Unconventional R proteins in the botanical tribe Triticeae

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Abstract

Plant immunity is triggered following the perception of pathogen-derived molecules by plant receptor proteins. Two protein families, membrane-localized receptor-like kinases (RLK) and intracellular nucleotide binding-leucine-rich repeat (NLR) receptors, play key roles in pathogen perception and in the initiation of downstream signaling cascades that lead to defense responses. In addition to RLKs and NLRs, recent research has identified additional protein families that function as plant resistance (R) proteins. In particular, the botanical tribe Triticeae, which includes the globally important crop species wheat and barley, has played a significant role in the discovery of ‘unconventional’ R proteins. In this review, we will summarize the current knowledge on unconventional R genes in Triticeae and the proteins they encode. The knowledge on unconventional R proteins will not only broaden our understanding of plant-pathogen interactions, but will also have great implications for disease resistance breeding in crops.
Introduction

Plants in natural and agricultural ecosystems experience a constant exposure to biotic stresses caused by pathogenic viruses, bacteria, oomycetes, fungi, nematodes, and insects. In contrast to abiotic stress (e.g., drought, heat and salinity), biotic stress is caused by living organisms that evolve over time. This can lead to the emergence of new, highly virulent pathogen strains, sometimes with catastrophic consequences for agriculture. For example, a new race of the wheat stem rust fungus *Puccinia graminis* f. sp. *tritici* that was first reported in Uganda in 1999 caused serious problems for wheat production across East Africa and other regions (1). The emergence and rapid spread of tropical race 4 (TR4) of the fungal pathogen *Fusarium oxysporum* f. sp. *cubense* threatens banana production worldwide (2). It is estimated that about one fifth of the global crop harvest is lost to pathogens and pests (3). The same study concluded that there was no major improvement in crop health between 2001 and 2017 on a global scale, which underscores the challenges associated with the emergence of new pathogen variants (3).

In contrast to vertebrates, plants lack an adaptive immune system (defined as the ability to create an immunological memory after an initial exposure to a pathogen). Plant immunity relies on a preformed or innate immune system that is based on the perception of pathogen-derived molecules by immune receptors. Two protein families have been predominantly associated with plant immunity; cell-surface localized receptor-like kinases (RLK) and intracellular nucleotide binding–leucine-rich repeat (NLR) receptors (4, 5). These receptors directly or indirectly perceive apoplastic and intracellular pathogen-derived molecules, respectively, which triggers signaling cascades leading to immune responses (6-10).

In addition to RLK and NLR encoding genes, recent research has identified additional gene families that function as resistance (R) genes in plants. In this review, we will refer to such resistance genes and the proteins they encode as ‘unconventional’. The term ‘unconventional’ will be simply used to refer to non-RLK and non-NLR encoding genes and does not make any assumptions about the resistance phenotype or the underlying molecular and biochemical mechanisms. While most of the groundbreaking discoveries about RLK and NLR-mediated perception and signaling were made in the model plant *Arabidopsis*
thaliana, a significant portion of the knowledge on unconventional $R$ genes comes from non-model plant species. In particular, research on the botanical tribe Triticeae, which includes the important crops wheat (Triticum spp.), barley (Hordeum vulgare) and rye (Secale cereale), has led to the identification of novel $R$ gene families. In this review, we will thus focus on the recent discoveries on unconventional $R$ genes in Triticeae. Our definition of an $R$ gene is based on a recent review stating that an $R$ gene is ‘the polymorphic genetic component that makes the crucial difference between resistant and susceptible genotypes within a species’ (11). The focus of this review is on active $R$ genes that provide dominant or semi-dominant resistance. We will not discuss susceptibility genes, i.e., recessive genes where a loss-of-function mutation results in increased resistance. The most famous susceptibility gene in Triticeae is the barley Mildew resistance locus o (mlo). Multiple natural and induced loss-of-function Mlo mutants results in broad-spectrum resistance against powdery mildew. Mlo encodes a plasma membrane-localized protein with seven transmembrane domains (12, 13). According to the above definition, for example, more than 450 $R$ genes have been described in the wheat gene pool (14). The cloning (i.e., the identification of the nucleotide sequence encoding a resistance protein) of these resistance genes, however, has been slow and tedious in Triticeae. A major factor that has limited gene cloning in wheat and barley are the large and repeat-rich genomes. For example, the 15-gigabase bread wheat genome is five times larger than the human genome (15). The development and implementation of genomics-based resistance gene cloning protocols in recent years has significantly sped-up gene cloning in Triticeae (16-19), which has led to the recent identification of several unconventional $R$ genes. In this review, we classify unconventional $R$ genes based on recognition specificity into ‘race-specific’ and ‘broad-spectrum’. We follow the definition of Johnson (1984) who defined the term race-specific as resistance that interacts differentially with pathogen races, i.e., that produces compatible and incompatible reactions against different pathogen races in the same host genetic background (20). The term race-specific can be applied both to complete and partial resistance genes. This classification is of relevance for breeding because it provides valuable information about the durability and optimal deployment of $R$ genes and $R$ gene combinations.
Unconventional $R$ genes: race-specific resistance

Race-specific $R$ genes confer resistance against some but not all races of a pathogen. The genetic concept of race-specific disease resistance has been formulated by Harold Henry Flor in the 1940s and is known as the gene-for-gene relationship (21). Working on flax ($Linum usitatissimum$) and flax rust ($Melampsora lini$), Flor discovered that the inheritance of resistance is determined by pairs of matching genes; a resistance ($R$) gene in the host and a corresponding avirulence ($Avr$) gene in the pathogen. On a molecular level, race-specific resistance is typically conferred by NLR encoding genes in the plant, which directly or indirectly perceive pathogen-derived virulence effectors. The modification or loss of the corresponding effector protein in certain pathogen strains prevents its recognition and results in susceptibility of the host plant (22).

Of the 42 race-specific $R$ genes cloned in Triticeae, 33 encode NLRs or NLRs with integrated domains (NLR-ID) (11). In addition to NLRs, there are several examples of unconventional $R$ genes that confer race-specific disease resistance in Triticeae (Figure 1; Table 1).

The wheat leaf rust ($P. triticina$) resistance gene $Lr14a$ encodes a protein containing an N-terminal domain with twelve ankyrin repeats (ANK) and six transmembrane helices (TM) at the C-terminus (23). ANK domains have been linked to protein-protein interactions (23). Phenotypically, $Lr14a$ is characterized by a ‘mesothetic’ seedling resistance, where fully developed leaf rust pustules and hypersensitive flecks (a hallmark of race-specific disease resistance responses) occur on the same leaf. This phenotypic response sets $Lr14a$ apart from most other race-specific wheat leaf rust resistance genes, but it is not yet understood if the mesothetic phenotype is due to the unusual protein architecture of $Lr14a$. $Lr14a$ transcript levels were specifically induced after inoculation with avirulent $P. triticina$ isolates, but were very low or undetectable prior to inoculation or after inoculation with a virulent isolate. The ANK domain of the $Lr14a$ protein shows structural similarities to transient receptor potential (TRP) ion channels and transcriptomic analyses revealed an induction of genes associated with calcium ion binding when $Lr14a$ was present (23). Similar to $Lr14a$, the Arabidopsis gene $ACCELERATED CELL DEATH 6$ ($ACD6$), a regulator of growth and
disease resistance, encodes an ANK-TM protein that might function as a calcium channel linked to enhanced calcium signaling (24, 25). Interestingly, the phenotypic expression of Lr14a is environmental and genotype dependent and can range from full resistance in some wheat cultivars to full susceptibility in others. This phenotypic variation has been associated with the segregation of genetic modifiers of Lr14a (23, 26). In Arabidopsis, two small peptide-encoding genes, MODULATOR OF HYPERACTIVE ACD6 1 (MHA1) and MHA1-LIKE, modulate the immune response of specific ACD6 variants. MHA1 and MHA1-LIKE can both bind to the ACD6 ankyrin repeats and may modulate its function (25). It is possible that the phenotypic expression of the Lr14a-mediated leaf rust resistance is modulated in a similar way. Genetic background effects, as observed for Lr14a and ACD6, are frequently observed in disease resistance breeding (27) and might in general be explained by the segregation of genetic suppressors and modifiers. The molecular basis of disease resistance modifiers, however, is only poorly studied and more research is required in order to understand the underlying molecular mechanisms and to fine-tune disease resistance in the future. In summary, Lr14a might function as a regulator of calcium fluxes that is transcriptionally induced by avirulent P. triticina isolates, possibly by the binding of an effector protein to the Lr14a promoter region.

The barley leaf rust resistance gene Reaction to Puccinia hordei 3 (Rph3) encodes a protein with multiple transmembrane helices. Similar to Lr14a, the expression of Rph3 was induced only by avirulent P. hordei isolates (28). The specific induction of Lr14a and Rph3 transcript levels by avirulent pathogen isolates is reminiscent of transcriptional activator-like (TAL) effector dependent R genes, also referred to as ‘executor R genes’, including the bacterial blight resistance genes Xa7, Xa10, Xa23 and Xa27 from rice (29). The promoter regions of these executor genes contain effector-binding elements that are targeted by TAL effectors. These promoters act as decoys and activate an immune response in the presence of specific bacterial blight effector proteins (30). Similarly, the induction of Lr14a and Rph3 by avirulent pathogen isolates might indicate that they also act as executor genes.
The race-specific wheat powdery mildew resistance gene *Pm4b* encodes a chimeric protein consisting of an N-terminal kinase domain followed by C2-domains and transmembrane regions (31). This unique domain architecture is exclusive to Triticeae and the stepwise evolution of *Pm4b* involved multiple gene fusion, duplication, and deletion events. Interestingly, *Pm4b* encodes two protein isoforms with different domain architectures that are the result of alternative splicing. The two isoforms form a complex in the endoplasmic reticulum (31).

So far, *Lr14a, Rph3* and *Pm4b* represent unique examples, meaning that no other *Lr14a, Rph3* or *Pm4b* homologs have been shown to function as *R* genes in Triticeae. Future research needs to establish if these genes are unique ‘exceptions’ or if other members of the same gene families are involved in plant immunity.

In contrast to *Lr14a, Rph3* and *Pm4b*, recent research in Triticeae has identified multiple examples of *R* genes encoding tandem kinase proteins (TKP). So far, six TKP encoding *R* genes have been cloned and functionally validated in Triticeae; the barley stem rust resistance gene *Rpg1*, the stripe rust resistance gene *Yr15 (WTK1)*, the powdery mildew resistance genes *Pm24 (WTK3)* and *WTK4* and the stem rust resistance genes *Sr60 (WTK2)* and *Sr62 (WTK5)* (Table 1) (32-37). Except *Rpg1*, all other TKP encoding genes have been cloned from cultivated wheat or wild wheat relatives (Table 1). Based on conserved catalytic amino acid residues, the kinase domains can be classified as kinases or pseudokinases and the domain architecture in TKPs includes ‘pseudokinase-kinase’, ‘kinase-pseudokinase’, and ‘kinase-kinase’ (38). Mutagenesis experiments have established that both domains are required for the disease resistance (33, 35, 37). Phylogenetic comparisons showed that TKPs evolved both as the result of gene duplications and gene fusions (37).

There are two intriguing questions when considering the recent discoveries on race-specific unconventional *R* gene: 1) Is the resistance conferred by such genes more durable compared to NLR-mediated disease resistance and 2) are the molecular mechanisms of pathogen perception and signaling fundamentally different from RLK and NLR-mediated immunity. Although the resistance conferred by the TKP-encoding genes *Rpg1* and *Yr15* is remarkably broad-spectrum (*Rpg1* has been described as durable for a long time),
there are a few stem and stripe rust isolates that are virulent on Rpg1 and Yr15, respectively (11). Virulent pathogen races have also been described for Sr60 and Sr62 (34, 39), indicated that TKP encoding genes confer race-specific resistance. Similar to the TKP encoding genes, virulence is found for Lr14a, Rph3 and Pm4b (23, 28, 31). Today, there is no evidence that the resistance conferred by race-specific unconventional R genes is more durable compared to NLR encoding genes. The resistance conferred by the NLR encoding wheat leaf rust resistance gene Lr22a, for example, has also been associated with a remarkable broad-spectrum specificity against most P. triticina isolates (18). The molecular function of race-specific unconventional R genes is not well understood and needs to be an area of intensive research in the near future. It has been hypothesized that pseudokinase domains in TKP might serve as decoys that directly perceive pathogen-derived effectors. The active kinase domain might then induce a downstream signaling cascade (38). Whether or not unconventional R proteins require RLKs or NLRs for perception or signaling is not yet established.

In summary, the discoveries on race-specific unconventional R genes represent an exciting new avenue for basic research and disease resistance breeding. However, a much more detailed understanding of the underlying molecular mechanisms will be required to make informed decisions on how to best utilize such genes in breeding programs.

Unconventional R genes: broad-spectrum resistance

The Triticeae gene pool contains several R genes that are classified as race non-specific, broad-spectrum or durable (Figure 1; Table 2). Phenotypically, the effect of these genes is very distinct from race-specific R genes and they often confer partial resistance that does not involve a strong hypersensitive response. Two remarkable examples are the durable, broad-spectrum resistance genes Lr34/Yr18/Sr57/Pm38 and Lr67/Yr46/Sr55/Pm46 (subsequently referred to as Lr34 and Lr67, respectively) of wheat. These two genes confer partial, race non-specific resistance against multiple biotrophic fungal pathogens, including the three
wheat rusts and powdery mildew. \textit{Lr34} has been extensively used in wheat breeding programs over the past 100 years and no adaptation in pathogen virulence has been observed. \textit{Lr34} encodes an ATP-binding cassette (ABC) transporter, which is a family of membrane-localized transporter proteins known to shuttle various substrates across membranes (40). The plant hormone abscisic acid (ABA) has been identified as a substrate of \textit{Lr34}, which is in line with the observation that the presence of \textit{Lr34} alters multiple ABA-related processes (41). \textit{Lr67} encodes a hexose transporter that might alter sugar transport in infected leaves (42). For both genes, only one resistance-conferring variant is known so far (\textit{Lr34res} and \textit{Lr67res}, respectively) that evolved after domestication by acquiring one or two critical amino acid changes (42, 43). In the case of \textit{Lr34res}, the deletion of one amino acid in a predicted transmembrane helix is associated with higher protein abundance of \textit{Lr34res} compared to the susceptible version \textit{Lr34sus}, which likely activates a constitutive ABA response (41). \textit{Lr34res} and \textit{Lr67res} can be functionally transferred into other cereal species where they provide resistance against a wide range of biotrophic and hemibiotrophic fungal pathogens (44-49). Together, these results show that \textit{Lr34res} and \textit{Lr67res} represent recent evolutionary inventions that alter the fluxes and distribution of two fundamental biological molecules, ABA and hexose. These two genes can thus not be placed in the typical models of plant-pathogen interactions (22, 50). Instead, these genes might result in a more hostile environment for pathogens, which might slow down their growth rates. Such constitutive physiological changes are much more durable because they cannot be overcome by a single loss-of-function mutation in a pathogen effector.

The wheat stripe rust resistance gene \textit{Yr36} confers broad-spectrum resistance against a wide range of \textit{P. striiformis} f. sp. \textit{tritici} isolates and encodes a chimeric protein with a kinase and a steroidogenic acute regulatory protein-related lipid-transfer (START) domain (=Wheat Kinase START 1; WKS1) (51). After stripe rust inoculation, WKS1 moves to the chloroplast where it binds and phosphorylates a thylakoid-associated ascorbate peroxidase (tPAX) (52), a major hydrogen peroxide scavenging enzyme. Phosphorylation of tPAX reduces its ability to detoxify peroxide and results in the gradual accumulation of reactive oxygen species (ROS) (52). In addition, WKS1 phosphorylates components of the photosystem II
and reduces photosynthetic activities, which further contributes to the accumulation of ROS (53). ROS bursts are a critical steps in RLK and NLR-mediated disease resistance signaling and usually occur in a matter of minutes after pathogen perception (54). In the case of Yr36, however, ROS accumulation and subsequent cell death seems to be much slower and requires several days (52).

Most of the cloned disease resistance genes in Triticeae confer resistance against foliar, biotrophic fungal pathogens, including rust and powdery mildew conferring fungi. Fusarium Head Blight (FHB), caused by the fungus *Fusarium graminearum*, is a destructive disease that mainly attacks wheat spikes. *F. graminearum* produces mycotoxins, which represent a concern for human and animal consumption. In contrast to rusts and powdery mildew, resistance to FHB is mostly quantitative. The quantitative resistance locus *Fhb1*, originally identified in the Chinese wheat cultivar Sumai3, represents the most widely used FHB resistance and the cloning of *Fhb1* has thus been the focus of multiple research groups. Interestingly, two neighboring genes on chromosome arm 3BS were reported to be *Fhb1*. A first study identified a pore forming toxin-like (PFT) encoding gene as *Fhb1*. The PFT protein was not associated with mycotoxin detoxification and it was hypothesized that PFT slows down fungal growth by associating with the fungal cell wall (55). Interestingly, two follow-up studies identified a neighboring gene, encoding a histidine-rich calcium binding protein (HRC) as *Fhb1* (56, 57). It is possible that both genes contribute to the quantitative resistance conferred by the *Fhb1* locus (58). Further research will be required to fully unravel the genetic and molecular mechanisms of *Fhb1*. The quantitative FHB resistance locus *Fhb7* was introduced into hexaploid bread wheat from the wild wheat progenitor *Thinopyrum elongatum*. A reference assembly-guided gene cloning strategy identified a gene encoding a glutathione-S-transferase (GST) as *Fhb7* (59). This GST protein detoxifies mycotoxins, which results in broad-spectrum FHB resistance. Interestingly, *Fhb7* showed strong homology to GST encoding genes from *Epichloë*, a fungal genus that forms endophytic symbioses with many grass species, and was most likely introduced into *Th. elongatum* through horizontal gene transfer (59). So far, no RLK or NLR encoding gene has been identified for FHB resistance and it is
interesting that FHB resistance seems to involve molecular pathways that do not fall into the classical models of plant-pathogen interactions.

A note on durable disease resistance

This review summarizes our current knowledge on unconventional $R$ genes in Triticeae. The most critical question for disease resistance breeding and $R$ gene deployment is the durability of $R$ genes and $R$ gene combinations. The classical definition of durable disease resistance states that it remains effective during prolonged and widespread use in an environment favorable to the disease (20). The major drawback of this definition is that the durability of a disease resistance can only be assessed after a crop cultivar has been grown for many years. It would be a huge advantage for disease resistance breeding if we were able to predict the durability of $R$ gene combinations without the need for first testing them for multiple years. The recent cloning of multiple $R$ genes from wheat cultivars with durable disease resistance has provided first insights into ideal gene combinations. A first example is the Swiss wheat cultivar Forno that was released in the 1980s and that continues to show high levels of leaf rust resistance against all $P. triticina$ isolates tested so far. The leaf rust resistance in Forno is quantitative and inherited by several loci (60). Two of the genes that contribute to the durable leaf rust resistance have been cloned. In addition to the broad-spectrum $Lr34$ gene, Forno also contains the race-specific unconventional $R$ gene $Lr14a$. Similarly, the South African wheat cultivar Kariega (released in 1993) shows durable stripe rust resistance that is the result of a combination of three major QTL. Among them are $Lr34$ ($Yr18$) and the race-specific stripe rust resistance gene $Yr27$, encoding a typical NLR (61). It is noteworthy that the race-specific genes $Lr14a$ and $Yr27$ contribute to the broad-spectrum and durable disease resistances in Forno and Kariega. These two examples indicate that durable resistance might be the result of optimal stacks of partial broad-spectrum resistance genes (like $Lr34$) and race-specific $R$ genes. In the case of Forno, the two cloned components $Lr34$ and $Lr14a$ are involved in different molecular processes, the modulation of ABA and calcium fluxes, respectively, which might further contribute to the durability of the leaf rust resistance in Forno (Figure 1).
With the cloning of additional disease resistance genes in the near future, we will gain a much better understanding of the $R$ genes contributing to durable disease resistance. This knowledge will be of fundamental importance to deploy optimal $R$ gene combinations, for example through the design of gene cassettes. A proof-of-concept study recently demonstrated that the use of $R$ gene cassettes represents a suitable strategy to engineer disease resistant wheat plants (62). The recent approval of the first genetically modified wheat in Argentina and Brazil (63) brings the concept of $R$ gene cassettes a step closer to application.

**Summary points**

- Unconventional $R$ genes confer both race-specific and broad-spectrum disease resistance.
- Unconventional $R$ proteins show unique domain architectures, including ankyrin-transmembrane (ANK-TM), predicted membrane protein, multiple C2-domains and transmembrane region kinase (MCTP-kinase), tandem kinase proteins (TKP), and transporter proteins.
- Optimal stacks of broad-spectrum unconventional $R$ genes with race-specific genes might result in durable disease resistance.

**Competing interests**

The authors declare that there are no competing interests associated with the manuscript.

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Author contribution

NA, LA, YW, and SGK wrote the review article.

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Figure legends

Figure 1. Schematic overview summarizing our current understanding of unconventional R proteins in Triticeae.

Numbers 1 to 4 summarize race-specific protein families (orange font color) while 5 to 9 refer to broad-spectrum resistance proteins (blue font color). (1) Tandem kinase protein (TKP) consists of two kinase domains, of which one is often classified as pseudokinase. TKP might act as decoys and directly perceive pathogen-derived virulence effectors. Alternatively, TKP-mediated resistance might involve NLRs. TKPs might for example be guarded by NLR immune receptors. (2) The executor like-transmembrane protein Rph3 consists of multiple transmembrane helices. Rph3 transcription is specifically induced by avirulent P. hordei isolates. (3) The ankyrin transmembrane protein Lr14a contains an N-terminal domain with twelve ankyrin repeats (ANK) and six transmembrane helices (TM) at the C-terminus. Lr14a might be involved in modulating calcium fluxes. Like Rph3, Lr14a is induced only by avirulent pathogen, which is reminiscent transcriptional activator-like (TAL) effector dependent R genes. (4) Pm4b consists of a kinase domain, multiple C2 domains and a transmembrane region. It encodes two isoforms with different domain compositions. The two isoforms form heteromers in the endoplasmic reticulum (ER). (5) Wheat Kinase START (Yr36) protein is a chimeric protein with a kinase and a START domain. Upon rust infection, WKS1 migrates to chloroplast to phosphorylate the thylakoid associated ascorbate peroxidase (tPAX) and the components of photosystem II (PSII), which results in the accumulation of reactive oxygen species (ROS). (6) The ABC transporter encoded by the Lr34 multi-pathogen resistance gene is a membrane-localized transporter protein modulating abscisic acid (ABA) fluxes. (7) The hexose transporter encoded by Lr67 might block the H+/hexose symport in infected leaves. (8) The glutathione S-transferase Fhb7 is an enzyme that detoxifies the Fusarium mycotoxins trichothecenes by conjugating the GSH unit. Solid arrows indicate experimentally validated pathways. Dotted arrows indicate the hypothesis proposed in the corresponding literatures.
Table 1. Race-specific unconventional *R* genes in Triticeae.

<table>
<thead>
<tr>
<th>Species</th>
<th>Gene</th>
<th>Domain architecture</th>
<th>Pathogen</th>
<th>Reference</th>
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<tr>
<td><em>Triticum aestivum</em></td>
<td>Lr14a</td>
<td>Ankyrin-transmembrane</td>
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<td>Tandem kinase protein</td>
<td>Bgt</td>
<td>(35)</td>
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<tr>
<td><em>Triticum cartlicum</em></td>
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<td>Serine/threonine kinase and multiple C2 domains and transmembrane regions</td>
<td>Bgt</td>
<td>(31)</td>
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<td>Pgt</td>
<td>(32)</td>
</tr>
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<td>Predicted membrane protein</td>
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<td>(34)</td>
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<td>(36)</td>
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<td><em>Triticum dicoccoides</em></td>
<td>Yr15</td>
<td>Tandem kinase protein</td>
<td>Pst</td>
<td>(33)</td>
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</tbody>
</table>

Pt = *Puccinia triticina* (wheat leaf rust); Pgt = *Puccinia graminis* f.sp. tritici (wheat stem rust); Ph = *Puccinia hordei* (barley leaf rust);
Pst = *Puccinia striiformis* f.sp. tritici (wheat stripe rust); Bgt = *Blumeria graminis* f.sp. tritici (wheat powdery mildew).
Table 2. Broad-spectrum unconventional \( R \) genes in Triticeae.

<table>
<thead>
<tr>
<th>Species</th>
<th>Gene</th>
<th>Domain architecture</th>
<th>Pathogen</th>
<th>References</th>
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</thead>
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<td>( Fg )</td>
<td>(55-57)</td>
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<tr>
<td>Thinopyrum elongatum</td>
<td>Fhb7</td>
<td>Glutathione S-transferase</td>
<td>( Fg )</td>
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<td>Triticum aestivum</td>
<td>Lr34/Yr18/Sr57/Pm38</td>
<td>ATP-binding cassette (ABC) transporter</td>
<td>Pt/ Pst/Pgt/Bgt</td>
<td>(40)</td>
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<td>(42)</td>
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<td>Yr36</td>
<td>START Kinase</td>
<td>( Pst )</td>
<td>(51)</td>
</tr>
</tbody>
</table>

\( Fg = \) Fusarium graminearum (wheat Fusarium head blight); \( Pt = \) Puccinia triticina (wheat leaf rust); \( Pgt = \) Puccinia graminis f.sp. tritici (wheat stem rust); \( Pst = \) Puccinia striiformis f.sp. tritici (wheat stripe rust); \( Bgt = \) Blumeria graminis f.sp. tritici (wheat powdery mildew).


