tropical Race 4, a pathogen causing wilt of banana (Were et al. 2022). *p*-Coumaric acid (>0.1 mM) inhibited asparagus growth (Kato-Noguchi et al. 2018).

4.2 | Flavonoids and Anthocyanins

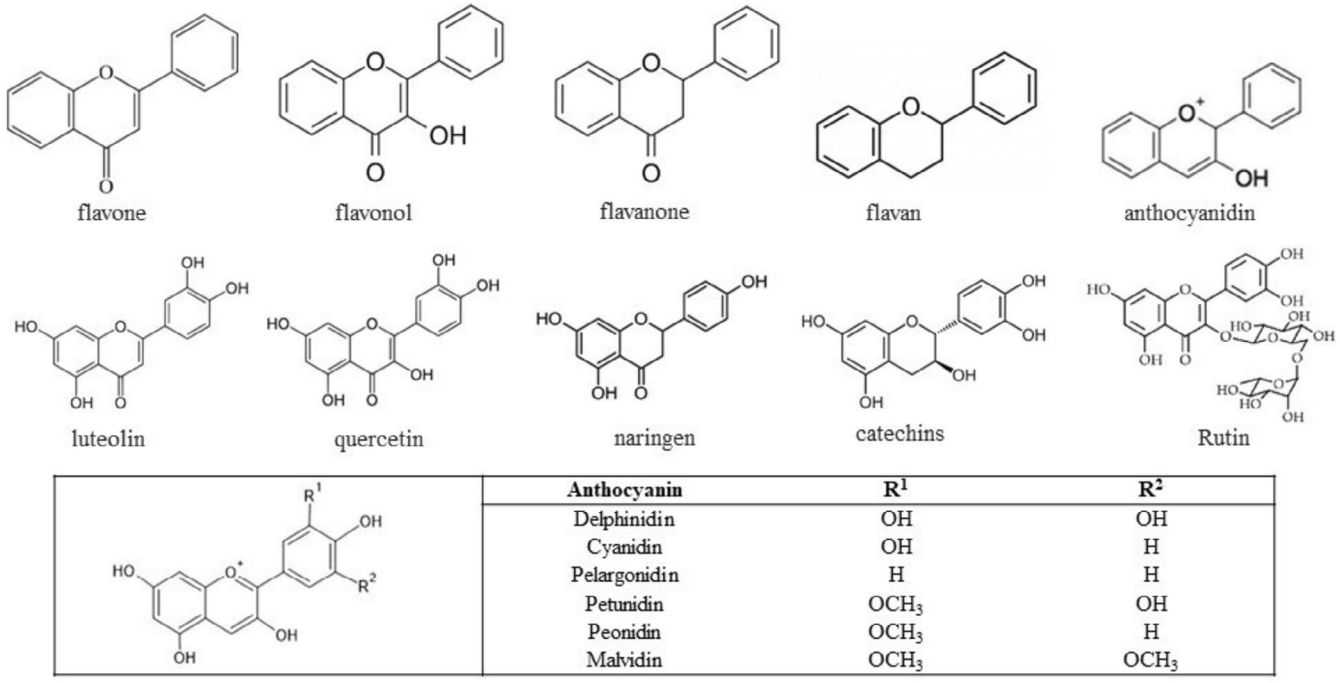
Flavonoids are the most abundant antioxidant phenolic compounds naturally produced in plants, with a C6-C3-C6 structure consisting of two benzene rings (A and B) connected by a three-carbon pyran ring (C) (Dias et al. 2021). The precursors of their biosynthesis are phenylalanine and malonyl coenzyme A. Flavonoids serve many functions including pigmentation and anti-inflammatory and antimicrobial activity (Jaramillo-Carmona et al. 2017; Jiménez-Sánchez et al. 2021; Mohamed et al. 2017). While white asparagus contains hydroxycinnamic acid derivatives, green asparagus spears and roots are rich in flavonoids (Guillén et al. 2008; Guo et al. 2022). Flavonoid classes in asparagus (Figure 2) include flavones (e.g., luteolin and apigenin), flavonols (e.g., quercetin, kaempferol and rutin), flavanones (e.g., naringenin and hesperidin), flavan (e.g., epicatechin gallate and catechins), anthocyanidins, isoflavones, coumarins, stilbenes, lignans and others (Ayhan and Rosenberg 2021; Jiménez-Sánchez et al. 2016; Pegiou et al. 2021). Among metabolites, rutin (quercetin-3-*O*-rutinoside) is the most abundant flavonoid in green asparagus spears (Chileh Chelh et al. 2023; Jimenez-Sanchez et al. 2016; Yi et al. 2019), while protodioscin ($C_{51}H_{84}O_{22}$) constitutes the majority of the saponin content in white asparagus (Pegiou et al. 2021). Rutin, nicotiflorin (kaempferol-3-*O*-rutinoside) and a small quantity of chlorogenic acid predominate in seedlings (Eum et al. 2020). Rutin (60%–80%) constitutes a significant portion of the antioxidant activity in green spears protecting cells and tissues (Motoki et al. 2012). Flavonoids detoxify ROS, degrade fungal CWDEs, fortify plant cell wall integrity, induce localised cell death and enhance resistance to wilt disease (Perincherry et al. 2021a). In vitro assays have demonstrated that flavonoids directly impede conidial germination and hyphal growth of *Fusarium* spp.

(Alami et al. 2022; Long et al. 2019; Rosado-Álvarez et al. 2014). They were shown to hinder the mycelial growth and hydrolase activity of *F. oxysporum* while causing alterations in the mycelium's ultrastructure (Mohamed et al. 2017). Moreover, flavonoids derived from *A. albus* inhibited the growth of *F. oxysporum* in vitro (Alami et al. 2022). Li et al. (2023b) reported the potential of quercetin in suppressing *Fusarium oxysporum* f. sp. *lycopersici* growth and enhancing wilt resistance in tomatoes. This suggests that flavonoids may similarly contribute to enhanced resistance against Fusarium wilt in asparagus. Overall, higher concentrations of flavonoids correlate with increased antioxidant activity, highlighting their significance in plant defence mechanisms (Alami et al. 2022).

Anthocyanins are a group of glycosylated polyphenolic flavonoids, water-soluble vascular pigments responsible for the green, white and purple colours in asparagus spears (Dong et al. 2019). Usually, they are unstable and easily degradable (Chrpová et al. 2021). Purple asparagus contains higher anthocyanin levels compared to the white and green varieties (Guo et al. 2020). This may indicate its potentially stronger antioxidant properties (Fan et al. 2015; Sakaguchi et al. 2008). Dong et al. (2019) identified 16 anthocyanins from the purple and green asparagus cultivars, of which peonidin, cyanidin (Figure 2) and their glucoside derivatives were the most prominent representatives. As antioxidants, anthocyanins protect plants from extreme temperature, drought and salinity by preventing lipid oxidation and membrane damage (Pervaiz et al. 2017). They also signal stress, scavenge ROS and act as photoprotectants under stress conditions (Chrpová et al. 2021). Bernardi et al. (2018) observed that high anthocyanin levels in plants like red maize cultivar Rostrato Rosso conferred resistance to *F. verticillioides*. These studies indicate the potential of anthocyanin to contribute to the defence mechanisms of asparagus against *Fusarium* spp.

4.3 | Saponins

Saponins are a group of glycosylated compounds composed of a water-soluble glycidic chain and lipid-soluble triterpenes or steroidal aglycone (Hamdi et al. 2018). Asparagus contains various saponins such as asparanin A, protodioscin, dioscin, prosapogenin, sarsasapogenin and yamogenin (Figure 3), contributing to its unique bitter taste. Hayes et al. (2008) described five saponins, including shatavarin (I, IV and V), immunoside and schidigerasaponin D5 (or asparanin A) from *A. racemosus* roots.



Anthocyanin	R^1	R^2
Delphinidin	OH	OH
Cyanidin	OH	H
Pelargonidin	H	H
Petunidin	OCH_3	OH
Peonidin	OCH_3	H
Malvidin	OCH_3	OCH_3

FIGURE 2 | Structures of some major subclasses of flavonoids and anthocyanins (Šamec et al. 2021).

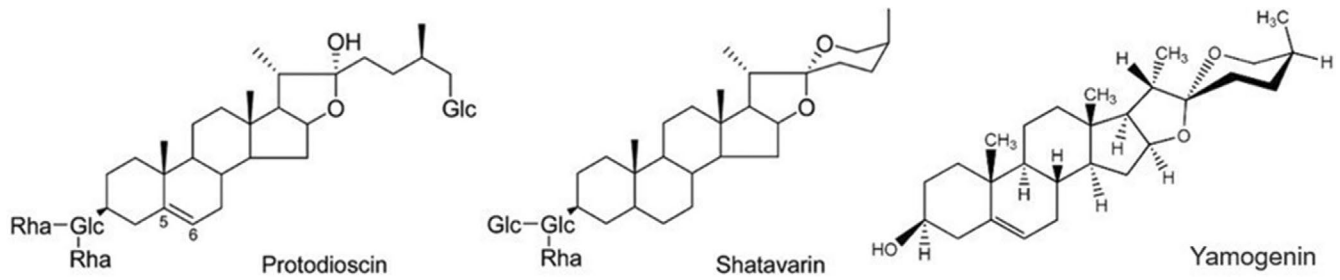


FIGURE 3 | Structures of protodioscin, shatavarin and yamogenin. Adapted from Jaramillo-Carmona et al. (2017).

Pegiou et al. (2021) identified shatavarin (VI or V, IX) and protodioscin in white asparagus spears. Protodioscin is the main saponin in many *Asparagus* species, such as *A. maritimus* and *A. prostratus* (Jaramillo-Carmona et al. 2017; Pegiou et al. 2019). Saponins are known for their potent antifungal and cytotoxic properties (Srivastava et al. 2018). During fungal attack, they promote oxidative stress in fungi, disrupt fungal cell membranes, suppress fungal enzymatic activity, protect the plant cell wall, induce cell death and activate defence-related signalling pathways (Shah et al. 2014).

4.4 | Benzoxazinoids

Benzoxazinoids (hydroxamic acids), such as 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA; Figure 4), are commonly found in grasses, cereals and certain other plants (Ferruz et al. 2016). Lalak-Kańczugowska et al. (2023) showed that DIMBOA from asparagus suppressed *F. proliferatum* growth and affected FB content and *FUM1* transcription in vitro. In wheat, DIMBOA inhibits the production of 15-acetyldeoxynivalenol and suppresses the expression of *TRI* genes, thereby providing resistance to *F. graminearum* (Etzerodt et al. 2015).

4.5 | Other Metabolites

Terpenoids, lignin, stilbenoids and alkaloids (Figure 4) are pivotal players in plant–microbe interactions (Mattio et al. 2021). Sulphur-containing phytoalexins in healthy plants exist as phytoanticipins, but under attack, they are converted to phytoalexin antimicrobial compounds. Sesquiterpenoid phytoalexins, like zealexins from maize, exhibited antifungal activity against several fungi (*F. graminearum*, *Aspergillus flavus*, *Rhizopus microsporus*) (Huffaker et al. 2011). Stilbenoids represent a small class of plant polyphenolic SMs synthesised prior to pathogen infection (Li et al. 2024). Among them, resveratrol (3,4′,5-trihydroxystilbenoid) is studied for its antioxidant properties (Valletta et al. 2021). Resveratrol possesses three hydroxyl (−OH) groups, while pterostilbene contains two methoxy ($-OCH_3$) groups and one −OH group (Valletta et al. 2021). Fungal invasion might also trigger lignin deposition and accumulation of lignans and volatile organic compounds (VOCs), which act as a barrier against *Fusarium* growth. Huang et al. (2007) isolated four distinct types of lignans, (+)-nyasol, 30-methoxynyasin, syringaresinol-40 and syringaresinol-4-0-b-D-glucopyranoside, from *A. officinalis* roots. Remarkably, both white and green spears were found to emit distinct VOC profiles, with higher concentrations typically observed at the upper parts, gradually decreasing towards the base (Pegiou et al. 2019). Among the diverse VOCs detected, a collection of aldehydes (pentanal, hexanal, (*E*)-2-hexenal, heptanal and octanal), aromatics (phenylacetaldehyde or benzeneacetaldehyde and 3-methylbutanal), alcohols and ketones (1-octen-3-ol, 1-penten-3-ol, 1-pentanol and 2-butanone), monoterpenes and benzenoids (such as styrene and limonene) and sulphur-containing compounds (asparagusic acid) emerged as key players in the chemical arsenal of asparagus (Pegiou et al. 2019). Moreover, alkaloids found in asparagus include asparagamine A, aspastipuline and 5-hydroxyaspastipuline (Galala et al. 2015). Alami et al. (2022) provided evidence of the inhibitory effects of alkaloids from *A. albus* on the growth of *F. oxsporum* in vitro.

5 | Synergistic Effects and Metabolite Profiles in the Asparagus–*Fusarium* Interaction

Plant metabolites do not act in isolation but often exhibit synergism to enhance their antimicrobial activity. When combined, these metabolites produce stronger antifungal responses compared to their individual components (Badr et al. 2022). This synergism is especially evident in plant extracts or exudates, which contain complex mixtures of metabolites with antimicrobial properties. In vitro studies have highlighted significant antifungal activity from extracts of asparagus against *F. oxysporum* and *F. proliferatum* (Alami et al. 2022; Desoukey et al. 2020; Górna et al. 2016; Rosado- Álvarez et al. 2014; Witaszak et al. 2020). Additionally, root exudates of *A. densiflorus* plants inhibited spore germination and germ tube growth of *F. oxysporum* and *F. proliferatum* in vitro (He et al. 2001). Furthermore, in vivo assays revealed the potential of *Lamiaceae* herb extracts to reduce *F. oxysporum* disease incidence and promote higher root and shoot biomass in asparagus plants (Ahmad and Matsubara 2020). Similarly, the use of extracts from other plant species, such as *Ammi visnaga* and *Eucalyptus globulus*, has been shown to enhance seed germination and enzymatic content (POX, polyphenol oxidase) and reduce H_2O_2 content in rice seedlings inoculated with *F. fujikuroi* (Kalboush and Hassan 2019). These findings suggest the synergistic potential of plant-derived metabolites to bolster plant defences against *Fusarium* spp.

Variations in metabolite profiles, driven by genotype, environmental conditions, and developmental stage might influence the strength of these synergistic actions of host metabolites in mitigating *Fusarium* infections in asparagus (Abdelrahman et al. 2020; Fuentes-Alventosa et al. 2008). Wild species like *A. prostratus* and *A. maritimus* possess distinct saponin

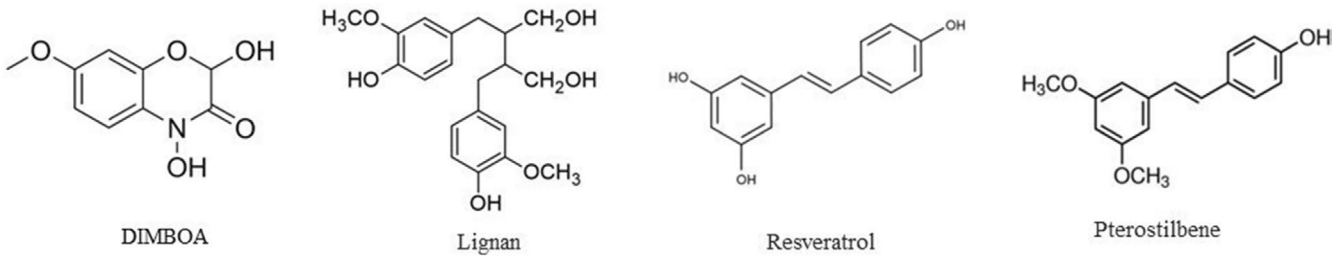


FIGURE 4 | Structures of 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA), lignans and the stilbenoids resveratrol and pterostilbene.

profiles compared to the more commonly cultivated *A. officinalis* (Jaramillo-Carmona et al. 2017), which may affect their resistance. Kulczyński et al. (2016) found that green asparagus extracts tend to have higher levels of flavonols and phenolic acids, which are associated with increased antioxidant activity, compared to the white variety. Other comparative studies indicate that white spears are characterised by significant levels of benzenoids, monoterpenes, unsaturated aldehydes and steroidal saponins, whereas green spears contain more C5 and C8 alcohols and aldehydes, along with higher levels of phenolic compounds (Pegiou et al. 2021). It has also been reported that white asparagus contains a higher level of sugars, proline and terpenoids (such as tocopherols, phytosterols and protodioscin), whereas green asparagus has high levels of flavonoids, policosanols and primary metabolites (organic and amino acids) (Kim et al. 2024). These differences in metabolite profiles suggest that metabolite diversity is not only species-dependent but also cultivar-specific, which can influence the plant's resistance to *Fusarium* spp.

Additionally, environmental factors such as temperature, light and nutrient availability profoundly impact these metabolite profiles. For example, higher temperatures tend to increase phenolic levels, while cooler temperatures elevate saponin content (Qaderi et al. 2023). Light quality is also crucial (Hamdi et al. 2020), as blue and white light promote anthocyanin accumulation, whereas red light and UV-C have the opposite effect (Yu and Fan 2021). In an experiment, where asparagus was grown in darkness, anthocyanins were not produced. Consequently, the biosynthetic and regulatory genes related to anthocyanin production were significantly downregulated, indicating that light is essential for anthocyanin accumulation in asparagus (Dong et al. 2019). In addition, terpenoid biosynthesis was activated under dark conditions, whereas flavonoid metabolism was activated under light conditions (Hamdi et al. 2020; Kim et al. 2024). Conversely, excessive shading can reduce polyphenol content and decrease antioxidant activity and activities of SOD, CAT and POX (Cao et al. 2022). Younger seedlings typically exhibit higher concentrations of SMs than older ones, which can influence the plant's overall resilience towards *Fusarium* infection. Such differences in metabolite composition suggest the need to select genotypes with enhanced metabolic profiles for improved resistance to *Fusarium* infections.

6 | Regulation of Host Metabolite Production in Response to *Fusarium* Infection

The regulation of host metabolite production plays a critical role in plant defence against *Fusarium* infection. Upon pathogen detection, plants activate the phenylpropanoid pathway, leading to the synthesis of antifungal metabolites such as phenolics, flavonoids and lignin (Deng et al. 2024; Luo et al. 2023). This pathway involves certain genes which contribute to the plant's resistance to fungal attack. For instance, the upregulation of lignin biosynthetic genes like cinnamate-4-hydroxylase (C4H) and cinnamyl alcohol dehydrogenase (CAD) helps form physical barriers against fungal colonisation (Boba et al. 2017; He et al. 2002). Similarly, the study by Boba et al. (2017) confirmed that the induction of this pathway led to the activation of genes involved in lignin, flavonoid and methyl salicylate biosynthesis in flax in response to *F. oxysporum* infection (Boba et al. 2017). Recently, 'multi-omics' analyses demonstrated that phenylpropanes, monoterpenoids and isoquinoline alkaloid biosynthesis were highly upregulated in *F. oxysporum*-infected roots (Deng et al. 2024). Plant hormones such as salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) play crucial roles in coordinating these pathways. The SA pathway, in particular, activates SAR, promoting the expression of genes like *NPR1* and *PR1* that are crucial for defence against biotrophic pathogens. Moreover, SA or its methylated form, methyl salicylate (MeSA), promotes lignification via PAL-dependent pathways, thereby contributing to the physical resistance against fungal infection (He and Wolyn 2005). In like manner, methyl jasmonate has been shown to enhance flavonoid and lignin production in response to *Fusarium* in various crops such as tomato, strengthening resistance (Król et al. 2015). TFs also play pivotal roles in regulating these metabolic pathways. NAC (no apical meristem), MYB, bHLH (basic helix–loop–helix), WRKY and APETALA2/ethylene response factor (AP2/ERF) families of TFs bind to the promoter regions of genes involved in SM production and hormone signalling (Chen et al. 2023; Javed and Gao 2023; Li et al. 2022, 2023c, 2024; Pan et al. 2018; Rojas et al. 2014). For example, WRKY TFs are known to be upregulated in resistant cultivars, leading to increased levels of phenolics and other antifungal compounds (Javed and Gao 2023; Soni et al. 2021). For example, the HvWRKY23 TF in barley regulates flavonoid biosynthesis to thicken the cell wall and limit *Fusarium* spread (Karre et al. 2019). Similarly, MYB58 and MYB63 regulate lignin biosynthesis in *Arabidopsis* during secondary cell wall formation (Zhou et al. 2009), while TaNAC032 in wheat modulates lignin biosynthesis to reinforce cell walls against *F. graminearum* (Soni et al. 2021). AP2/ERF factors, like ERF1 and ERF4, mediate ET signalling to optimise SM synthesis during stress (Deng et al. 2024; Li et al. 2024). Epigenetic modifications such as DNA methylation and histone modification also influence metabolite production by modulating defence gene expression during *Fusarium* infection. Le et al. (2014) demonstrated that DNA demethylases like ROS1 (REPRESSOR OF SILENCING 1) and DML2/3 (DEMETER-LIKE 2, 3) in *Arabidopsis* have been implicated in resistance to *F. oxysporum*. Epigenetic regulation has been implicated in modifying WRKY-mediated stress responses (Javed and Gao 2023) and signalling pathways such as MAPK, which are crucial for resistance in wheat (Kumar et al. 2020). Epigenetic modifications interact with SM pathways, MAPK signalling, hormonal transduction and WRKY-mediated *Fusarium* stress to ensure coordinated defence responses (Kumar et al. 2020). Conversely, *Fusarium* pathogens can disrupt these defences by secreting effector proteins or mimicking hormones that disrupt plant immune signalling, leading to altered tissue development and increased susceptibility.

7 | Challenges in Breeding for Resistance Against *Fusarium* in *Asparagus*

Currently, no asparagus cultivar exhibits complete resistance to *Fusarium* spp. Breeding for resistance genotypes is challenging due to the adaptability and genetic diversity of these pathogens. Over time, *Fusarium* strains can evolve to bypass certain host defences, often developing mechanisms to neutralise or even exploit host metabolites to persist and inflict damage on the plant

TABLE 4 | Studies on the interaction between *Asparagus* and *Fusarium*.

Plant type	Factors	Dose employed/*Fusarium* strain	Application effects	References
A. officinalis 'Mary Washington'	Root and shoot extracts multiple SMs	400 and 600 $\mu g\,mL^{-1}$ Foa	More inhibition percentage of mycelial growth with increasing dosage	Desoukey et al. (2020)
A. albus	Alkaloids, flavonoids, tannins	10% *F. oxysporum*	Antifungal	Alami et al. (2022)
A. officinalis	Spear extracts multiple SMs	*F. proliferatum*	Upregulation of 73 proteins mainly involved in sugar metabolism, stress response (HSP, SOD and glutaredoxins) and signal transduction	Górna et al. (2017)
A. officinalis	White spear extracts multiple SMs	*F. proliferatum* in vitro	Induced high FB expression until day 7, expression of *FUM1* gene	Górna et al. (2016)
A. officinalis	Methanolic fraction	*F. proliferatum* in vitro	Increased mycelium dry weight, higher fumonisin content, expression of *FUM1*	Witaszak et al. (2020)
A. officinalis	Salicylic acid	20 $mg\,L^{-1}$	Activated SAR, induced POX, PAL and lignin, reduced root disease severity	He and Wolyn (2005), Waśkiewicz et al. (2013b)
A. officinalis	Extracts	10 $ng\,\mu L^{-1}$ in vitro	Increased mycelia growth and fungal biomass, higher FBs detected	Stepień et al. (2015)
Triguero asparagus byproduct extracts	Flavonoids (quercetin, kaempferol, isorhamnetin glycosides)	Foa in vitro	Inhibition of mycelial and spore germination, no inhabitation of asparagus seed growth and germination	Rosado-Álvarez et al. (2014)
A. officinalis	Caffeic acid, rosmarinic extracts of *Lamiaceae* herb	20% wt/vol in vivo Foa	Lower disease incidence and indices, higher shoots/roots biomass	Ahmad and Matsubara (2020)
A. officinalis 'Welcome'	Caffeic acid, ferulic acid, malic acid	0.01, 0.1%, wt/vol, Foa in vitro	More Fao severity with 0.01%, 0.1% caffeic, ferulic and malic acids	Liu and Matsubara (2018)
A. officinalis	Caffeic acid, ferulic acid, quercetin, malic acid from root extracts	0.01, 0.1%, wt/vol, Foa in vitro	Suppressed propagation of Fao, more inhibition as concentration increases	Liu and Matsubara (2018)

(Continues)

TABLE 4 | (Continued)

Plant type	Factors	Dose employed/*Fusarium* strain	Application effects	References
A. officinalis	DIMBOA, protodioscin (1000, 100, 10 and 1 μg mL^{-1})	*F. proliferatum* in vitro	DIMBOA inhibited fungal growth, delayed FB_1 synthesis and expression of *FUM6*, protodioscin inhibited fungal growth, induced *FUM6, FUM19* expression but did not affect the content of FB_1	Lalak-Kańczugowska et al. (2023)
A. officinalis	Ferulic acid, chlorogenic acid, neochlorogenic acid (1000, 100, 10 and 1 μg mL^{-1})	*F. proliferatum* in vitro	Suppressed fungal growth except chlorogenic acid, ferulic acid inhibited the expression of *FUM6* but not *FUM19*	Lalak-Kańczugowska et al. (2023)
Water extracts from white asparagus spears	Quercetin-3 glucoside, kaempferol 3 rutinoside, iso-3-Rut (1000, 100, 10 and 1 μg mL^{-1})	*F. proliferatum* in vitro	Induced expression of *FUM19*, only iso-3-Rut suppressed fungal growth	Lalak-Kańczugowska et al. (2023)
A. officinalis 'Grande' and 'Vegalin'	Origin, temperature (15°C–30°C), and water availability	Foa 10^6 conidia mL^{-1} in vitro	High water availability combined with high temperature positively influenced pathogenicity of Foa	Brizuela et al. (2021)
A. officinalis 'Grande'	Sodium ethyl *p*-hydroxybenzoate, sodium hypochlorite, cinnamic aldehyde, water-soluble chitosan	*F. concentricum* 10^6 conidia mL^{-1} in vitro	Antifungal activities against mycelial growth and sporulation, as well as the spore germination	Zhang et al. (2022)
A. officinalis	Nutrient availability (P, Mn, Fe, Ca), soil dehydrogenase, low soil pH	Populations in asparagus fields	More FCRR, increased *Fusarium* population with increase soil fertility (P) especially in September, higher disease incidence in asparagus fields	Yergeau et al. (2006)
A. officinalis 'Guelph Millenium' and 'Jersey Giant'	Mn availability, planting depth, plantation age	Asparagus field	More FCRR with low Mn availability. Mn enhanced phenolic acids autotoxic in asparagus field soil, more infection due to plantation age	Hamel et al. (2005)
A. officinalis 'Welcome'	Salinity (200 mM NaCl)	Foa 10^6 conidia mL^{-1}	Increased susceptibility of asparagus to Fao, promoting disease infection	Haque and Matsubara (2018)

(Continues)

TABLE 4 | (Continued)

Plant type	Factors	Dose employed/*Fusarium* strain	Application effects	References
A. officinalis	Inoculation density	*F. oxysporum* (Foxy I–Foxy III) 10^4, 10^5, 10^6 conidia mL^{-1} in vitro	As the inoculation density increases, the more the infection	Kathe et al. (2019)
A. officinalis 'Grande'	1% pelleted poultry manure	Foa 10^6 conidia mL^{-1}	Reduced disease severity, increased plant biomass	Borrego-Benjumea et al. (2015)
A. officinalis	Nonpathogenic *F. oxsporum*	Foa 10^6 conidia mL^{-1} in vivo	ISR associated with the increase in antioxidants	Elmer (2004), He et al. (2002), Liu and Matsubara (2016), Reid et al. (2002)
A. officinalis	AMF-arbuscular mycorrhiza	*F. proliferatum* 10^6 conidia mL^{-1} in vivo	Lower disease severity, disease tolerance was related to specific increase in free amino acids	Haque and Matsubara (2018), Liu and Matsubara (2016), Matsubara et al. (2014)
A. officinalis 'Welcome'	AMF-arbuscular mycorrhiza	Foa 10^6 conidia mL^{-1}	Lower disease severity, increased SOD, APX, 1,1-diphenyl-2-picrylhydrazyl polyphenol and ascorbic acids	Nahiyan and Matsubara (2012)
A. officinalis	*Phialocepahala fortinii* CKG.I.11 endophyte	Foa in vivo greenhouse	Suppressed disease severity organically	Surono and Narisawa (2018)
A. officinalis 'Grande'	PGPR strains *Bacillus paralicheniformis* Hvs2, *B. velezensis* FC37, 10^8–10^9 bacteria mL^{-1}	*F. proliferatum*, Foa, *F. redolens* in vitro and in vivo 10^6 conidia mL^{-1}	Degraded fungal cell wall and fungal mycelium growth by the production antimicrobial compounds with the exception of *F. redolens*, improved plant growth parameters, reduced diseased severity	de la Lastra et al. (2021a)
A. officinalis 'Grande'	*Trichoderma* spp.	Foa 10^6 in vitro, 5×10^6 conidia mL^{-1} for semi-field trial	*Trichoderma* spp. inhibited Foa growth in vitro, less disease control in greenhouse (seedlings and plants) and semi-field tests	Brizuela et al. (2023)
A. officinalis 'Grande'	*Yarrowia lipolytica, saccharomycetous yeast* (10^6–10^9 cells mL^{-1}	*F. proliferatum* 10^6 in vitro	Reduced disease incidence, respiration, energy (ATP content), boost defence related enzymes, and increase antifungal SM content of asparagus	Godana et al. (2024), Hu et al. (2021)
A. officinalis	Nano CuO and MnO	Foa, *F. proliferatum*, *F. solani*	Reduced disease severity	Elmer et al. (2023)

Abbreviations: APX, ascorbate peroxidase; DIMBOA, 2,4-dihydroxy-7-methoxy-1,4 benzoxazin-3-one; FB, fumonisin B; FCCR, Fusarium crown and root rot; Foa, *Fusarium oxysporum* f. sp. *asparagi*; HSP, heat shock protein; iso-3-Rut, isorhamnetin-3-*O*-rutinoside; ISR, induced systemic resistance; PAL, phenylalanine ammonia-lyase; POX, peroxidase; SAR, systemic acquired resistance; SM, secondary metabolite; SOD, superoxide dismutase.

under specific conditions. The persistent *Fusarium* soilborne spores exacerbate disease pressure, and the polygenic nature of resistance traits complicates breeding efforts. Additionally, asparagus' long breeding cycle delays progress, and environmental factors influence resistance expression, leading to inconsistent results. The narrow genetic diversity in cultivated varieties further limits the pool of resistant traits, while screening for resistance remains labour-intensive and requires controlled conditions. Efforts to improve genetic diversity using landraces and wild relatives remain under-used, despite their potential for introducing resistance traits and enhancing bioactive compound production. To overcome these challenges, breeders must adopt innovative approaches. Table 4 gives a concise summary of research conducted on the interaction between *Asparagus* and *Fusarium*, emphasising disease dynamics, host responses and emerging solutions.

8 | Conclusions and Future Perspectives

Advances in research on host resistance metabolites involved in plant–microbe interactions have provided novel opportunities to generate *Fusarium*-resistant crop germplasms. Using host metabolites for *Fusarium* resistance involves several strategies. Analytical methods such as gas chromatography–mass spectrometry (GC–MS), liquid chromatography –mass spectrometry (LC–MS) and nuclear magnetic resonance (NMR) spectroscopy allow for precise metabolome and transcriptome profiling. Biotechnological advancements support these efforts by targeting specific genes or pathways necessary for metabolite production and enhancing natural defences through selective breeding and genetic engineering. Resistance-related metabolite traits can be derived from wild relatives or landraces that have co-evolved with the pathogen. Furthermore, gene-editing tools like CRISPR-Cas9 allow for precise editing or modification of defence-related genes involved in metabolite biosynthesis. Thus, editing TFs to enhance the phenylpropanoid pathway could increase the level of antifungal metabolites in asparagus.

The interaction between asparagus and *Fusarium* pathogens presents an intricate interplay of biochemical processes that are crucial for understanding plant–pathogen dynamics. The asparagus plant deploys a complex array of metabolites, including phenolic and non-phenolic compounds, which act as both antimicrobial agents and signalling molecules within its defence pathways. Detailed information about the response of asparagus towards *Fusarium* spp. at the molecular and metabolic levels is lacking at present. This review highlights the need for in-depth genetic and biochemical studies to elucidate host–pathogen interactions, identify resistance markers and clarify the mechanisms of action of specific antimicrobial SMs. *Fusarium* strains respond variably to host metabolites, emphasising the importance of understanding the composition and antioxidant properties of these compounds.

Future research should focus on characterising metabolite profiles in both resistant and susceptible asparagus varieties, aiming to identify biomarkers linked to resistance. Additionally, it is essential to investigate the regulatory networks governing metabolite production, as the genes involved could potentially be targeted using advanced biotechnological methods, such as Clustered regularly interspaced short palindromic repeats (CRISPR) and RNA interference (RNAi), to boost natural plant defences. These genetic approaches could also facilitate a deeper understanding of how environmental conditions affect metabolite production, which could inform agronomic practices that optimise plant health. Integrating metabolomics with genomic, transcriptomic and proteomic analyses will provide a more holistic view of the asparagus–*Fusarium* interaction, helping researchers to identify novel targets for genetic improvement. In practical terms, developing asparagus biostimulants from these metabolites would enable sustainable and organic-friendly disease management, further minimising environmental impact. Field studies are also crucial to assess the real-world effectiveness of enhanced metabolite profiles and to determine how they translate to disease resistance in various agricultural settings.

In conclusion, host metabolites play a central role in plant defence against *Fusarium*, providing both direct antifungal activity and signalling capacity that primes plant immunity. As a natural, multifaceted approach, purified metabolites align well with sustainable farming practices and present a viable method to control *Fusarium* disease. A continued focus on molecular biology, metabolomics and genetics will help to develop *Fusarium*-resistant asparagus cultivars, ultimately reducing economic losses and supporting sustainable asparagus production. By advancing research on this front, it is possible to build a foundation for effective, environmentally conscious strategies against *Fusarium* pathogens, enhancing resilience in the face of evolving agricultural challenges.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analysed.

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