Isabel M. L. Saur, Ralph Hückelhoven (2021) Recognition and defence of plant-infecting fungal pathogens. Journal of Plant Physiology 256: 153324 https://doi.org/10.1016/j.jplph.2020.153324

Accepted Manuscript

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Recognition and defence of plant-infecting fungal pathogens

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Abstract

Attempted infections of plants with fungi result in diverse outcomes ranging from symptom-less resistance to severe disease and even death of infected plants. The deleterious effect on crop yield have led to intense focus on the cellular and molecular mechanisms that explain the difference between resistance and susceptibility. This research has uncovered plant resistance or susceptibility genes that explain either dominant or recessive inheritance of plant resistance with many of them coding for receptors that recognize pathogen invasion. Approaches based on cell biology and phytochemistry have contributed to identifying factors that halt an invading fungal pathogen from further invasion into or between plant cells. Plant chemical defence compounds, antifungal proteins and structural reinforcement of cell walls appear to slow down fungal growth or even prevent fungal penetration in resistant plants. Additionally, the hypersensitive response, in which a few cells undergo a strong local immune reaction, including programmed cell death at the site of infection, stops in particular biotrophic fungi from spreading into surrounding tissue. In this review, we give a general overview of plant recognition and defence of fungal parasites tracing back to the early 20th century with a special focus on *Triticeae* and on the progress that was made in the last 30 years.

Abbreviations: AVR, avirulence; BX, Benzoxazinoid; DAMP, damage-associated molecular pattern; ETI, effector triggered immunity; f-actin, filamentous actin; FHB, Fusarium head blight; LRR, Leucine-rich repeats; LysM, Lysin Motif; MAMP, microbe associated molecular pattern; NB-LRRs or NLRs, nucleotide-binding leucine-rich repeat receptors; PAMP, pathogen associated molecular pattern; PRR, pattern recognition receptor; PTI, pattern triggered immunity; QTLs, quantitative trait loci; R, resistance; RK, receptor kinase; RLK, receptor-like kinase; RLP receptor like protein; STB, Septoria tritici bloch; SNB, Stagonospora nodorum blotch, SNB; S, susceptibility; SA, salicylic acid; SF, susceptibility factor; wall associated receptor kinase, WAK.

Keywords: avirulence, barley, cell-autonomous defence, disease resistance, disease susceptibility, effector, fungal parasite, plant immunity, receptor, *Triticeae*, wheat

1 Introduction

For adapted fungal pathogens, plants represent a source of nutrients and provide a favourable environment for parasitic growth. In the past, plant breeders could only distinguish between crops displaying disease symptoms and those lacking symptoms upon pathogen attack. Today we know that when pathogens attempt host infection, they face the plant immune system. But even before the advent of modern genetics and molecular biology, phytopathologists had described the existence of variable races of fungal phytopathogens with differential infection phenotypes on individual host lines and had observed that radiation can affect disease outcomes by eliciting inheritable changes in host and pathogen genes (Anderson and Hart, 1956; Bridgmon and Wilcoxson, 1959; Caldecott et al., 1959; Flor, 1942, 1960). Because of their economic importance, studies of crops and crop pathogens were of particular interest in the middle of the 20th century. These studies led to the conceptualization and development of the first near-isogenic lines with specific resistance traits and ultimately the onset of cereal crop genetics (Briggle, 1969; Day, 1966; Dyck and Samborski, 1968; Flor, 1956; Moseman and Schaller, 1960).

The ability to genetically manipulate plants and the development of model plants later laid the groundwork for a clearer elucidation of what occurs at the molecular level when a plant becomes infected with fungi. The basic principles of molecular plant-microbe interactions were largely elucidated based on the results of studies from model plant-pathogen systems but are thought to be generally applicable to interactions between *Triticeae* and fungal pathogens.

1.1 Recognition of fungal pathogens by plants

It has been known for decades that fungal-derived components must be recognised by plant hosts as specific host responses elicited during infection were associated with reduced fungal proliferation (Esquerretugaye et al., 1979; Hadwiger and Beckman, 1980; Kogel, G. et al., 1988; De Wit and Roseboom, 1980). Some of these components, termed fungal elicitors, were biochemically purified and their actions on host cells further defined (De Wit and Kodde, 1981; Kogel, G. et al., 1991), and many of these molecules were shown to induce a host cell death response (Beardmore et al., 1983; De Wit et al., 1984; Mayama et al., 1986). Currently, elicitors are generally divided into two classes. The first are so-called microbe- or pathogen-associated molecular patterns (M/PAMPs), which are molecules commonly released by a whole class of microbes including non-pathogens. The second class are referred to as effectors, which are specific to certain pathogen species or races and manipulate host components to promote the pathogen's virulence (Jones and Dangl, 2006).

1.2 Recognition of microbial patterns

M/PAMPs are variable molecules that include peptides and polysaccharide. M/PAMPs are usually recognised in the host apoplast through extracellular-facing transmembrane proteins such as receptor kinases (RLKs) and receptor-like proteins (RLPs) called pattern recognition receptors (PRRs) (Fig. 1) (Couto and Zipfel, 2016; Smakowska-Luzan et al., 2018). The fungal cell wall component chitin is a prominent example for a fungal M/PAMP (Felix et al., 1993; Kombrink et al., 2011; Kuchitsu et al., 1993). The first PRRs that sense chitin were isolated from *Arabidopsis thaliana* and rice (Kaku et al., 2006; Miya et al., 2007), and later, functional homologs were also identified in *Triticeae* (see section '*Triticeae* cell surface receptors that mediate *Triticeae* basal immunity to fungal parasites' below). Detection of M/PAMPs by PRRs activates signal transduction across the plasma membrane and ultimately induces immune responses collectively called pattern-triggered immunity. One well-described immune response that is effective against fungal invaders involves the production and secretion of plant chitinases to limit fungal proliferation (Schlumbaum et al., 1986). Penetration of host cellular structures by fungal invaders may also be arrested by immune responses such as host cell wall

strengthening including lignification and callose deposition (Bishop et al., 2002; Ellinger et al., 2013). Fungal attempts to penetrate host cells can also lead to the release of so-called damage-associated molecular patterns (DAMPs) from the intracellular to the extracellular space. DAMPs are host components released or produced and secreted upon cell or cell wall damage and are perceived by PRR-like proteins in the extracellular space leading to signalling outputs similar to PTI (Choi and Klessig, 2016). The perception of D/M/PAMPs may greatly contribute to resistance to infection by non-adapted pathogens (Niks and Marcel, 2009; Nürnberger and Lipka, 2005).

1.3 Recognition of fungal virulence effectors

The fungal elicitors now designated effectors are pathogen components that promote fungal proliferation on certain hosts (see section Effectors hijack *Triticeae* immunity and steer host physiology toward fungal accommodation' below). Most effectors are proteinaceous or metabolites, and fungal effectors are sometimes described as toxins. The molecular functions of numerous fungal effectors in their host apoplast have been described in detail (He et al., 2020; Lo Presti et al., 2015), and the molecular principles underlying plant-microbe interactions suggest that at least some effectors target the microbial recognition machinery of the host downstream of D/M/PAMP recognition, i.e. they interfere with PTI (Dodds and Rathjen, 2010; Jones and Dangl, 2006). Effectors that activate cell death pathways to promote virulence have also been described, but these are specific to necrotrophic fungi, which benefit from dead host tissue (Tan et al., 2010; Wang, X.L. et al., 2014).

The localised host cell death also known as the hypersensitive response (HR) elicited during host infection with obligate biotrophic pathogens or by the infiltration of biotroph elicitors into host tissue is usually the result of effector recognition by host immune receptors encoded by host *Resistance* (*R*) genes (Cui et al., 2015; Dodds and Rathjen, 2010; Greenberg and Yao, 2004). The HR is likely heavily implicated in arresting the invasion of biotrophs because these pathogens strictly require living host cells for proliferation, as already speculated by Ward and Marshall in 1902 and Stakman in 1915 (Stakman, 1915; Ward, 1902). The termination of fungal infection by R protein function is known as race-specific resistance and also referred to as effector-triggered immunity (ETI) (Fig. 1). The genetic concept underlying ETI was first described by Harold Flor, who investigated the genetic basis of flax (*Linum usitatissimum*) resistance to the flax rust fungus *Melampsora lini* (Flor, 1942). Flor's investigations led to development of the gene-for-gene theory, which stipulates that resistance to a specific pathogen isolate is determined by an *R* gene in the plant, and a corresponding *Avirulence* (*Avr*) gene in the pathogen (Flor, 1956). It has become clear that *Avr* genes usually encode effector proteins and that these effectors are secreted (Fig.1) by pathogens to promote pathogen proliferation on susceptible hosts e.g. to overcome basal host immune responses (i.e. PTI).

1.4 Resistance as a host response to fungal effectors

It is thought that the evolution of secreted effector proteins resembles an evolutionary response to plant basal immunity and further led to the evolution of plant *R* genes that often encode proteins for effector recognition (receptors). As such, the recognition specificities of *R* genes towards pathogen effectors and the resulting ETI are often considered an evolutionary countermeasure to adapted plant pathogens.

Because many *R* genes were found to encode nucleotide-binding leucine-rich repeat receptors (NB-LRRs or NLRs) the terms 'R protein' and 'ETI' are often used to specifically describe NLRs with recognition specificities towards intracellular effectors encoded at pathogen *Avr* genes. However, studies on the interaction of fungal pathogens of cereals and other crops have shown that *R* gene products are variable. For example, they can also encode receptors for the detection of apoplastic effectors (Fig.1) or have enzymatic activity that disables the virulence function of fungal effectors (e.g.

detoxification of fungal toxins; see sections '*Triticeae Resistance (R)* gene products as sensors of fungal invasion' and 'Non-conventional *Resistance* genes, *Susceptibility* genes and major QTL' below).

Here, we provide an overview of mechanisms underlying fungal defence in *Triticeae*. We highlight molecular principles of pathogen recognition and discuss advances in understanding fungal effector-mediated infection strategies. Finally, we summarize findings that give insight into how a *Triticeae* host terminates fungal infection. We apologize to those colleagues, who contributed to the field but whose publications have not been discussed here, because we aimed to condense available data to a digestible volume.

2 Triticeae cell surface receptors that mediate basal immunity to fungal parasites

Plants sense fungal pathogens at the cell surface/plasma membrane via PRRs (Fig. 1). Indeed, an immunogenic elicitor from *Puccinia graminis* f. sp. *tritici* was isolated already in the late 1980ies and found to specifically bind to the plasma membrane of wheat and barley cells (Kogel, G et al., 1988; Kogel, Gerd et al., 1991). In spite of this early demonstration, we still know relatively little about *Triticeae* cell surface PRRs. Some candidate PRRs have been identified in *Triticeae*. For instance, a candidate barley chitin receptor (HvCEBiP) with two putative glucan-binding LysM motifs in the extracellular domain of the predicted protein has been identified based on its similarity to the rice chitin-elicitor binding protein CEBiP and on its function in basal resistance to the blast fungus *Magnaporthe oryzae* (formerly known as *Magnaporthe grisea*)(Tanaka et al., 2010). In wheat, candidates for both TaCEBiP and its co-receptor TaCERK1 (chitin elicitor receptor kinase1) have been described for the interaction with *Zymoseptoria tritici* (formerly known as *Mycosphaerella graminicola*), the causal agent of Septoria tritici bloch (STB) (Lee et al., 2014). Despite the sequence- and function-based evidence that these proteins indeed play roles in chitin sensing and signalling, direct evidence for binding to chitin-derived elicitors or for functions in canonical early PTI-responses is still lacking.

Chitin and laminarin (a mixed-linked glucan elicitor considered mimicking fungal and oomycete elicitors) elicit typical PTI responses, such as an apoplastic pH-shift, an oxidative burst and MAP kinase activation in *Triticeae* (Felle et al., 2004; Scheler et al., 2016; Torres et al., 2017; Wanke et al., 2020; Wawra et al., 2016). Although numerous elicitor activities have been described in *Triticeae* (Schweizer et al., 2000; Shetty et al., 2009; Vander et al., 1998), we are not aware of any further candidate PRR that senses a structurally characterised fungal elicitor. However, additional cell surface RLKs act in basal resistance to fungal pathogens and as such qualify as candidate PRRs. This is further supported by their predicted structural similarity to known PRRs. RLKs with ectodomains containing leucine-rich repeats, wall-associated epidermal growth factor domains, malectin domains, legume lectin domains, or cysteine-rich domains function in resistance to fungal pathogens based on evidence from forward or reverse genetics in barley or wheat (Table 1). Little is known about the molecular nature of corresponding fungal elicitors that act on *Triticeae*. Identifying immunogenic ligands of candidate PRRs remains challenging. *Vice versa*, identifying the receptor for a known elicitor can also be challenging in *Triticeae* and other cereals. However, with the development of early elicitor response assays in *Triticeae*, classical forward genetics, the recent proliferation of resources for genome-wide association studies and pan-genomics in *Triticeae*, these challenges are now becoming experimentally tractable.

Cell surface receptor-like kinases also contribute to cell wall sensing and mechanosensing (Ackermann and Stanislas, 2020), and fungi exert physical pressure on plant cell walls and membranes when growing between or into living cells. Mechanical stress is by itself sufficient to trigger aggregation of cytoplasm and cell polarization to the site of applied force (Gus-Mayer et al., 1998; Hardham et al., 2008). Although they have not been reported in *Triticeae*, mechanosensors and cell wall sensors could potentially be identified based on homology to sensors from model plants.

3 Triticeae Resistance (R) gene products as sensors of fungal invasion

Specific lines of a certain host plant that completely lack or display only low susceptibility to an adapted pathogen are known as resistant. Already decades ago, breeding for resistant traits and the generation of near-isogenic cereal lines, also known as inbred or backcross lines, demonstrated that resistance traits can be dominantly or recessively inherited (Jorgensen, 1994; Keller et al., 2018). Mapping and cloning of dominantly inherited *R* genes and the molecular characterisation of their products has shown that most *R* genes encode immune receptors to sense specific pathogen effectors, which in this context are also designated AVR effectors (Fig. 1).

The wheat wall-associated receptor kinase (WAK) STB6 is the only so-far identified *Triticeae R* gene that recognises a specific fungal AVR in the apoplast (Brading et al., 2002). *Stb6* is also the only isolated *R* gene effective against *Z. tritici* and the effector recognised by STB6 is a small cysteine-rich effector protein designated AvrStb6 with yet unknown virulence function (Table 2) (Zhong et al., 2017). The majority of immune receptor-encoding *Triticeae R* gene loci, which are effective against fungal pathogens, have a complex genetic architecture encoding multiple *NLR* genes. In the last 20 years, numerous NLR-encoding *R* genes have been mapped and the NLRs that underlie resistance have been molecularly isolated. Here, we solely discuss NLRs that have been at least to some extend molecularly characterised and for which corresponding pathogen AVR effectors have been cloned (Table 2).

Identified Avirulence effectors recognised by Triticeae NLRs

The only barley NLRs for which the matching AVR effectors have been isolated are encoded at the *Mildew locus a* (*Mla*). *Mla* resistance against the barley powdery mildew fungus *Blumeria graminis* f. sp. *hordei* (*Bgh*) has been studied for over 60 years (Jorgensen, 1994; Moseman and Schaller, 1960). *Mla* was found to be subjected to extensive functional diversification and over 20 *Mla* NLR alleles have so far been molecularly isolated from different barley lines each conferring disease resistance to an individual set of *Bgh* isolates (Halterman et al., 2001; Maekawa et al., 2019; Seeholzer et al., 2010; Zhou et al., 2001). Very recently, six AVR effectors recognized by MLA (designated AVR_a effectors) have been cloned and five of them encode sequence-unrelated small secreted proteins belonging to different candidate secreted effector families (Lu et al., 2016; Saur et al., 2019).

3.1 Recognition of powdery mildew effectors

Similar to barley *Mla*, wheat Pm3 NLRs have undergone extensive functional diversification, as documented by the molecular isolation of 17 *Pm3* recognition specificities towards isolates of the wheat powdery mildew fungus *Blumeria graminis* f. sp. *tritici* (*Bgt*) (Bhullar et al., 2009; Bhullar et al., 2010; Yahiaoui et al., 2004). The first powdery mildew AVR effector to be isolated was *Bgt* $AvrPm3^{a2/f2}$ recognized by the *Pm3a* and *Pm3f* alleles and this was followed by the isolation of the sequence unrelated $AvrPM3^{B2/C2}$ and $AvrPM^{3D}$ effector genes recognized by *Pm3b*, *Pm3c* and *Pm3d,* respectively (Bourras et al., 2019; Bourras et al., 2015). Similarly, the wheat *Pm2* resistance gene and its matching *Bgt* AVR gene *AvrPM2* have only been isolated recently (Praz et al., 2017; Sanchez-Martin et al., 2016). However, it remains to be determined if sequence variation of different *Pm2* alleles represent functional diversification towards *Bgt* isolates carrying independent *AvrPm2* variants (Jin et al., 2018).

3.2 Recognition of rust effectors

The emergence of new destructive races of the wheat stem rust fungus *Puccinia graminis* f. sp. *tritici* (*Pgt*) (TTKS race cluster, also termed Ug99) provided an impetus for research that culminated in the isolation and molecular characterization of new stem rust *R* genes (Singh et al., 2011). The archetypal NLR encoding *R* genes Sr50 (from rye) and Sr35 (from *Triticum monococcum)* were found to provide

resistance to Ug99 races when introgressed into common wheat (Periyannan et al., 2013; Saintenac et al., 2013). Only a few years after the cloning of these *NLR* genes, the effectors AvrSr50 and AvrSr35 recognized by Sr35 and Sr50, respectively, were isolated (Chen et al., 2017; Salcedo et al., 2017). AvrSr50 encodes a typical small secreted protein without sequence similarity to characterized proteins, whereas the *AvrSr35* candidate gene encodes a 578–amino acid protein with a predicted signal peptide (SP). Notably, infiltration of the 64 kDa recombinant AvrSr35 protein (without SP) into wheat leaves and heterologous expression of the construct in *N. benthamiana* leaf cells resulted in a *Sr35* dependent cell death response (Bolus et al., 2020; Salcedo et al., 2017), suggesting pathogen-independent uptake of AvrSr35 into wheat cells and recognition by the intracellular NLR Sr35.

3.3 Molecular functions of intracellular fungal AVR effectors on Triticeae hosts

The molecular functions of the so-far isolated fungal AVR effectors that act in *Triticeae* remain elusive. First insight may however come from the isolated *B. graminis* AVR effectors. Although most of the yet isolated *Blumeria* AVR effectors share no sequence homology, structural prediction platforms propose an RNase-like fold for most of the isolated AVR effector genes from barley and wheat powdery mildews, suggesting functional diversification of *Triticeae* NLRs in response to a conserved effector fold (Bourras et al., 2019; Bourras et al., 2015; Praz et al., 2017; Saur et al., 2019). Such RNase-like proteins make up the largest class of *B. graminis* candidate secreted effectors (Frantzeskakis et al., 2018; Pedersen et al., 2012; Pliego et al., 2013). A function in interference with the function of host ribosome-inactivating proteins in cell death regulation has been suggested, but effective ribosomal RNA association could not be detected (Pennington et al., 2019). Because the effectors also lack the residues crucial for RNA processing that overlap with the residues of the RNA binding pocket (Irie, 1997; Pedersen et al., 2012), the role of this fold in *B. graminis* virulence remains to be determined. Further, significant structural similarity was detected between the *P. tritici* ToxB effector and *M. oryzae* effectors AVR1-CO39 and AVR-Pik (de Guillen et al., 2015) and the virulence targets of this structural fold are most likely heavy metal-associated (HMA) domain containing proteins. This presumption is based on the observation that AVR1-CO39 and AVR-Pik are recognized by the HMA domains integrated into the structure of the respective rice NLRs RGA5 and Pikp-1 (Cesari et al., 2013; Maqbool et al., 2015). In fact, genome analysis revealed that approximately 10% of plant NLRs contain such variable integrated domains (Ellis, 2016), likely serving as mimics of AVR virulence targets and effector decoys. *Triticeae* NLRs harbour integrated domains previously shown to act as effector targets in other plant species. These include protein kinases, protein phosphatases, and transcription factors or class Exo70 exocyst subunits. Some of the identified domains have not yet been connected to pathogen virulence functions, but it is suggested that host non-NLR proteins with these domains are effector targets specifically on *Triticeae* or other grasses (Bailey et al., 2018; Ellis, 2016; Sarris et al., 2016).

3.4 NLRs as susceptibility factors

Because *R* protein activation is often associated with localized cell death, it is perhaps not surprising that effectors from necrotrophs target *Triticeae* NLRs as part of their virulence strategies. Toxin effectors produced by the necrotrophic fungi *Parastagonospora nodorum* and *Pyrenophora tritici-repentis*, causing Stagonospora nodorum blotch (SNB) and tan spot on wheat, respectively, apparently hijack immune receptors that typically provide resistance against biotrophic fungi (Fig. 1). The WAK kinase SNN1 and the NLR encoded by *Tn1* are targeted by the fungal toxins Tox1 and ToxA leading to cell death, and as such *Snn1* and *Tn1* are considered *Susceptibility* (*S*) genes in this context (see below)(Table 2) (Faris et al., 2010; Lamari et al., 2003; Liu et al., 2004; Shi, G.J. et al., 2016).

4 Non-conventional *resistance* genes, *susceptibility* genes and major QTL

Although the modes of action of the majority of molecularly characterised *R* genes involve receptor-mediated recognition of a pathogen's effector, genetic dissection of resistance traits has shown that a number of non-receptor-like coding *R* genes exist in nature (Table 3). Some of these confer gene-for-gene resistance, while others mediated broad-spectrum disease resistance. In addition, it has become clear that recessively inherited resistance traits are often associated with the disruption of gene function, which is why these genes are also referred to as *S* genes.

4.1 Detoxification of fungal metabolite effectors

Hm1 was the first *R* gene to be cloned (Johal and Briggs, 1992) and confers resistance to *Cochliobolus carbonum* race 1 (CCR1), which causes leaf spot of corn (*Zea mays*). However, virus-induced silencing of the barley *Hm1* homolog makes barley susceptible to CCR1, suggesting that *HvHm1* underlies or at least contributes to non-host resistance of barley to *Cochliobolus carbonum* (*Sindhu et al., 2008)*. Although the underlying mechanism is consistent with the gene-for-gene theory, *Hm1* does not encode a receptor. Instead, the reductase encoded by *Hm1* detoxifies the race-specific CCR1 toxin *Helminthosporium carbonum* (HC), which otherwise leads to disease symptoms in susceptible lines lacking *Hm1* (Johal and Briggs, 1992). Similarly, *Fhb7* encodes a glutathione S-transferase with the ability to detoxify trichothecene toxins produced by *Fusarium graminearum*, the major causal agent of the Fusarium head blight (FHB) disease of wheat and barley. Strikingly, *Fhb7* originates from a fungal endophyte, from which it was introduced into wheat by horizontal gene transfer (Wang et al., 2020). In addition to *Fhb7*, *Fhb1* provides effective resistance to FHB (Buerstmayr et al., 2009). Recently, the histidine-rich calcium-binding protein encoded by *TaHRC* was suggested to underlie *Fhb1* resistance. Disruptions such as early stop codons, intron retention and silencing of *TaHRC* boosts FHB resistance (Li et al., 2019; Su et al., 2019), and as such *TaHRC* may be considered a susceptibility factor, although its' exact molecular function remains to be determined.

4.2 Broad spectrum resistance to powdery mildews by recessive mlo genes

Another susceptibility factor is encoded at the *Mildew locus O* (*Mlo*), and homozygous mutations (*mlo*) lead to powdery mildew resistance in monocots and dicots (Büschges et al., 1997; Kusch and Panstruga, 2017). *mlo* was originally identified in barley as a recessive locus conferring broad-spectrum resistance to *Bgh* (Jorgensen, 1992). *mlo* resistance has been durable despite extensive field cultivation in spring barley. Resistant *mlo* genotypes display similar defence responses as susceptible *Mlo* genotypes albeit in an earlier and stronger fashion, and, hence MLO may control or suppress basal defence (Hückelhoven et al., 1999; Peterhänsel et al., 1997; Piffanelli et al., 2002; von Ropenack et al., 1998; Zierold et al., 2005). The MLO protein possesses seven transmembrane domains and was found to associate with calmodulin, and this association contributes to susceptibility. MLO may also function in cell death regulation as *mlo* mutants shows spontaneous cell death phenotypes and the cell death suppressor BAX Inhibitor-1 can ectopically complement *mlo*-resistance. However, the mechanism by which MLO suppresses defence towards powdery mildews in *Triticeae* remains to be determined (Büschges et al., 1997; Hückelhoven et al., 2003; Kim et al., 2002; Wolter et al., 1993).

4.3 Adult plant resistance and broad spectrum disease resistance loci

A large proportion of quantitative trait loci (QTLs) for resistance to wheat rust fungi encode adult plant resistance genes (APR, reviewed in (Ellis et al., 2014)). Only some of these have been cloned and at least so far the identity of the *APR* does not give any insights into possible generalities underlying this specific type of resistance. The APR genes cloned so far include the leaf rust (caused by *Puccinia triticina*) resistance genes *Lr34* (encoding a putative ABC transporter (Krattinger et al., 2009)), *Lr67* (encoding a putative hexose transporter (Moore et al., 2015), the yellow (or stripe) rust (caused by *Puccinia striiformis f. sp. tritici, Pst*) resistance genes *Yr36* (encoding a protein kinase (Fu et al., 2009))

and *Yr15* (encoding a tandem kinase-pseudokinase (Klymiuk et al., 2018)). How functions of these genes translate into rust resistance is not yet known. Importantly, *Lr34* also underlies powdery mildew resistance encoded by the *Pm38* locus and stripe/yellow rust resistance at *Yr18*. Such multiple disease resistance loci effective against the obligate biotrophic powdery mildew and multiple rust fungi has also been reported for *Lr67/Yr46/Sr55/Pm46* and *Lr46/Yr29/Sr58/Pm39* but the latter APR gene has not yet been isolated.

5 Effectors hijack *Triticeae* immunity and steer host physiology toward fungal accommodation

Fungal pathogens utilize so-called effector molecules to manipulate host functions for their own benefit (Fig. 1). The largest class of characterised effectors are probably effector proteins, but small RNAs and metabolites are also secreted during host infection and these molecules have been shown to act as virulence factors too.

5.1 Proteinaceous effectors secreted by fungal pathogens during host infection

Proteinaceous effectors can either be secreted into the host apoplast or enter the host cell interior by mechanisms which are currently unknown. Although conceptual modes of action are well established for effectors, the large variety of effectors is a major obstacle hindering molecular characterization of effector functions. Effectors often suppress host functions in immunity, others may be toxic and inhibit several cell functions in a manner, which is not specific to a given host. However, effectors are also postulated to exert intricate effects on host susceptibility factors (see below) that steer host functions to best meet the demands of pathogen nourishment or accommodation of pathogen infection structures in intact cells (e.g. haustoria of rust or powdery mildew fungi, invasive hyphae of *M. oryzae*). In such cases, pathogen effectors may be considered as activators of co-opted host physiological or developmental programs. Effectors that trigger programmed cell death in the host via specific host receptor proteins represent another case of activating effectors,; these necrotrophic effectors are often referred to as toxins (see Table 2, ToxA and Tox1) (McDonald and Solomon, 2018). This effector-mediated cell death can be considered as an inverse of the gene-for-gene concept, because here single receptor genes mediate host susceptibility, while matching single pathogen genes encode host-specific toxins (Faris and Friesen, 2020; Faris et al., 2013).The apoplastic effector Pep1 from the smut fungus genus *Ustilago spec.* seems to be a classical example of a fungal suppressor because it can suppress host PTI (reactive oxygen species production) across the border of families and even promotes susceptibility to powdery mildew when ectopically expressed in barley (Hemetsberger et al., 2015). LysM effectors represent an excellent example of a conserved suppressive fungal effector function: LysM effectors were initially described as effectors from*Cladosporium fulvum*, but were subsequently also found as virulence factors in *Z. tritici*, *M. oryzae and other fungal pathogens* (*Bolton et al., 2008; Lee et al., 2014; Marshall et al., 2011; Mentlak et al., 2012). Z. tritici* Mg3LysM, Mg1LysM and MgxLysM have partly redundant function and together they can protect fungal hyphae from chitinase activity. Mg3Lysm sequester chitin elicitors that otherwise would trigger CEBiP/CERK1-dependent immunity, and hence Mg3Lysm is crucial for virulence (Lee et al., 2014; Marshall et al., 2011). The Mg1Lysm effector was recently shown to protect fungal cell walls. A structure-biology based model suggests that it does so by forming a chitin-ligand independent oligomer. However, this oligomer seems to be nucleated by an initial chitin-binding effector dimer (Sánchez-Vallet et al., 2020). Mechanistically similar, the fungal-specific β-glucan-binding lectin FGB1, which is expressed by the non-parasitic endophyte *Piriformospora indica* (*synonym: Serendipita indica),* binds to elicitor-active cell wall glucans and may outcompete host glucan receptor binding (Wawra et al., 2016). Degradation of the host's cell wall is another example for a conserved function of effectors acting in the apoplastic space. Cell wall-degrading enzymes have been described as effectors in numerous *Triticeae* infecting fungi (Brunner et

al., 2013; Choi et al., 2013). These enzymes *may counteract host cell wall defence mechanisms* and were shown to be crucial during early infection/penetration e.g. by *F. graminearum* and *M. oryzae (Carapito et al., 2008; Phalip et al., 2009; Skamnioti and Gurr, 2007).*

All these well-studied fungal effectors mostly act in the host apoplast. In turn, the small secreted protein Zt6 from *Z. tritici* is one of the few molecularly characterized effectors from *Triticeae* infecting fungi that is able to enter the wheat cell cytoplasm. Zt6 is a functional RNase and can non-selectively hydrolase ribosomal RNA from a variety of organisms. Alternatively, because loss of virulence cannot be observed for *zt6* mutants, Zt6 may act promiscuously towards other microbes (Kettles et al., 2018). A molecular function inside wheat host cells has also been proposed for the stripe rust effector PstGSRE1 (glycine-serine-rich effector): PstGSRE interacts with wheat LOL2 (lesion simulating disease 1(LSD1)-like zinc finger protein) to interfere with nuclear transport of LOL2 and LOL2-regulated reactive oxygen species-stimulated defence gene expression (Qi et al., 2019).

5.2 Fungal effectors as suppressors of host cell death

In addition, recent findings suggest that some intracellular effectors can suppress of ETI: *Bgt* SvrPm3$^{A1/F1}$ suppresses cell death triggered by the recognition of *Bgt* AvrPm3$^{A2/F2}$ through wheat Pm3 in heterologous *Nicotiana benthamiana* (Bourras et al., 2015; Bourras et al., 2016; Parlange et al., 2015). Similarly, several *P. graminis* and *P. striiformis* effectors can suppress cell death mediated by the co-overexpression of unrelated *R* genes in the *N. benthamiana* system (Ramachandran et al., 2017). This supports the notion that fungal virulence not only acts on basal plant defence but may also target the ETI cell death response for triggering susceptibility. The high number of effectors encoded by some fungal parasites, including *B. graminis*, could be down to a strategy of keeping certain *R*-gene products under control of fungal suppressors and hence phenotypically silent (Thordal-Christensen, 2020).

5.3 *Fungal effectors as activators of host susceptibility pathways*

It is postulated that biotrophic fungal effectors also activate host susceptibility pathways, which serve the pathogen´s demands rather than control host immunity (Fig. 1) (Engelhardt et al., 2018). One possible example for this might be ROPIP1 from *Bgh*. This effector is translocated into the host cytosol by an unknown mechanism where it interacts with a host ROP GTPase called RACB. RACB has a function in polar cell development of healthy plants, e.g. in outgrowth of root hairs. RACB further controls cytoskeleton organization without necessarily negatively regulating host immune responses. It has thus been suggested that ROPIP1 acts on RACB to take over its function in polar cell growth and promote ingrowth of the fungal haustorium into intact epidermal cells of barley, because the cellular mechanisms underlying haustorial ingrowth and root hair outgrowth appear similar (Hoefle et al., 2011; Nottensteiner et al., 2018; Opalski et al., 2005; Scheler et al., 2016).

5.4 Secondary metabolites as fungal effectors

Fungal secondary metabolites are potent mycotoxins and often have modes of action that are not specific to a given host. Despite this lack of specificity, one can consider mycotoxins as both cell death-inducing agents that support the necrotrophic lifestyle and suppressors of host defence, because dead or dying cells cannot defend themselves effectively from infection. Recently, it was shown that fungal toxins such as trichotecenes are produced in specialized infection cushions of *F. graminearum* and that trichotecenes may promote local weakening of the host tissue (Mentges et al., 2020). Infection stage-specific regulation of genes for secondary metabolite production was also reported for other *Triticeae* pathogens, e.g. the fungus *Ramularia collo-cygni* (Sjokvist et al., 2019), which has recently developed into a global threat to barley production (Havis et al., 2015).

5.5 Small RNAs as fungal effectors through RNA interference

Cross-kingdom RNA interference represents another strategy used by pathogenic fungi in their hostile take-over of plant cellular processes and involves fungi exploitation of the host gene silencing machinery by secretion of small RNAs with sequence complementarity to host defence genes (Weiberg et al., 2013). In *Triticeae,* the wheat *PR2* defence gene was suggested to be targeted by a micro RNA-like RNA from *Pst* (Wang et al., 2017). Recently, target prediction revealed that *B. graminis* and *P. triticina* express small RNAs that could induce silencing of *Triticeae* host genes (Dubey et al., 2019; Kusch et al., 2018). It will be exciting to learn how exactly RNAs traffic between fungi and plants and why some host factors are targeted at the protein and other at the RNA level.

6 Susceptibility factors, Achilles heels of *Triticeae* crops

Our understanding of disease susceptibility has advanced greatly in the last 20 years and with that susceptibility became a target of breeding and targeted mutagenesis approaches (Büschges et al., 1997; Dangl et al., 2013; Engelhardt et al., 2018; Faris et al., 2010; Hückelhoven, 2005; Lapin and Van den Ackerveken, 2013; van Schie and Takken, 2014). Initially, this field of research was driven by forward genetic approaches that identified the genes underlying recessively inherited resistance such as barley *mlo* and Arabidopsis *pmr* genes (Büschges et al., 1997; Schulze-Lefert and Vogel, 2000). Today, it is increasingly accepted that host susceptibility factors (SFs, Fig.1) contribute to pathogenesis using diverse modes of action. Some SFs may control host immunity in environments or situations, in which the plant does not face biotic stress or when a biotic stress response needs to be downregulated after successful defence. Corresponding *S* gene mutants constitutively activate defence responses and often show spontaneous cell death and lesion mimic phenotypes. Other SFs may serve pathogen demands for nutrient supply or cellular re-organization during pathogen accommodation (Engelhardt et al., 2018; Lapin and Van den Ackerveken, 2013; van Schie and Takken, 2014).

6.1 The use of s *mutants and* S *factors for breeding*

Corresponding *s* mutants often show developmental failure or growth depression. SFs can be direct effector targets but can also indirectly support pathogen success by contributing to “being a good host”, which is crucial for the proliferative success of biotrophic parasites. The physiological roles of SFs in their hosts means that *s* mutants are often pleiotropic and difficult to use in breeding programs (Hückelhoven et al., 2013; van Schie and Takken, 2014). However, the future use of TILLING (Targeting Induced Lesions In Genomes) or allele mining in wild grasses to uncover *S* allele diversity or targeted mutagenesis approaches using endonucleases could pave the way for efficient conversion of susceptibility into resistance (Engelhardt et al., 2018). Indeed, it was demonstrated that *mlo*-resistance can be established in hexaploid wheat by targeted nuclease-mediated mutagenesis (Wang, Y. et al., 2014) but also by selection of the right alleles from TILLING without major pleiotropic effects (Acevedo-Garcia et al., 2017). As mentioned above, necrotrophs also exploit SFs by necrotrophic effectors. Such SFs resemble components involved in mediating immunity to biotrophs such as NLRs or RLKs (Faris et al., 2010; McDonald and Solomon, 2018; Shi, G. et al., 2016).

7 Cell autonomous and non-cell autonomous defence in *Triticeae*

Strikingly, we still only poorly understand what actually stops a fungus from growing on resistant plants. However, we know that tissue-wide resistance is achieved by a number of preformed metabolic antimicrobial compounds collectively called phytoanticipins (VanEtten et al., 1994). E.g. Benzoxazinoids (BXs) are phytoanticipins mostly found in grasses and their biosynthesis from indole has been thoroughly studied (Elnaghy and Shaw, 1966; Elnaghy and Linko, 1962; Niculaes et al., 2018).

Biocidal BXs are thought to rapidly act against microbial invaders because a variety of BXs are preformed and stored as non-toxic BX-glycosides (de Bruijn et al., 2018). They are hydrolysed by glucosidases upon pathogen-provoked tissue disruption and release the toxic aglycone BXs into the plant apoplast, thereby directly affecting microbial proliferation (Morant et al., 2008). BXs may also affect fungal penetration attempts, because at least in maize, BX treatment regulates cell wall callose deposition and BX-mediated resistance to *Setosphaeria turcica* acts before the onset of major tissue damage (Ahmad et al., 2011).

After pathogen contact, activated plant defence reactions contribute to restricting fungal development. The cell biology of host-parasite interactions suggested that cell-autonomous defence contributes to diverse forms of full and quantitative resistance to fungal parasites (Fig. 2). As mentioned above, one obvious measure to stop biotrophic fungal growth is the hypersensitive reaction (HR), which involves programmed cell death of cells in direct contact with fungal infection structures (Dickman and Fluhr, 2013; Koga, 1994; Moerschbacher et al., 1990; Schiffer et al., 1997; Stakman, 1915; Tiburzy and Reisener, 1990). In some cases, such as late-acting barley *Mla*-gene mediated resistance, however, it is the surrounding cells rather than the cell under direct attack that die during HR (Boyd et al., 1995; Hückelhoven et al., 1999; Hückelhoven, R. et al., 2000; White and Baker, 1954). This might insulate the penetrated cells from further nutrient supply. However, it is not fully understood whether cell death is required for resistance or whether other defence reactions expressed during HR are sufficient to limit fungal growth (Görg et al., 1993; Kiraly et al., 1972; Koga, 1994; Schiffer et al., 1997). Interestingly, even necrotrophic fungi, which profit from host cell death, can be restricted from invasive growth by local HR, e.g. if HR is initiated early during the interaction in the epidermis of barley under attack from *Bipolaris sorokiniana* (Fig. 2) (Kumar et al., 2002).

7.1 Penetration resistance, a cell autonomous defence

Penetration resistance (prehaustorial resistance) is another important mechanism of cell-autonomous defence (Hückelhoven, 2014; Jarosch et al., 1999; Kumar et al., 2002; Neu et al., 2003; Niks and Dekens, 1991; Zeyen et al., 2002). Forward and reverse genetics and cell biological approaches have elucidated many of the details of cell-autonomous penetration resistance in the barley-powdery mildew pathosystem (Hückelhoven and Panstruga, 2011; Schulze-Lefert and Vogel, 2000). Studies on non-host, basal and *mlo*-mediated resistance have reported the formation of local cell wall appositions or papilla under fungal appressoria (Fig. 2) (Aist, 1976; Hückelhoven, 2014; Stolzenburg et al., 1982; Zeyen et al., 2002). Indeed, Smith already in 1900 described in detail papillae formed in response to grass powdery mildew fungi as host cell wall extensions formed during accommodation of fungal haustoria (Smith, 1900). Corner described papillae as associated with the failure of fungal penetration but concluded that host toxins may stop fungal development (Corner, 1935). Other results suggest that local secretion of defence compounds at sites of attempted fungal penetration is crucial for stopping fungal invasion. This involves the formation of multivesicular bodies and requires membrane vesicle budding, transport, tethering and eventually fusion with the target membrane for delivery of defence compounds (An et al., 2006; Bohlenius et al., 2010; Collins et al., 2003; Douchkov et al., 2005; Ostertag et al., 2013). Additionally, a functional filamentous (f)-actin and microtubule cytoskeleton is crucial for successful penetration resistance (Kobayashi et al., 1997; Miklis et al., 2007; Nottensteiner et al., 2018; Opalski et al., 2005) and the production of hydrogen peroxide in papilla is correlated with the efficacy of defence (Hückelhoven et al., 1999; Hückelhoven et al., 2000; Piffanelli et al., 2002; Thordal-Christensen et al., 1997) (Fig. 2). The mechanistic role of hydrogen peroxide and other reactive oxygen species in penetration resistance is not entirely clear because direct genetic evidence is sparse for *Triticeae* (Christensen et al., 2004; Navathe et al., 2019; Qi et al., 2019; Torres et al., 2017; Zimmermann et al., 2006). Because crosslinking of cell wall phenols and proteins depends on hydrogen peroxide, the most likely function of this molecule may lie in in oxidative cell wall crosslinking. Despite intense

research focus and an increasing understanding of the chemical composition of papilla (Chowdhury et al., 2014; Chowdhury et al., 2016), is remains unclear whether structural/mechanical or chemical defence, e.g. by phenlyamide phytoalexins (Stoessl, 1966; Ube et al., 2019a; Ube et al., 2019b; von Ropenack et al., 1998), or a combination of both leads to the arrest of fungal growth in the plant cell wall of *Triticeae* (Hückelhoven, 2014; Zeyen et al., 2002). Recent evidence, however, suggests that cell wall carbohydrates, amongst them callose and arabinoxylan, may be crucial for penetration resistance in barley (Chowdhury et al., 2014; Chowdhury et al., 2016; Douchkov et al., 2016). See also the book chapter of Zeyen and co-workers for earlier detailed evidence of chemical composition and metabolic pathways involved (Zeyen et al., 2002). By contrast, the function of those factors, which mediate the penetration resistance of *Triticeae* to powdery mildew fungi, has not been elucidated in comparable detail in the context of prehaustorial resistance to cereal rust fungi (Collins et al., 2007). However, the function of the gene *REQUIRED FOR mlo-SPECIFIED RESISTANCE 1* (*ROR1*) and of filamentous actin (f-actin) in penetration resistance against *B. graminis* and *M. oryzae* appears similar (Freialdenhoven et al., 1996; Jarosch et al., 2005). In summary, transport and secretion appear to be important for local fungal arrest on *Triticeae*, but is the identity of the molecules that are transported and secreted remains unknown. Recently, it was shown that not only fungi but also plants use small RNAs for cross kingdom RNAi. This biotechnological method of silencing pathogen genes through the expression of double-stranded RNAs in the host plant (host-induced gene silencing) thus emerges as a potential natural plant defence mechanism (Hou et al., 2019; Koch and Kogel, 2014; Nowara et al., 2010; Schaefer et al., 2020). Hence, it appears possible that, apart from peptides and small chemical defence compounds, RNAs are also secreted to fend off parasitic fungi. Interestingly, *Pgt* expresses PgtSR1, a silencing suppressor that apparently can interfere with the host´s RNAi machinery and weakens host resistance (Yin et al., 2019).

7.2 Systemic induced resistance and susceptibility to fungal pathogens in Triticeae

Fungus-induced non cell-autonomous local or systemic immunity to fungal pathogens has also been observed in *Triticeae* (Colebrook et al., 2012; Lyngkjaer and Carver, 2000; Schweizer et al., 1989; Waller et al., 2005). In particular, salicylic acid (SA) and its synthetic analogues such acibenzolar-S-methyl/benzothiadiazole have been implicated in systemic defence responses against fungi (Conrath, 2006; Görlach et al., 1996; Wang, X.D. et al., 2018). In *A.thaliana*, endogenous SA levels are strongly induced during biotic stress but this is not the case for barley plant infected with *B. graminis* (Hückelhoven et al., 1999; Saja et al., 2020; Vallelian-Bindschedler et al., 1998) and whilst SA induces the up-regulation of typical pathogen-related (PR) genes via the SAR master regulator NPR1 in *A. thaliana* (Pieterse et al., 1998), SA does not have the same effect in wheat and barley (Vallelian-Bindschedler et al., 1998; Wang, X.D. et al., 2018). Nevertheless, application of SA or acibenzolar-S-methyl to barley reduced *Bgh* infection (Besser et al., 2000; Lenk et al., 2018), and barley NPR1 functions in basal resistance to powdery mildew but not in systemic resistance induced by bacterial infection (Dey et al., 2014). In addition, overexpression of Arabidopsis NPR1 in wheat can lead to enhanced disease resistance to FHB (Makandar et al., 2006) and both NPR1 and PR proteins were identified as targets of effectors from *Pst, P. nodorum* and *Bgh* in yeast assays (Breen et al., 2016; Wang et al., 2016; Zhang et al., 2012), suggesting an important role of the typical PR genes also in defences against fungal pathogens in *Triticeae.* However, how this is connected to SA levels remains to be determined. Similarly, the function of other endogenous hormones in *Triticeae* resistance to fungal parasites may be specific to particular host-parasite interactions. Very recently, it was demonstrated that successful fungal pathogens can also induce susceptibility to subsequent infection by the same or other pathogens in distant tissues. During such an interaction, *Z. tritici* changes host secondary metabolite (i.e. benzoxazinoids and phenylpropanoids) composition and leaf microbiome of a

susceptible wheat host genotype, which together may contribute to systemic induced susceptibility (Seybold et al., 2020).

8 Future research

Our understanding of *Triticeae* pathology has advanced greatly in the last 30 years, progress, which has been largely facilitated by better genomic resources and the development of tools for functional genetics in small grain cereals (Alqudah et al., 2020; Feuillet et al., 2012; Hensel, 2020; Lin et al., 2020; Monat et al., 2019). Despite this, our mechanistic understanding of the factors that promote or hinder pathogen proliferation on a particular host genotype is still fragmented. We now have a relatively clear picture of some resistance and susceptibility factors regulating individual fungal-host interactions, but models explaining their biochemical functions are only available for classic receptor proteins. For some susceptibility factors, cumulative evidence suggests a role in developmental or physiological pathways that allow fungi to flourish in the susceptible host, but we urgently need deeper understanding and more precise genetic strategies such as targeted gene knock-out plants to improve our molecular understanding of a susceptibility pathways. We also largely lack structural data on *Triticeae* resistance proteins and higher-order complexes that they may form, which would likely also give insight into their modes of action. With respect to pathogen biology, further studies should provide a better understanding of how virulence effectors, in particular those acting inside plant cells, manipulate *Triticeae* host function and whether and how candidate RNA-based effectors function in two-way cross kingdom RNAi. Small host molecules, such as hormones, secondary metabolites and peptides with hormone-like functions are also incompletely understood or have been understudied in *Triticeae* resistance to fungal parasites. With the recently developed genomic, genetic, proteomics and metabolomics resources, we foresee rapid developments in functional biology of *Triticeae* disease resistance and susceptibility. This will most likely bear new mechanisms, which did not evolve in dicot models and pave the way for translational research and development within *Triticeae* and other grass crop plants.

9 Concluding remarks

More than hundred years of research on the interaction of fungal pathogens with *Triticeae* hosts revealed a treasure of fascinating mechanisms of host resistance to diseases caused by those fungi. We learned about host resistance genes, mechanisms of pathogen recognition, cellular and chemical defence. We have seen a major investment of society in supporting this research because the food supply threatened by pathogens and the biological principles underlying diseases are of socioeconomic and cultural value. However, society is divided when it comes to whether and how those common cultural goods should be applied by conventional and modern technologies. Although the authors do not see a general solution for this problem, they feel that educating pupils and undergraduates in respect to the beauty and diversity of plant disease resistance could perhaps help to tear down the walls. Our vision, indeed, would be a more biological way of crop protection that makes use of natural disease resistance in context of both conventional and organic farming systems. The definition of what "natural disease resistance" is might be based on whether a plant is resistant to diseases due to a mechanism that similarly takes place in nature rather than based on whether such a plant was collected in nature, bred by conventional crossing or hybridization, selected by molecular means or engineered by gene technologies. In these days of climate change, future yield gaps and global political insecurities, society needs to be active and progressive for tackling approaching challenges to agriculture and global food supply. In front of a crisis, the probably worst option to choose is not to choose. Thus, we propose to choose the potential of natural plant immunity independent of the technical route of its exploitation.

Conflict of interest

The authors have no conflict of interest to declare. Funding agencies have not been involved neither in the selection of the topic of this review nor at any stage of writing or editing.

Acknowledgments

I.M.L.S. is supported by the Daimler and Benz Foundation. Projects in the R.H. lab have been recently supported by the German Research Foundation (SFB924, HU886/8, HU886/11), by the Bavarian State Ministry of the Environment and Consumer Protection (TGC01GCUFuE69781), by the BASF Plant Science GmbH and by German Ministry of Economics and Energy (AIF 17221 N).

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Tables

Table 1. Examples of candidate cell surface receptors with potential function in basal resistance to fungal parasites in *Triticeae*

Gene	Predicted ectodomain	Function in basal resistance to	Reference
Barley			
CEBiP	Lysin Motif (LysM)	*Magnaporthe oryzae*	(Tanaka et al., 2010)
Rphq2/Rph22	Legume-like (L-type) lectin	*Puccinia hordei*	(Wang, Y. et al., 2019)
RNR8/LEMK1	Leucine-rich repeats/Malectin	*Blumeria graminis*	(Rajaraman et al., 2016)
LRRK-6H	Leucine-rich repeats (LRR)	*Fusarium graminearum*	(Thapa et al., 2018)
Wheat			
CEBiP	Lysin Motifs (LysM)	*Zymoseptoria tritici*	(Lee et al., 2014)
CERK1	Lysin Motifs (LysM)	*Zymoseptoria tritici*	(Lee et al., 2014)
LRRK-6D	Leucine-rich repeats	*Fusarium graminearum*	(Thapa et al., 2018)
WAK2	Galacturonan-binding wall-associated/ Epidermal Growth Factor-like	*Fusarium graminearum*	(Gadaleta et al., 2019)
RLK-V1	Malectin/Leucine-rich repeats	*Blumeria graminis*	(Hu et al., 2018)
LecRK-V	Legume-like (L-type) lectin	*Blumeria graminis*	(Wang, Z. et al., 2018)
RLK1, RLK2	Leucine-rich repeats	*Blumeria graminis*	(Chen et al., 2016)
CRK2	Cysteine-rich	*Puccinia triticina*	(Gu et al., 2020)
XA21	Leucine-rich repeats	*Puccinia striiformis*	(Wang, J. et al., 2019)

Table 2. Cloned *receptor* genes with matching isolated fungal effectors.

Gene	Receptor	Provides resistance to	Regulation	Recognised gene	Effector protein	References
Barley						
Mla1	NLR	*Blumeria graminis* f. sp. *hordei*	*R*[$] *gene*	*AVR$_{a1}$*	Small secreted	(Halterman et al., 2001; Zhou et al., 2001 Seeholzer et al., 2010; Lu et al., 2016; Saur et al., 2019)
Mla7	NLR	*Blumeria graminis* f. sp. *hordei*	*R gene*	*AVR$_{a7}$*	Small secreted	
Mla9	NLR	*Blumeria graminis* f. sp. *hordei*	*R gene*	*AVR$_{a9}$*	Small secreted	
Mla10	NLR	*Blumeria graminis* f. sp. *hordei*	*R gene*	*AVR$_{a10}$*	Small secreted	
Mla13	NLR	*Blumeria graminis* f. sp. *hordei*	*R gene*	*AVR$_{a22}$*	Small secreted	
Mla22	NLR	*Blumeria graminis* f. sp. *hordei*	*R gene*	*AVR$_{a22}$*	Small secreted	
Wheat						
Stb6	WAK*	*Zymoseptoria tritici*	*R gene*	*AvrStb6*	Small secreted, cysteine rich	(Brading et al., 2002; Zhong et al., 2017)
Sr35	NLR	*Pucinia graminis* f. sp. *tritici*	*R gene*	*AvrSr35*	66 kDa, secreted	(Saintenac et al., 2013; Salcedo et al., 2017)
Sr50	NLR	*Pucinia graminis* f. sp. *tritici*	*R gene*	*AvrSr50*	Small secreted	(Mago et al., 2015; Chen et al., 2017)
Pm2	NLR	*Blumeria graminis* f. sp. *tritici*	*R gene*	*AvrPm2*	Small secreted	(Sanchez-Martin et al., 2016 ; Praz et al., 2017)
Pm3a	NLR	*Blumeria graminis* f. sp. *tritici*	*R gene*	*AvrPm3$^{a2/f2}$*	Small secreted	(Yahiaoui et al., 2004; Bhullar et al., 2009, 2010; Bourras et al., 2015, 2019)
Pm3b	NLR	*Blumeria graminis* f. sp. *tritici*	*R gene*	*AvrPm3$^{b2/c2}$*	Small secreted	
Pm3c	NLR	*Blumeria graminis* f. sp. *tritici*	*R gene*	*AvrPm3$^{b2/c2}$*	Small secreted	
Pm3d	NLR	*Blumeria graminis* f. sp. *tritici*	*R gene*	*AvrPm3^{d3}*	Small secreted	
Tsn1	NLR	*Parastagonospora nodorum, Pyrenophora tritici-repentis*	*S*[§] *gene*	*SnToxA, PtrToxA*	Small secreted	(Faris, J. D. et al., 2010; Lamari et al., 2003; Liu et al., 2004; Shi, G.J. et al., 2016)
Snn1	WAK	*Parastagonospora nodorum*	*S gene*	*SnTox1*	Small secreted	

**WAK: Wall-associated receptor kinase*

[$] R gene: Resistance gene

[§] S gene: Susceptibility gene

Table 3. *Resistance* and *susceptibility* genes without predicted receptor/recognition domain.

Gene	Protein	Provides resistance to	Resistance	References
Barley				
Hm1	Reductase	*Cochliobolus carbonum* race 1	*Race specific (race 1)*	(Johal and Briggs, 1992; *Sindhu et al., 2008)*
***mlo*[§]**	Serpentine transmembrane protein	*Blumeria graminis* f. sp. *hordei*	Broad spectrum	(Jorgensen, 1992; Büschges et al., 1997)
Wheat				
Fhb1	histidine-rich calcium-binding	*Fusarium graminearum*	Broad spectrum	(Buerstmayr et al., 2009; Su et al., 2019; Li et al., 2019)
Fhb7	glutathione S-transferase	*Fusarium graminearum*	Broad spectrum	(Buerstmayr et al., 2009; Wang et al., 2020)
***mlo*[§]**	Serpentine transmembrane protein	*Blumeria graminis* f. sp. *tritici*	Broad spectrum	(Wang et al. 2014; Acevedo-Garcia et al., 2017)
Lr34/Yr18/Pm38	ABC transporter-like	*Puccinia triticina* *Puccinia striiformis* f. sp. t*ritici* *Blumeria graminis* f. sp. *tritici*	Broad spectrum APR[$]	(Krattinger et al., 2009)
Lr67/Yr46/ Sr55/Pm46	Hexose transporter-like	*Puccinia triticina* *Pucinia graminis* f. sp. t*ritici,* *Pucinia striiformis* f. sp. t*ritici* *Blumeria graminis* f. sp. *tritici*	Broad spectrum APR	(Moore et al., 2015)
Yr36	Protein kinase	*Pucinia striiformis* f. sp. t*ritici*	Broad spectrum APR	(Fu et al., 2009)
Yr15	tandem kinase-pseudokinase	*Pucinia striiformis* f. sp. t*ritici*	Broad spectrum APR	(Klymiuk et al., 2018)

[$] APR: adult plant resistance

[§]The dominant *MLO* gene is a susceptibility gene that encodes a serpentine transmembrane protein with similarity to G-protein coupled receptors

Figures

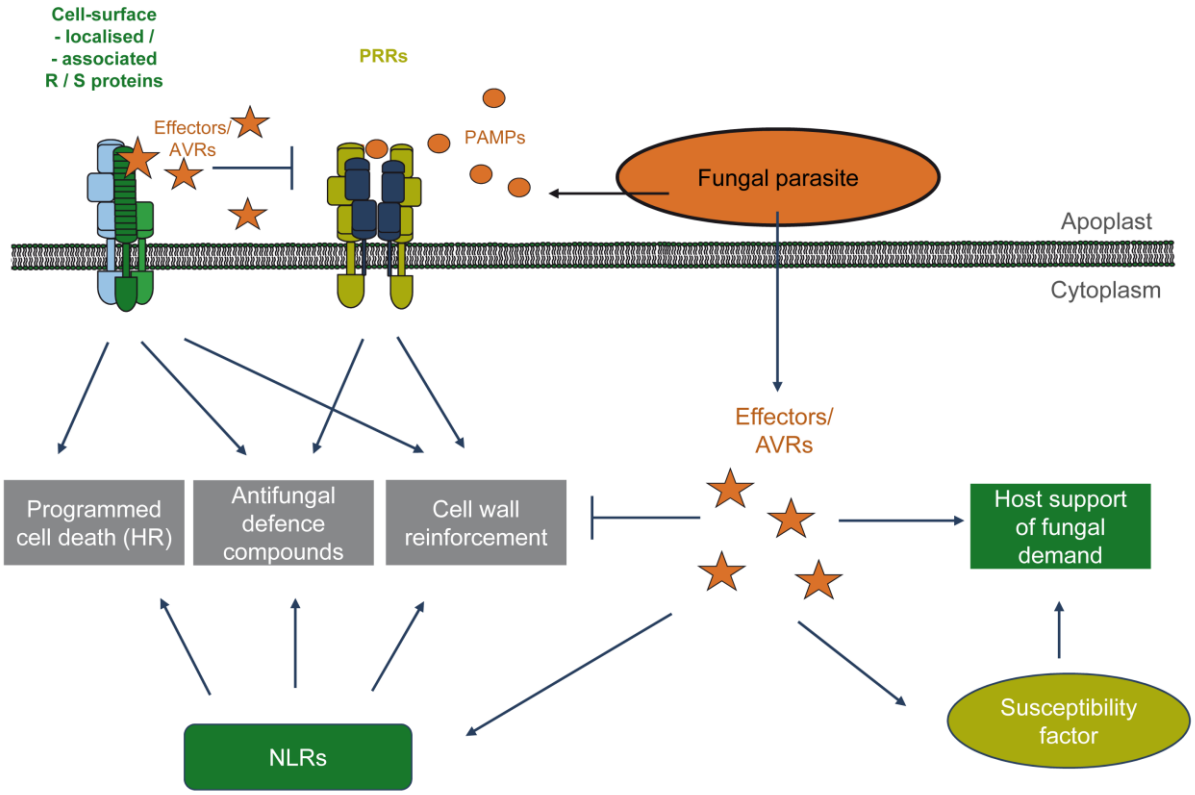

Figure 1. Key factors mediating plant resistance or susceptibility to parasitic fungi. Plants use several strategies to combat fungal invaders. Surface-localised pattern recognition receptors (PRRs) perceive conserved microbe-/pathogen-associated molecular patterns (M/PAMPs) released from the fungal parasite. This recognition leads to defence mechanisms involving cell wall strengthening and the production of anti-fungal defence compounds. Adapted fungi interfere with PRR signalling through the secretion of virulence effectors into the plant apoplast or cell interior and support the parasites requirements independently of defence suppression. In turn, immune complexes containing resistance proteins (R proteins) allow resistant plants to specifically recognise effectors in the apoplast or the cell interior, where R proteins re-enforce defence signalling, which is often associated with a hypersensitive response (HR), a specialized form of programmed cell death. Fungal effectors may also activate factors and pathways that fulfil the pathogen's requirements rather than control host immunity. This activation can involve host susceptibility factors.

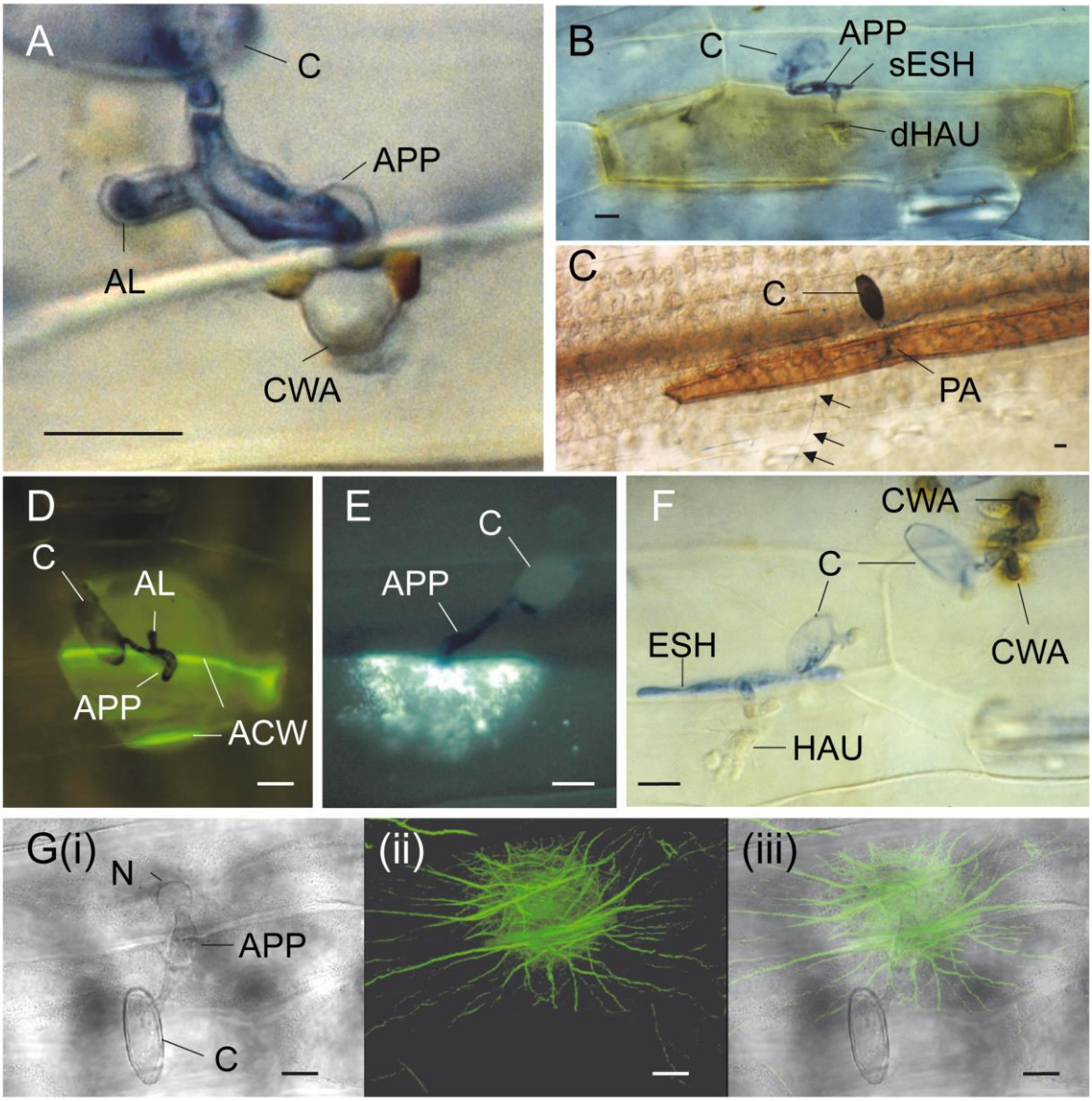

Figure 2. Cellular defence reactions of barley leaf epidermal cells to fungal attack. A) Fully developed cell wall apposition below an appressorium of *Bgh*. B) Single cell HR in barley carrying the *Mla12*-resistance gene for recognition of AVR_{A12} from *Bgh*. The cell in the image shows 3,3,-diaminobenzidine staining for H_2O_2 (Hückelhoven and Kogel, 2003). C) Single-cell barley HR response to attempted penetration by *B. sorokiniana*. Note that the fungus failed to penetrate but continued growing on the leaf surface (Kumar et al., 2002). D) Yellow-autofluorescent material (long pass filter) excited by blue light in barley epidermal cells under attack from *Bgh*. E) Fluorescent aniline blue-stained callose depositions in barley under attack from *Bgh*. F) Barley epidermal cells that are mounting defence responses against *Bgh* and show 3,3,-diaminobenzidine staining for H_2O_2 in cell wall appositions, or support fungal haustorium formation and do not stain for H_2O_2 (Hückelhoven et al., 2000). G) Barley cell with focussed and dense f-actin organization and nuclear positioning at sites of attempted penetration. (i) transmission channel at the confocal laser scanning microscope, (ii) Alexa488®-Phalloidin stained f-actin, (iii) merged pictures (Opalski et al., 2005). Sclae bars = 20µm. ACW, anticlinal cell wall; AL, appressorial lobe; APP, appressorium; C, conidium; CWA, cell wall apposition; dHAU, disintegrated haustorium; ESH, elongated secondary hyphae; HAU, haustorium; sESH, short elongated secondary hyphae; N, nucleus; PA, penetration attempt.