The evolving battle between yellow rust and wheat: implications for global food security

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Abstract
Wheat (Triticum aestivum L.) is a global commodity, and its production is a key component underpinning worldwide food security. Yellow rust, also known as stripe rust, is a wheat disease caused by the fungus Puccinia striiformis Westend f. sp. tritici (Pst), and results in yield losses in most wheat growing areas. Recently, the rapid global spread of genetically diverse sexually derived Pst races, which have now largely replaced the previous clonally propagated slowly evolving endemic populations, has resulted in further challenges for the protection of global wheat yields. However, advances in the application of genomics approaches, in both the host and pathogen, combined with classical genetic approaches, pathogen and disease monitoring, provide resources to help increase the rate of genetic gain for yellow rust resistance via wheat breeding while reducing the carbon footprint of the crop. Here we review key elements in the evolving battle between the pathogen and host, with a focus on solutions to help protect future wheat production from this globally important disease.

Abbreviations
APR Adult plant resistance
DMIs Demethylation inhibitors
GWAS Genome-wide association scan
MAS Marker assisted selection
NLR Nucleotide-binding leucine-rich repeat
Pst Puccinia striiformis Westend f. sp. tritici
QTL Quantitative trait locus
QoIs Quinone outside inhibitors
SDHIs Succinate dehydrogenase inhibitors
YR Yellow rust

Wheat (Triticum aestivum L.) is one of the most important staple crops, with global demand predicted to increase to 324 kg/year (per capita) by 2050 (Alexandratos and Bruinsma 2012). Wheat production faces numerous threats, with 10–16% of global wheat harvests estimated to be lost due to pests and diseases (Oerke 2006; Strange and Scott 2005). Yellow rust (YR), also known as stripe rust, is a major disease of wheat caused by the biotrophic fungal pathogen, Puccinia striiformis Westend f. sp. tritici (Pst). YR infection is most commonly noted on wheat leaves, where the resulting damage to photosynthetic tissues leads to reduced light interception and radiation use efficiency, thus lowering yields (Fig. 1a–b). However, YR infection can also take place on the structures of the wheat ear such as the glumes, lemma and palea, particularly during moderate to severe epidemics, resulting in reduced grain yield and quality (Bouvet et al. 2021a; Cromey 1989; Wellings, 2003; 2009) (Fig. 1c). Recurrent Pst epidemics have occurred in the majority of wheat growing areas over the past 60 years and can cause significant yield losses and reductions in grain quality if not adequately controlled (Wellings 2011). Notably, the past two decades have seen the rapid global emergence of more aggressive and genetically diverse Pst populations adapted to warmer temperatures (Hovmøller et al. 2016; Hubbard et al. 2015; Milus et al. 2009), with concomitant impact on the YR resistance ratings of many wheat varieties. YR resistance breeding targets have had to adapt to tackle the rapidly changing Pst threat, and sources of genetic resistance for the development of improved wheat varieties are continually being sought. This is now being aided by advances in wheat genomics approaches, as well
The complex pathogen lifecycle

*Pst* goes through five different spore stages and requires two plant host species for completion of its lifecycle (Fig. 1d). The two broad stages of the *Pst* lifecycle are classified as: (i) the asexual stage, which occurs on wheat (the primary host), and (ii) the sexual stage which occurs on *Berberis* species (the alternate host). In wheat, YR disease occurs during the asexual stage of the *Pst* lifecycle and is caused by multiple cycles of dikaryotic (i.e. two nuclei in each cell: n + n) *Pst* urediniospores re-infecting the primary host via wind dispersal. During the initial stage of wheat infection, urediniospores germinate on the leaf surface and eventually form an appressorium from which hyphae develop and enter the leaf tissue via the stomata. Growing hyphae develop into a dense network extending between and inside host mesophyll cells. Among this network, haustoria infection structures will form and specifically develop in host cell walls to extract nutrients (Szabo and Bushnell 2001). On the leaves of mature susceptible plants, disease symptoms are visible 12–14 days after infection, consisting of yellow to orange coloured urediniospores that erupt from pustules arranged in characteristic stripes that follow the veins down the leaf blade (Chen et al. 2014), which can lead to successive rounds of secondary infections. On resistant to mildly susceptible varieties, symptoms will range from non-sporulating flecks (a sign of hypersensitivity) to necrotic and chlorotic patches with no to limited sporulation. Towards the end of the wheat growth season, diploid teliospores may be produced by some isolates via karyogamy. These readily germinate to produce a promycelium of four cells, with meiosis subsequently resulting in a single haploid nucleus that forms a basidiospore able to infect the alternate host (Chen et al. 2014).

Although much less is known about the sexual stage, *Berberis* species were long speculated to support the *Pst* lifecycle (e.g. Straib 1937; Hart and Becker 1934), as well as the related rust species, *Puccinia graminis* f. sp. *tritici* (causal agent of stem rust). Historically this resulted in efforts to eradicate *Berberis* species in many European and North American countries (Barnes et al. 2020). However, it was not until 2010 that *Berberis* species were formally confirmed to support development of *Pst* pycnia and aecia (Jin et al. 2010). Curiously, *Berberis* species infected with *Pst* are rarely observed in the wild (Zhao et al. 2011, 2013). This may be due to the difficulty in finding an environment that simultaneously accommodates germination of teliospores (part of the asexual stage; enclosed in telia that form on wheat leaves towards the end of the infection season and produce basidiospores) and basidiospores (part of the sexual stage; forming on barberry leaves and requiring dew for germination), both of which have short viability (Wang and Chen 2015). A recent study showed *Berberis* species do not play a role in YR epidemics in the US Pacific Northwest (Wang and Chen 2015), and an additional alternate host, *Mahonia aquifolium* (Oregon grape) has been identified (Wang and Chen 2013). The main importance of the sexual *Pst* stage to wheat infection is the generation of novel combinations of standing genetic variation, resulting in new genetically recombined isolates that can cause widespread epidemics and rapid changes in wheat resistance profiles.
Pathogens on the move: patterns of Pst dispersal and the rise of divergent lineages and aggressive races

Over the years, monitoring of virulence changes in Pst populations in the major wheat producing regions has revealed notable changes in pathogen movement and adaptation. These studies were based on pathogenicity surveys, which use sets of differential wheat lines carrying known resistance genes, either near isogenic lines or cultivars, for the characterisation of pathotypes at the seedling stage (Wellings et al. 2009). More recently, molecular and genomics techniques have been used to infer Pst population structure and genetic diversity, confirming patterns of adaptation hypothesised in pathotype-based approaches. Here, we summarise key findings and events from the past three decades, specifically focusing on patterns of spore dispersal and Pst evolution and adaptation.

Blowing in the wind

Pst urediniospores are windborne and can disperse at continental scales. Coupled with the obligate nature of the pathogen (requiring living tissue to survive), this has led to different scenarios for the observed seasonal and geographic patterns of dispersal. One such model is the local extinction and re-colonisation model, illustrated for example in China where regions of the Sichuan and Gansu provinces in which Pst prevails all year round act as a source of inoculum to the more northerly provinces in which wheat is predominantly grown as a winter crop (Brown and Hovmøller 2002; Zeng and Luo 2006). In this way, Pst populations re-establish at the beginning of each wheat cropping season in those regions where Pst spores are usually unable to over-winter. A similar pattern of spore movement according to prevailing winds and the seasonality of the cropping seasons has been speculated in pathotype-based approaches. For example in China where regions of the Sichuan and Gansu provinces in which Pst prevails all year round act as a source of inoculum to the more northerly provinces in which wheat is predominantly grown as a winter crop (Brown and Hovmøller 2002; Zeng and Luo 2006). In this way, Pst populations re-establish at the beginning of each wheat cropping season in those regions where Pst spores are usually unable to over-winter. A similar pattern of spore movement according to prevailing winds and the seasonality of the cropping seasons has been speculated in pathotype-based approaches.

Pathogen evolution and adaptation

Prior to 2000, pathogenicity surveys and molecular studies using isolates collected across the main wheat-producing regions in Europe, Australia and America typically reported Pst populations were clonal in nature, and that pathotypes exhibited close-relatedness and low genetic variation—predominantly underpinned by single step-wise mutations (Hovmøller et al. 2002, 2016; Enjalbert et al. 2005; Chen 2005; Steele et al. 2001; Chen et al. 2010; Ali et al. 2014a; Hubbard et al. 2015). Such clonally derived Pst mutations have caused several severe YR epidemics, due to the ‘breakdown’ of specific wheat Yr resistance genes present in large acreages across the agricultural landscape. Notable examples include breakdown of Yr17 in Northern Europe (Bayles et al. 2000), Yr27 in Ethiopia (Solh et al. 2012), and Yr9 in America, the Middle East and the Indian sub-continent (Chen et al. 2010; Singh et al. 2004). Before the year 2000, the only exceptions to such patterns of low Pst genetic variation were observed in isolates from the Himalayan (Nepal and Pakistan) and near Himalayan (China) regions, which exhibited high levels of genetic recombination, high ability for sexual reproduction and high genetic diversity (Duan et al. 2010; Mboup et al. 2009; Ali et al. 2014b). These areas were therefore classified the putative centres of Pst origin (Ali et al. 2014b). However, the last two decades have seen the emergence of unusual virulence profiles and aggressive strains across the world. The most noteworthy event was the rise of two strains, PstS1 and PstS2, across the USA (Chen et al. 2002; Markell and Milus 2008), Europe (Hovmøller and Justesen 2007) and Australia (Wellings 2007) in the space of just three years in the early 2000s. A global study of pre- and post-2000 Pst races combining detailed virulence pathotyping and DNA fingerprinting found that while these while these two strains were genetically similar to each other, they were highly divergent from previous races in their respective geographic regions (Hovmøller et al. 2008).
their increased aggressiveness (ability to yield more spores and for disease symptoms to occur more quickly) and high-temperature adaptation—which was later demonstrated in the detailed study by Milus et al. (2009). In addition to \( \text{PstS1} \) and \( \text{PstS2} \), additional atypical occurrences of \( \text{Pst} \) races have since been reported. Enjalbert et al. (2005) demonstrated high levels of genetic divergence between the \( \text{Pst} \) population in northern France and a single clone specific to the South. What was atypical was that this single pathotype was maintained for a long time in this region, despite the presence of gene flow between Northern and Southern \( \text{Pst} \) populations. This isolate was later found to be more closely related to the Central Asian-Mediterranean population (Ali et al. 2014a). Similarly, instances of strong genetic divergence have also been revealed in North Western Europe (Flath and Barthels 2002; Hovmøller and Justesen 2007a). Two groups of highly divergent pathotypes from the ‘old’ North-Western European population exhibited three to four times higher levels of genetic diversity (Hovmøller et al. 2007). In 2011, two novel \( \text{Pst} \) races disrupted the European \( \text{Pst} \) landscape (www.wheatrust.org). Named after the host varieties on which they were first detected, one race was virulent on wheat cv. ‘Warrior’ and the other was virulent on cv. ‘Kranich’. These were later characterised as \( \text{PstS7} \) and \( \text{PstS8} \), respectively (Ali et al. 2017), and were detected simultaneously across Europe and infected varieties that had exhibited durable adult plant resistance. Both races were distinct from the typical European isolates in that they produced an unusually high number of teliospores (Hubbard et al. 2015; Hovmøller et al. 2016). Additional \( \text{Pst} \) races have been characterised (\( \text{PstS10} \) also known as ‘Warrior (-)’, \( \text{PstS4} \) ‘Triticale aggressive’) and together with the other new genetically diverse \( \text{Pst} \) races, have come to largely dominate within Europe (Ali et al. 2017; Hovmøller et al. 2016; Hubbard et al. 2015). Collectively, these atypical observations, further supported by genetic diversity studies, have led to speculation of an aerial-induced foreign incursion, which would be the first of its kind in Europe since the establishment of \( \text{Pst} \) in Europe during the nineteenth century. Beyond Europe, rapid invasions and the subsequent \( \text{Pst} \) population changes have been responsible for a number of \( \text{YR} \) epidemics in Central Asia, North and East Africa (Ali et al. 2017).

### Chemical control of yellow rust

Review of global \( \text{YR} \) epidemics shows most wheat-growing regions document recurrent crop losses of 5–10%, with occasional losses of up to 25% (Welling 2011). However, following the global spread of aggressive \( \text{Pst} \) races since 2000, surveys highlight an increase in both the number of countries being significantly hit by such races, and the extent of the losses incurred (Beddow et al. 2015). Indeed, the financial implications of this change in \( \text{Pst} \) race structure estimated that a global average of US$ 158 million was lost annually pre 2000s, compared to US$ 979 million post 2000 (Beddow et al. 2015). Wheat growers have two principal options to protect against the effects of \( \text{YR} \) on yield: (i) protect their crop with agro-chemicals that limit initial infection and progression of pathogen colonisation, and/or (ii) grow wheat varieties with adequate levels of genetic resistance. Systemic fungicides that are absorbed into the plant became commercially available in the 1980s and have since formed an important part of integrated control measures against \( \text{YR} \) (Chen 2005). Several products with different modes of action are available for protection against \( \text{YR} \) (reviewed by Chen and Kang 2017), with timely application a key aspect of an effective fungicide programme. Such an approach has, for example, prevented significant financial losses in periods of severe epidemics in the USA (Line 2002). While fungicide control provides an essential tool in combatting sudden \( \text{YR} \) epidemics and in situations where growing resistant varieties is not an option, over-dependence on their use comes with negative environmental impacts and notable financial cost to growers. For example, in Australia an estimated A$ 359 million per year is spent on fungicides for \( \text{YR} \) control (Murray and Brennan 2009). In the mid-to-long term, regular \( \text{Pst} \) exposure to fungicides also increases the risk that \( \text{Pst} \) populations develop resistance to frequently used chemistries. Historically, \( \text{Pst} \) has been classified as being at low-risk of developing fungicide resistance. However, of the three classes of fungicides active against \( \text{Pst} \) (demethylation inhibitors, DMIs; succinate dehydrogenase inhibitors, SDHIs; quinone outside inhibitors, QoIs), \( \text{Pst} \) resistance has evolved against two. Low levels of DHI resistance have been reported, and while high proportions of isolates carrying resistance associated mutations have been reported in some countries (Cook et al. 2021), DHI resistance has so far had limited agronomic-scale significance (Oliver 2014). SDHIs active against rusts have only been introduced relatively recently, giving less time for \( \text{Pst} \) resistance to evolve. Nevertheless, sets of geographically diverse isolates have been identified that carry a mutation homologous to that linked to SDHI resistance in the related rust species \( \text{P. pachyrhizi} \) (Cook et al. 2021). In the face of additional considerations such as changing regulation surrounding permissible chemistries, such evidence has led to the suggestion that the \( \text{Pst} \) risk classification should be upgraded (Oliver 2014), fungicide resistance management practices be considered, and that systematic monitoring for \( \text{Pst} \) fungicide resistance should be implemented (Cook et al. 2021). Lastly, the optimisation of fungicide timing, as well as improved fungicide application technologies, represents areas where additional research and development is required (Carmona et al. 2020).
Genetic control of yellow rust

More than 300 wheat genomic regions conferring YR resistance have been reported (Rosewarne et al. 2013; Wang and Chen 2017). Of these, ~80 are permanently named yellow rust resistance (Yr) genes (recently summarised by Jamil et al. 2020). Two main classes of YR resistance (R) genes are commonly described. The first is termed ‘all stage resistance’ (or ‘seedling resistance’) and confers qualitative resistance—typically to one or a low number of Pst isolates. The second is termed ‘adult plant resistance’ (APR) and confers quantitative or partial resistance. While these R gene classifications are useful, additional categories are also used, based on criteria such as phenotypic response (infection type, race specificity, resistance levels), temperature sensitivity, durability, the number of genes involved (monogenic versus polygenic) and the size of gene effect (Chen 2013). One of the issues that comes with defining YR resistance with such a broad range of criteria is the assumptions associated with each of them. For example, APR is typically non-race specific, more durable than seedling resistance and conditioned by genes with minor or partial effect. Nevertheless, some APR genes have been shown to exhibit race specificity, such as Yr11, Yr12, Yr13 and Yr14 (Johnson 1992; McIntosh et al. 1995).

All-stage resistance

Initially expressed at the seedling stage, all-stage resistance extends throughout the growth of the wheat plant and is characterised by a hypersensitive response. It is generally effective against some, but not all, Pst races and is therefore also referred to as ‘race-specific resistance’. All-stage resistance is underpinned by the gene-for-gene model in the pathogen, with resistance conferred by an incompatibility (Avr)-gene interaction. This results in a qualitative resistance phenotype that can be easily assessed, historically making it a popular selection criterion in breeding programmes, and more recently, for gene cloning. The majority of catalogued YR genes exhibit this type of phenotype, and many become ineffective against present-day Pst races. This type of resistance has commonly been shown to be a short-term strategy for YR control. Indeed, the deployment of varieties with single or low-numbers of all-stage resistance Yr genes over large acreages inevitably exerts high selective pressure on the pathogen, forcing it to evolve and mutate until host resistance is broken down, and leading to cycles of ‘boom and bust’ (McDonald and Linde 2002).

Adult plant resistance

Adult plant resistance (APR) is characterised by slow rusting (a long period of latent infection, small lesion size) (Guo et al. 2008) or partial resistance, typically manifests at the adult plant stage, and has long been established as a durable source of YR resistance. Two notable examples are Yr18/Lr34/Sr67/Pm38, extensively deployed in spring wheat cultivars through the international breeding programme at CIMMYT (Singh et al. 2005) and Yr16, an APR gene commonly used in early European varieties such as ‘Cappelle Desprez’, a major hub in the European wheat pedigree (Fradgley et al. 2019). While APR is primarily non-race specific, examples of APR specificity to Pst races do exist, such as Yr12 and Yr13 (Johnson 1992; McIntosh et al. 1995). Such APR race-specificity was initially reported by Johnson (1988) and has recently been observed in Europe following the spread of atypical Pst races (Sørensen et al. 2014). For example, while the APR resistance allele conferred by the founder Claire at the QTL QYr.niab-2D.1 was effective in the UK during the 2015 and 2016 seasons (Bouvet et al. 2021b), it has since broken down (Simon Berry, personal communication). Another example is that of Yr49, which was initially found to be non-race specific against all Australian Pst isolates, but when tested against Chinese races showed racespecificity (Ellis et al. 2014). These occurrences undermine the durability of APR and puts into question whether this pathotype criteria should be used to describe this type of resistance. It has been suggested that as some APR genes confer resistance against multiple biotrophic pathogens, this characteristic is a good indicator of durability. Examples include Yr18/Lr34/Sr67/Pm38 (Spilmeyer et al. 2005; Lillemo et al. 2008), Yr29/Lr46/Sr58/Pm39 (Lagudah, 2011), Yr30/Lr27/Sr2 (Mago et al. 2011) and Yr46/Lr67/Sr55/Pm46 (Herrera-Foessel et al. 2014). Interestingly, some of these genes are also associated with traits such as leaf tip necrosis (Yr18/Lr34/Sr67/Pm38, Singh et al. 1992; Yr29/Lr46/Sr58/Pm39, Rosewarne et al. 2006; Yr46/Lr67/Sr55/Pm46, Herrera-Foessel et al. 2014) and pseudo-black chaff (Yr30/Lr27/Sr2, Kota et al. 2006). Finally, some APR resistances are more effective at high temperature (usually 25–30 °C), and are termed High Temperature Adult Plant (HTAP) resistance. Yr36 was initially characterised as HTAP (Uauy et al. 2005), with subsequent studies showing resistance was effective over 25 °C at all growth stages (Fu et al. 2009), and that the lower effective temperature range is 18 °C (Bryant et al. 2014).

Cloned yellow rust resistance genes

Nucleotide Binding Sequence Leucine Rich Repeat (NBS-LRR) proteins are the most common class of proteins encoded by plant R genes, and act predominantly by recognising the effector molecules that pathogens produce to
inhibit host defence responses (Jones et al. 2016). To help fight against potential infecting pathogens, plant NLR gene families have radiated and diversified, for example via localised gene duplication or mutation within their LRR domains that bind pathogen effectors (Sarris et al. 2016). Furthermore, some NBS-LRRs contain additional ‘integrated’ domains, the most common of which are kinase and DNA-binding domains (Andersen et al. 2020; Steuernagel et al. 2020), and are thought to be involved in receptor activation or downstream signalling (Sarris et al. 2016). Furthermore, some NBS-LRRs contain additional ‘integrated’ domains, the most common of which are kinase and DNA-binding domains (Andersen et al. 2020; Steuernagel et al. 2020), and are thought to be involved in receptor activation or downstream signalling (Sarris et al. 2016). Of the 19 genes conferring all-stage resistance to wheat rusts (yellow rust, stem rust, leaf rust) that have been cloned, 17 encode NBS-LRRs (Table 1). Furthermore, all but two of these 17 NBS-LRRs contain coiled coil (CC) domains towards their N-termini; the exceptions being Yr7 and the allelic R genes Yr5/Yr6SP, each of which contains an N-terminus integrated BED zinc finger domain (Marchal et al. 2018) and Sr60, which is race-specific but confers a partial resistance phenotype and encodes a protein with two putative kinase domains (Chen et al. 2020). Finally, the broad-spectrum ASR gene Yr15 encodes a tandem kinase-pseudokinase protein (Klymiuk et al. 2018) similar to that encoded by the barley stem rust resistance gene Rpg1 (Brueggeman et al. 2002), and has recently been shown to be allelic with YrG303/YrH52 (Klymiuk et al. 2020).

The ongoing changes and rapid spread of Pst populations around the world has led to growing interest in more durable sources of resistance. To date, three adult plant YR resistance genes have been cloned. Yr36 encodes a protein with a kinase and a START lipid-binding domain (WHEAT KINASE START 1, WKS1; Fu et al. 2009), and is thought to regulate reactive oxygen species (ROS) via phosphorylation of the thylakoid ascorbate peroxidase protein, resulting in increased levels of ROS during immunity

### Table 1 Cloned wheat rust resistance (R) genes.

| Cloned YR resistance genes | Original source | Chr | R gene class | NCBI protein accession number | Gene functional annotation | Reference |
|----------------------------|-----------------|-----|--------------|--------------------------------|---------------------------|-----------|
| Lr1                        | T. aestivum     | 5D  | ASR          | ABS29034                       | CC-NBS-LRR                | Cloutier et al. (2007) |
| Lr10                       | T. aestivum     | 1A  | ASR          | AAQ01784                       | CC-NBS-LRR                | Feuillet et al. (2003) |
| Lr21                       | Ae. tauschii    | 1D  | ASR          | ACO53397                       | NBS-LRR                   | Huang et al. (2003)   |
| Lr22a                      | Ae. tauschii    | 2D  | ASR          | AR038244                       | CC-NBS-LRR                | Third et al. (2017)  |
| Sr13                       | T. turgidum ssp. durum | 6A  | ASR          | ATE88995                       | CC-NBS-LRR                | Zhang et al. (2017)  |
| Sr21                       | T. monococcum   | 1D  | ASR          | AVK42833                       | CC-NBS-LRR                | Chen et al. (2018)   |
| Sr22                       | T. monococcum   | 7A  | ASR          | CUM44200                       | CC-NBS-LRR                | Steuernagel et al. (2016) |
| Sr33                       | Ae. tauschii    | 1D  | ASR          | AGQ17384                       | CC-NBS-LRR                | Periyannan et al. (2013) |
| Sr35                       | T. monococcum   | 3A  | ASR          | AGP75918                       | CC-NBS-LRR                | Saintenac et al. (2013) |
| Sr45                       | Ae. tauschii    | 1D  | ASR          | CUM44213                       | CC-NBS-LRR                | Steuernagel et al. (2016) |
| Sr46                       | Ae. tauschii    | 2D  | ASR          | AYY61514                       | CC-NBS-LRR                | Arora et al. (2019)  |
| Sr50                       | Secale cereale  | 1R† | ASR          | ALO61074                       | CC-NBS-LRR                | Mago et al. (2015)   |
| Sr60                       | T. monococcum   | 5A  | ASR          | LRR12123                       | Tandem kinase              | Chen et al. (2020)   |
| SrTA1662                   | Ae. tauschii    | 1D  | ASR          | Not listed                     | CC-NBS-LRR                | Arora et al. (2019)  |
| YrAS2388                   | Ae. tauschii    | 4D  | ASR          | QDW65446                       | CC-NBS-LRR                | Zhang et al. (2019)  |
| Yr5/YrSP                   | T. spelta album | 2B  | ASR          | QEQ12705/QEQ12706             | BED-NBS-LRR               | Marchal et al. (2018) |
| Yr7                        | T. aestivum     | 2B  | ASR          | QEQ12704                       | BED-NBS-LRR               | Marchal et al. (2018) |
| Yr10†                      | T. aestivum     | 1B  | ASR          | AAG42168                       | CC-NBS-LRR                | Liu et al. (2014)   |
| Yr15/YrG303/YrH52          | T. turgidum ssp. dicoccoides | 1B  | ASR          | AXC33067                       | TKP                        | Klymiuk et al. (2018) |
| Yr18/Lr34                  | T. aestivum     | 7D  | APR          | ACN41354                       | ABC transporter            | Krattinger et al. (2009) |
| Yr36                       | T. turgidum ssp. dicoccoides | 6B  | APR          | ACF33187                       | Kinase-START               | Fu et al. (2009)     |
| Yr46/Lr67                  | T. aestivum     | 4D  | APR          | ALL26331                       | Hexose transporter         | Moore et al. (2015)  |

ASR all-stage resistance. APR adult plant resistance. Lr leaf rust, Sr stem rust, Yr yellow rust. TKP tandem kinase-pseudokinase. Chr. chromosome.

†In bread wheat, the Sr50 locus from rye has been translocated to chromosome 1D

*See also Yuan et al. (2018), who indicate the CC-NBS-LRR gene identified by Liu et al. (2014) may not be the underlying gene
(Gou et al. 2015). More recently, WKS1 has been shown to phosphorylate a protein component of photosystem II, sbO, resulting in reduced photosynthesis, leaf chlorosis and Pst resistance (Wang et al. 2019). Yr18/Lr34 encodes an ABC transporter (Krattinger et al. 2009) involved in the translocation of abscisic acid (Krattinger et al. 2019) while Yr46/Lr67 encodes a hexose transporter (Moore et al. 2015).

Designing yellow rust resistant wheat

Pyramiding multiple resistance genes with additive effects into single genetic backgrounds should help prevent dramatic breakdown of wheat Pst field resistance. This first iteration of resistance gene pyramiding was developed using conventional breeding techniques. Indeed, the CIMMYT wheat breeding programme has made extensive use of the ‘Yr18 complex’ (Yr18 and at least two to three additional slow-rusting genes), which has provided durable resistance against yellow rust (Singh et al. 2005). Tools to help such approaches are available. These include protocols for the use of diagnostic molecular markers for marker-assisted breeding for many of the cloned resistance genes listed above (https://maswheat.ucdavis.edu/), as well as ‘speed breeding’ methods that include the use of extended day lengths and controlled temperatures to shorten the wheat lifecycle (Watson et al. 2018). Indeed, knowledge of which resistance genes are present within breeders germplasm/released wheat varieties would help prioritise parental lines for future breeding efforts. However, combining numerous unlinked genes via crossing is time-consuming. For example, a recent crossing scheme for the incorporation of 12 resistance genes in a single recurrent background involved 20 generations (Hafeez et al. 2021). Additionally, sources of YR resistance commonly originate from species related to T. monococcum and Aegilops tauschii (T. monococcum and Aegilops tauschii) and wild or cultivated tetraploid wheats (T. turgidum ssp. dicoccoides and T. turgidum ssp. durum, respectively), resulting in introgression of linked chromosomal regions from the donor progenitor species. Such introgressed regions may have a negative effect on crop performance; for example, while Sr60 has recently been introduced into bread wheat via the introgression of a small T. monococcum segment containing the R gene, it nevertheless contains linked PUROINDOLINE genes which will affect grain texture (Chen et al. 2020). Furthermore, it can be challenging when crossing germplasm within conventional breeding programmes to maintain the desired resistance gene combinations in the progeny, as the loci are inherited independently. In practice therefore, the combinations of resistance genes deployed by breeders will also depend on the genetic architectures controlling many other agronomically important traits. This means that key resistance loci may be at risk of being used alone, leaving them exposed to be overcome by the pathogen. Such considerations mean development of resistance gene cassettes containing multiple R genes could provide a useful breeding tool, providing multiple sources of resistance inherited as a single genetic unit. Assuming their effects will be additive (i.e. show no epistasis), the three cloned APR genes, Yr18, Yr36 and Yr46, possibly combined with one or more ASR genes such as Yr15, represent obvious immediate targets. Indeed, a transgene cassette containing four stem rust ASR genes and one APR gene has recently been shown to confer broad-spectrum field resistance (Luo et al. 2021). However, such approaches do not come without their challenges: genetic modification regulations and consumer acceptance remains an important barrier in many parts of the world, relatively low numbers of Yr genes have been cloned, and further work is needed to determine how specific genes work in combination within the context of inbred lines and F1 hybrids. Towards tackling some of these issues, proposals to generate an R gene atlas for the major diseases of wheat have been made (Hafeez et al. 2021). Such concepts would be aided by the systematic identification and monitoring of the corresponding Pst effectors and their standing variation across the agricultural environment, and should be extended to identify, characterise and eliminate wheat susceptibility (S) genes that act to increase YR susceptibility (e.g. Corredor-Moreno et al. 2021). Underpinning such aims is the availability of new genomic techniques and resources in wheat that complement classical map-based cloning methodologies (recently reviewed by Adamski et al. 2020). For example, candidate gene association mapping using diversity panels of wheat or wheat relatives genotyped via reduced representation sequencing of classes of genes known a priori to be prevalent in disease resistance (such as NBS-LRRs or wall-associated kinases). This method, termed ‘RenSeq’ (Jupe et al. 2013), alongside functional validation via chemical mutagenesis of germplasm containing the functional allele of interest, has been used to identify the wheat stem rust resistance genes Sr46 and Sr1TA1662 (Arora et al. 2019). Such association mapping approaches can be extended to include more representative coverage across the genome, for example using promoter/exome capture arrays (Gardiner et al. 2019) or whole-genome sequencing at low-coverage combined with imputation of SNPs and haplotypes, aided by the use of reference genome assemblies (e.g. for bread wheat: IWGSC, 2018; Walkowiak et al. 2021). Furthermore, the availability of Pst genome assemblies (e.g. Cantu et al. 2011, 2013; Zheng et al. 2013; Schwessinger et al. 2018, 2020) and mutant populations (Li et al. 2020), as well as gene expression resources and interrogation tools for both species (e.g. Adams et al. 2021) should help identify and characterise pathogen effectors. Detailed knowledge of the specificity of the recognition interactions between wheat R...
genes and their corresponding Pst effectors could be used, for example, to monitor the functionality of each component of R stacks, and to design synthetic R genes engineered to recognise multiple races (as demonstrated for example by editing of the rice NBS-LRR gene PikP to recognise multiple variants of the effector AvrPik from the rice blast pathogen Magnaporthe oryzae; De La Concepcion et al. 2019). Similarly, identification of wheat S genes would allow their elimination, via marker assisted approaches, mutation breeding or gene editing. Finally, further understanding of the exact developmental stages at which different adult plant resistance genes become effective, how best to deploy these in the agricultural landscape to best protect the crop from infection throughout the key growth stages, and understand which R genes exhibit the lowest yield cost, will further help protect wheat against the effects of YR.

**Future perspectives**

The wide-ranging spread of new genetically diverse Pst races has meant that YR is likely to become an increasing threat to global wheat production, resulting in lower yields and increased financial and environmental costs. Here, we conclude with a series of bullet-point recommendations for future research and development in YR management over the next decade:

**Host genetics**

1. Systematic programmes to identify and clone known and novel R genes, particularly those conferring adult plant or non-host resistance.
2. Informed design and development of durable R gene pyramid combinations, via traditional crossing and/or R gene cassettes.
3. Identification and targeted removal of susceptibility (S) genes from breeders’ germplasm.

**Monitoring**

1. Regional and international networks to rapidly monitor the emergence and spread of Pst pathotypes.
2. Field networks to monitor R gene effectiveness at regional/international scales.

**Agronomy**

1. Regional monitoring for the emergence and spread of fungicide resistances.
2. Innovation in fungicide application technology and crop monitoring to allow more timely, accurate and efficient fungicide application.

Implementation of these recommendations will work best when national programmes are integrated or coordinated at a regional, or even global, level. Such coordination would require funding over timescales that go beyond what typically available for crop disease resistance research, and might best be best addressed by establishing regional coordination centres. Such networks would need to ensure fast and efficient data release and work closely with the crop breeding industry. Ultimately, the success of advances in integrated YR management approaches will depend on timely communication of information to wheat growers. Therefore, trusted grower-facing networks and sources of information that can rapidly and succinctly inform and advise farmers of threats and best practice within each growing season will become increasingly critical in realising future ambitions to better protect wheat yields from diseases such as YR.

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**Declarations**

**Conflict of interest** On behalf of all authors, the corresponding author states that there is no conflict of interest.

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