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Biotic stresses in the anthropogenic hybrid triticale (×*Triticosecale* Wittmack): current knowledge and breeding challenges

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Abstract Triticale (×*Triticosecale* Wittmack) is the intergeneric hybrid derived by crossing wheat (*Triticum* spp.) and rye (*Secale* spp.). Consequently, the same spectrum of fungal diseases occurring on the parent crops can impede optimal triticale production. With the expansion of the triticale growing area, the scientific interest into these fungal pathogens has gained momentum. This review considers the major fungal diseases occurring on triticale: powdery mildew, rust diseases, and *Fusarium* head blight and highlights breeding strategies or opportunities to control these pathogens. Although there are several models to explain the emergence of pathogens in newly introduced crops, for powdery mildew on triticale, it is accepted that it emerged through a host range expansion of wheat powdery mildew. Moreover, this host range expansion of wheat powdery mildew occurred recently, multiple times and at different locations in Europe. For rust diseases and *Fusarium,* evidence for such an abrupt host shift is rather thin and suggests an evolution in disease incidence and virulence confluent with evolving management practices, variable seasons, mutations, recombination and variety selection. In order to overcome these fungal pathogens in triticale, plant breeding is a powerful tool. Despite the multiple parallelisms between fungal diseases in triticale and wheat, the narrow genetic background, partially due to the narrow genetic background of the parental crops, is a serious issue in triticale breeding. It remains a challenge for future breeding strategies to broaden the genetic background of new varieties that are being developed, through introgression and deployment of new sources of disease resistance. Especially, quantitative and multi-pathogen sources of resistance have to be considered. In this way, triticale can retain its position as important low input farming cereal crop.

Keywords Triticale · Disease emergence · Powdery mildew · Rust · *Fusarium* head blight · Resistance breeding

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Introduction

Triticale (×*Triticosecale* Wittmack) is the intergeneric hybrid between the female parent wheat (*Triticum* spp.) and the male parent rye (*Secale* spp.). This artificial cereal combines the robustness (cold and disease tolerance and its adaptation

to unfavourable soils and climates) of rye with the productivity and nutritional qualities of wheat (Walker et al. 2011). Unfortunately, the good baking qualities of wheat were not inherited and therefore, triticale is mostly used as a feed source for poultry, pigs and ruminants (Feuillet et al. 2008; McGoverin et al. 2011). Nevertheless, ongoing research indicates that triticale has some potential for use in human consumption and remarkable improvement has been made on bread making quality during the last decades (Martinek et al. 2008). Besides its use in animal feed production, environmental awareness has aroused interest in the use of triticale within bio-fuel production (Pronyk and Mazza 2011).

Interestingly, triticale often out-yields wheat in unfavourable growing conditions (Haesaert and De Baets 1994; Bassu et al. 2011). Therefore, triticale is often grown in areas not suitable for wheat due to abiotic stresses such as drought and acid soils (Salmon et al. 2004). In acid soils, aluminum remains in a cationic form that is toxic to plants, reducing growth and yield. Research has demonstrated that triticale is moderately tolerant to aluminium toxicity compared to wheat (Niedziela et al. 2012; Kochian 1995). Suboptimal growing conditions as indicated above prevail in many parts of the world, and continue to gain importance due to climate change and soil degradation by unfavourable soil management and industrialization (Oettler 2005). Finally, triticale is suitable for low input farming because of its lower demands on nutrients and because it is endowed with a fairly high level of resistance towards pests and diseases. As the area sown to triticale has increased, with changes in management practices, changing seasons and varieties, the extent and spectrum of diseases found on triticale has been changing (Oettler 2005). Biotic stresses, in particular fungal pathogens, have become a serious concern in triticale production in recent years, impacting the quality and quantity of its yield.

This review focuses on the major fungal pathogens occurring on triticale. The disease emergence and spread of powdery mildew and the evolution of rust diseases and *Fusarium* head blight on triticale are described in detail. For general overviews of the biology of these pathogens, we refer to previous reviews (Parry et al. 1995; Bolton et al. 2008; Singh 2008; Hovmøller et al. 2011; Troch et al. 2014). Additionally, resources and challenges for disease control by resistance breeding in triticale are discussed.

Triticale production

The first systematic and commercial breeding of triticale began in the 1960s. Production has grown from less than 4 million tonnes in the late 1980s to more than 13 million tonnes in 2010 (FAOSTAT 2013). In the late 1980s commercial cultivation of triticale was mainly restricted to Poland with an annual production of 2.4 million tonnes or 60 % of the worldwide production. During the last decade, triticale has gained considerable importance throughout the world as its production area has expanded from 2.5 Mha in 2000 up to almost 4 Mha in 2012 (Fig. 1, FAOSTAT 2013). Current production of triticale is concentrated in Europe with more than 90 % of the world production (Fig. 2, FAOSTAT 2013). The top 5 leading producers of triticale in Europe are Poland (3.3 million tonnes), France (2.3 million tonnes), Germany (2.3 million tonnes), Lithuania (0.4 million tonnes) and Hungary (0.3 million tonnes). Other important producers on the European continent are Belarus (1.8 million tonnes), and Russia (0.5 million tonnes). Finally, in Oceania and South America, Australia (0.3 million tonnes) and Brazil (0.1 million tonnes) are the leading producers.

Major triticale diseases

The development of new crop species and their associated agro-ecosystems has an impact on the occurrence and spread of plant pathogens (Stukenbrock and McDonald 2008). The rather recent introduction of triticale in cropping systems and its subsequent increased growing area, along with the widespread use of genetically uniform varieties, provides an ideal case study for disease occurrence and spread. Triticale has, since its commercialization in the late 1960s shown good resistance to most cereal diseases. However, this situation has changed in the last decade as several fungal pathogens have adapted to this recently introduced host. In the following paragraphs, we review the major diseases that occur on triticale

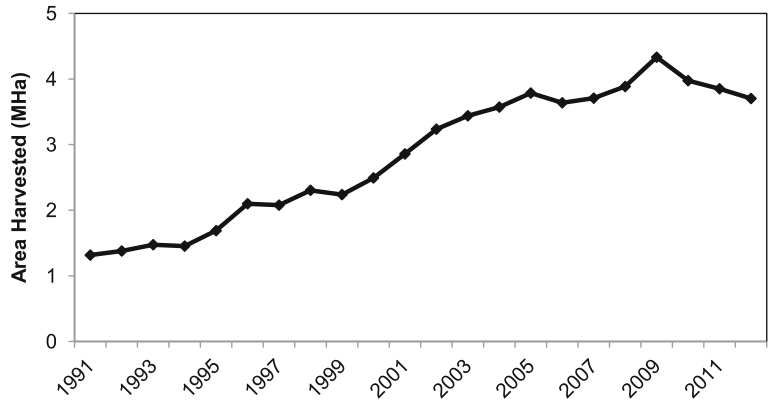

Fig. 1 Worldwide triticale area harvested (Mha) from 1991 to 2012 (FAO 2013)

(Fig. 3). Some fungal pathogens like *Stagonospora nodorum* (Berkeley) Castellani & Germano (teleomorph: *Phaeosphaeria nodorum* (E.Müller) Hedjaroude) causing leaf and glume blotch (Pojmaj and Pojmaj 1998; Oettler and Schmid 2000; Schinkel 2002), *Septoria tritici* Berkeley & Curtis (teleomorph: *Mycosphaerella graminicola* (Fuckel) J.Schröter) causing leaf blotch (Haesaert et al. 2006), and *Rhynchosporium secalis* (Oudemans) J.J.Davis causing scald (Welty and Metzger 1996) have also been observed in triticale, but are not included in this review. Reports of disease incidence caused by these pathogens are scarce, probably because these diseases are not (yet) broadly spread on triticale.

Powdery mildew

Powdery mildew (*Blumeria graminis* (de Candolle) Speer) is a widespread fungal disease of many monocotyledonous and dicotyledonous plants that is caused by *Ascomycetes* of the order *Erysiphales* (Bélanger et al. 2002). These pathogens are obligate biotrophs, which implies that their growth and reproduction is fully dependent on the living host. With the expansion of the triticale growing area during the last decade, powdery mildew (Fig. 3a) emerged on this new host and became a significant disease. Diseases are defined as 'emergent' if they have recently become a cause of concern due to an increase in virulence, infection of a novel host, and/or occurrence in a new area (Giraud et al. 2010).

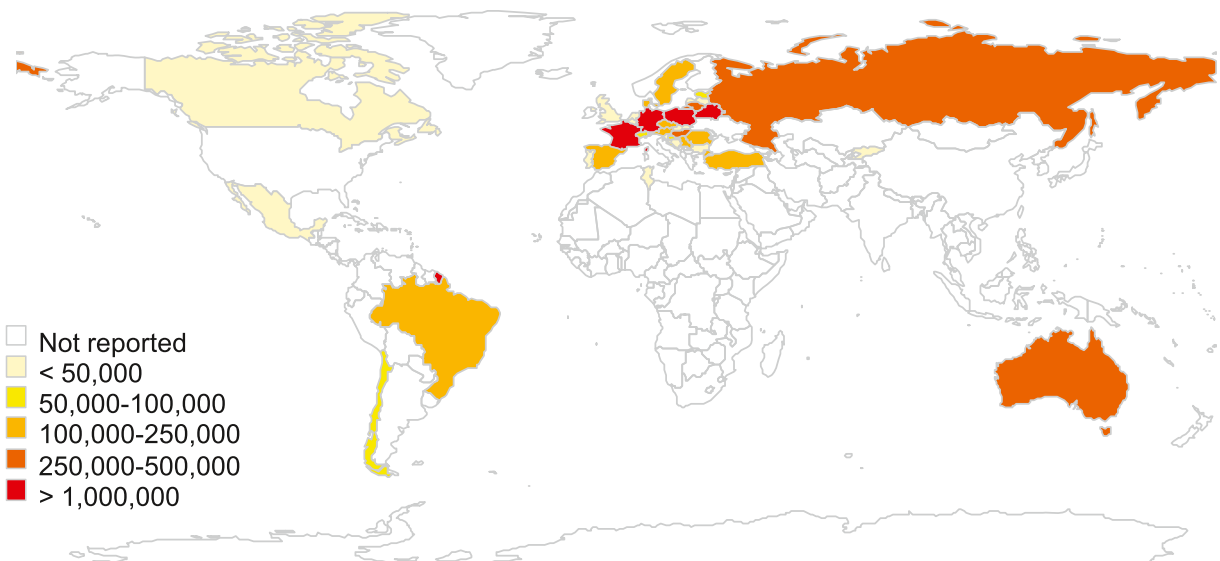

Fig. 2 Production quantity (tonnes) of triticale producing countries reported by FAO in 2012 (FAO 2013)

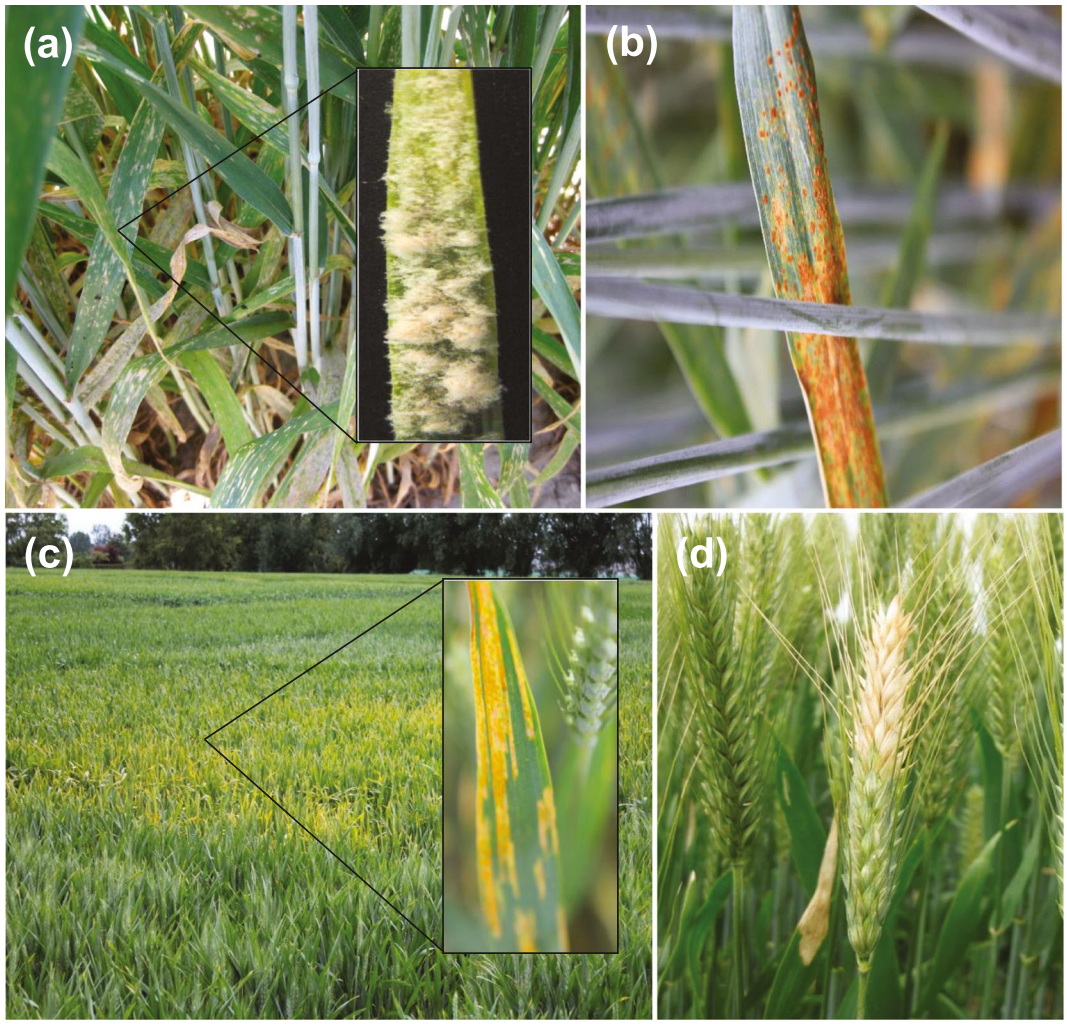

Fig. 3 Major diseases that threaten triticale production. **a** Powdery mildew caused by *Blumeria graminis*; **b** Brown (leaf) rust caused by *Puccinia triticina* **c** Yellow (stripe) rust caused by *Puccinia striiformis*; **d** Head blight caused by *Fusarium* spp.

The emergence of powdery mildew on triticale has been reported independently in several European countries. In 1977 Linde-Laursen reported that certain octoploid triticale lines were susceptible to wheat powdery mildew (*B.g.* f.sp. *tritici*), while both hexa- and octoploid triticale lines were resistant to rye powdery mildew (*B.g.* f.sp. *secalis*). They concluded that it is unlikely that resistance to powdery mildew derived from rye would be permanent when incorporated in triticale or wheat. At the 6th International Triticale Symposium, powdery mildew was recognised as an emerging problem of triticale in Poland (Strzembicka et al. 2006). In a multi-year survey between 1995 and 2013 in Belgium, the emergence of powdery mildew was abrupt. It is now one of the most important recurrent problems in triticale culture in Belgium (Fig. 4). Also in Switzerland, France and Germany, an increasing susceptibility of triticale to powdery mildew was recorded by several independent research groups, leading to new challenges for the breeder (Schori et al. 2007; Walker et al. 2011; Flath 2011; Klocke et al. 2013).

Recent pathology and genetic research demonstrated that this 'new' powdery mildew on triticale emerged through a host range expansion of powdery mildew of wheat (Walker et al. 2011; Troch et al. 2012). This means that wheat powdery mildew (*B.g.* f.sp. *tritici*) has evolved the capacity to colonise a new host species, triticale. A detailed phylogeographical study revealed that this host range expansion of wheat powdery mildew to the

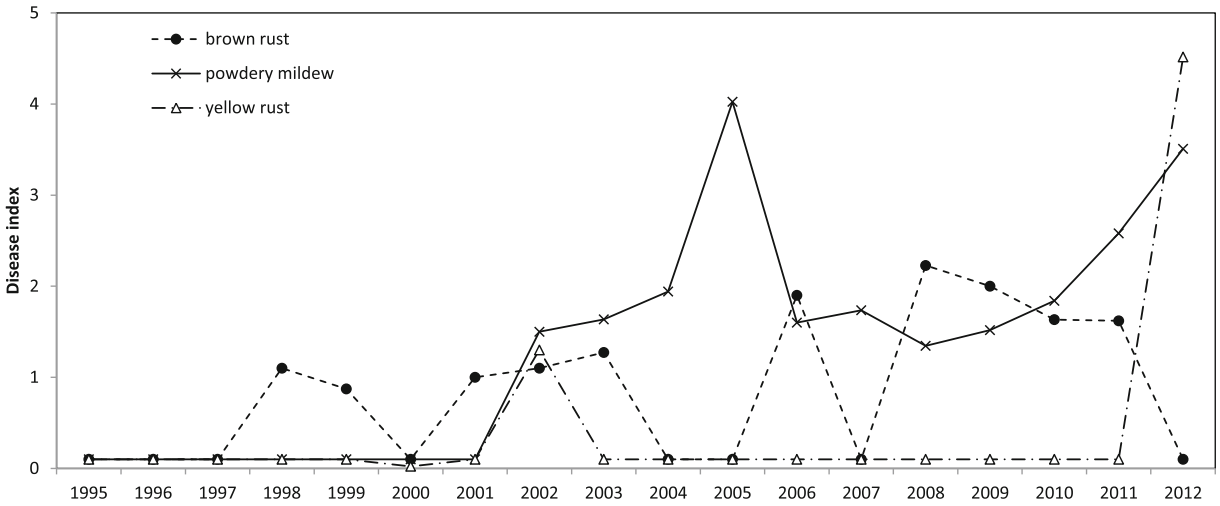

Fig. 4 Evolution of *Blumeria graminis, Puccinia triticina and Puccinia striiformis* symptoms from 1995 to 2012 in Belgium. In each year at least two locations in Belgium were assessed for disease symptoms. DI = disease index is a scale between 0 and 9 based on % of leaf surface covered with disease symptoms. The disease index is 0 for a healthy plant and 9 for a plant completely covered with fungal symptoms

new host triticale occurred recently, multiple times at different locations in Europe (Troch et al. 2012).

Rust diseases

Among the most important diseases in wheat that significantly reduce yield are those caused by the rusts, ranked 3rd of the most scientifically/economically important fungal pathogens (Dean et al. 2012). Rust fungi belong to the order *Uredinales* in the *Basidiomycetes* (Bolton et al. 2008). Like powdery mildew, rust fungi are highly specific obligate biotrophic pathogens.

Three rust diseases occur on wheat, namely leaf (brown) rust caused by *Puccinia triticina* Eriksson & Henning, stem (black) rust caused by *Puccinia graminis* f.sp. *tritici* Eriksson & Henning and stripe (yellow) rust caused by *Puccinia striiformis* f.sp. *tritici*. Westendorp Leaf rust is the most common rust disease of wheat and occurs more regularly and in more world-wide regions than stem rust or stripe rust (Bolton et al. 2008). In rye, leaf rust is caused by *Puccinia recondita* f. sp. *secalis* Rob. Ex. Desm., while stem (black) rust is caused by *Puccinia graminis* f.sp. *secalis* Eriksson & Henning and stripe (yellow) rust is caused by *Puccinia striiformis* f.sp. *secalis*. Westendorp.

For a long time, triticale had been considered relatively resistant to rusts (Mergoum et al. 2004). However, triticale disease scoring in the German National Trials during 1988–2001 showed that leaf and stripe rust occurred at a high level and were of increasing importance (Schinkel 2002). In Poland, hexaploid triticale has suffered significant losses due to leaf rust during the last decade (Sodekiewicz and Strzembicka 2004). Also in southern Russia significant crop losses due to this disease were reported (Mikhailova et al. 2009). A multi-year survey in Belgium for rust diseases on triticale depicted a striking fluctuating presence of the pathogens. Brown- and stripe rust were a recurring problem with fluctuating levels of infection while black rust was not present (Fig. 4).

Leaf rust How *P. triticina* causing leaf rust (Fig. 3b) adapted to triticale upon its introduction remains unknown. Information might come from recent evidence suggesting that diversity of *P. triticina* is correlated with adaptation to (wheat) hosts with different ploidy levels. Phylogenetic analyses showed the clear initial divergence of *P. triticina* isolates collected from *Aegilops speltoides* (the likely B genome donor of modern wheat) in Israel from the other isolates that were collected from tetraploid (AB genomes) durum wheat and hexaploid (ABD genomes) common wheat. Coalescence-based genealogy samplers also indicated that *P. triticina* on *A. speltoides*, diverged initially, followed by *P. triticina* isolates from durum wheat in Ethiopia and then by isolates from common wheat. Isolates of *P. triticina* found worldwide on cultivated durum wheat were the

most recently coalesced and formed a clade nested within the isolates from common wheat. By a relative time scale, the divergence of *P. triticinia* as delimited by host specificity appears very recent (Rodrigue and Kolmer 2013). Analyses have revealed that leaf rust on triticale is caused by pathotypes of the wheat leaf rust fungus that have become virulent to triticale genotypes (Sodekiewicz et al. 2008). A microsatellite analysis of *P. triticina* races in South Africa concluded that a leaf rust race collected from triticale in 2005 probably represents a mutation from an existing race, because it showed 96 % genetic homology with other races collected from wheat (Visser et al. 2012).

Stripe rust Large-scale epidemics caused by new virulent and aggressive strains of yellow (stripe) rust (Fig. 3c) have been reported on wheat (Hovmøller et al. 2010). These severe and widespread yellow rust epidemics have been ascribed to new and more aggressive races adapted to warmer environments (Hovmøller et al. 2011). In 2001, a high level of infection by yellow rust on triticale was reported in Germany, with yield losses up to 21 % (Tian et al. 2004). Field observations in Poland showed that yellow rust was becoming an increasingly serious threat to triticale cultivation (Sodekiewicz et al. 2009). In 2009, a new pathotype of yellow rust spread rapidly and overcame resistance in triticale cultivars in Denmark (Solh et al. 2012). A significant proportion of CIMMYT triticale germplasm is susceptible to yellow rust (Zhang et al. 2010). Cereal rust reports of Australia document that current Australian triticale varieties are relatively resistant to leaf and stem rust, but increasingly susceptible to yellow rust (Wellings et al. 2012). In Belgium, low occurrence of infections by yellow rust were recorded in the last decade on triticale whereas in 2012 a spectacular increase of the pathogen was recorded (Fig. 4), an observation that was not consolidated in 2013 (data not shown).

Stem rust Black (stem) rust in wheat and rye is caused by *P. graminis* f.sp. *tritici* and *secalis* respectively (Berlin et al. 2012). Both can reside on the alternate host *Berberis* spp. which may increase the number of races in the pathogen. Stem rust is one of the most destructive diseases of wheat worldwide and its specialisation in different races has impacted strongly wheat breeding and production (Dean et al. 2012). Numerous wheat cultivars protected by single genes have become susceptible to stem rust, often with devastating "boom-and-bust" effects (Dean et al. 2012). The occurrence of race Ug99 and its variants in Eastern Africa with virulence for a commonly used resistance gene, attracted increased funding and research in some countries, as 90 % of the world's wheat is susceptible (Pretorius et al. 2000; Singh et al., 2008). Recent information on occurrence of stem rust on triticale is scarce although stem rust was recognised as the most important disease on triticale in Australia (Adhikari and McIntosh 1998). Stem rust was also observed on triticale in Poland, where varietal differences in susceptibility were observed (Wakulinski et al. 2006).

Fusarium spp.

Fusarium head blight (FHB) is widespread on small grain cereals, including wheat, oat, rye, barley and triticale. FHB can be caused by different species all infecting the host resulting in indistinguishable symptoms (Fig. 3d). They can occur alone or in species complexes. The main species of the FHB complex in Europe on triticale are *Fusarium graminearum Schwabe*, *F. culmorum* (W.G.Smith) Saccardo, *F. avenaceum* (Corda) Saccardo, *F. poae* (Peck) Wollenweber and *Michrodochium. nivale* (Fries) Samuels & I.C.Hallett (Vanheule et al. 2014). FHB affects grain yield through premature death of spikelets and abnormal grain filling. Besides reducing yield, research interest in FHB is primarily fuelled by the fact that most members of the disease complex produce mycotoxins. The most important mycotoxin is the trichothecene deoxynivalenol (DON) due to its omnipresence. DON has been shown to act as a virulence factor which helps the pathogen to spread in the rachis especially in wheat (Parry et al. 1995; Kazan et al. 2012; Walter et al. 2010; Audenaert et al. 2014).

In general, triticale cultivars are less susceptible towards *Fusarium* infection than wheat cultivars (Góral et al. 2013). However, in recent years FHB epidemics in triticale have increased in frequency (Veitch et al. 2008). Opoku et al. (2013) detected high levels of *F. langsethiae* in triticale. *F. langsethiae* Torp & Nirenberg is a recently characterised species within the genus *Fusarium*, mainly infecting oats and spring barley. The predominance of the different *Fusarium* species may differ from year to year and from region to region and is related to tillage systems, intercrops, temperature, and moisture requirements. Dry and warm weather conditions favour *F. poae*, whereas *F. graminearum* prefers

warm and humid conditions. *F. culmorum* and *Michrodochium* prefer cooler temperatures (Opoku et al. 2013).

In triticale, a positive correlation between disease symptoms and grain DON content exists although it is less convincing than in wheat (Miedaner et al. 2004). As triticale is used in animal production, mycotoxins pose a serious threat for animal health (Langevin et al. 2004). Research on mycotoxins is often limited to DON, although the presence of other mycotoxins with a toxicity often exceeding that of DON multiple fold is often overlooked. Other type B trichothecenes such as nivalenol, type A trichothecenes such as T-2, HT-2, and diacetoxyscirpenol, zearalenon and fumonisins can be produced by different members of the FHB complex. In addition, there is increasing interest in emerging mycotoxins such as beauvericin, fusarenon-X, moniliformin and enniatins and in masked mycotoxins (e.g. DON-3G) which comprise derivatives of the parent mycotoxins (Pereira et al. 2014).

FHB intensity and mycotoxin contamination are influenced by a complex interaction between environmental growing conditions and local agronomic practices. Intense rainfall during the period of anthesis, the most susceptible growing stage for infection, disperses *Fusarium* inoculum from crop residues and promotes FHB infection. No tillage or minimum tillage systems favour FHB infection due to their capacity to survive saprophytically on crop residues of host plants as maize, small grain cereals and grasses. Probably the most obvious sources of inoculum for the development of FHB epidemics arise from infected crop residues and therefore crop rotation is important to avoid inoculum build-up (Pereyra and Dill-Macky 2008, Landschoot et al. 2011, 2012).

In several studies it has been shown that fungicide application in triticale generally results in a reduction of *Fusarium* symptoms. Nevertheless, the impact of these fungicides on mycotoxins levels are highly variable ranging from increased levels to decreased levels compared to the untreated control fields (Mankeviciene et al. 2008; Audenaert et al. 2010; Gaurilcikiene et al. 2011; Audenaert et al. 2011).

History of triticale breeding

Triticale (×*Triticosecale* Wittmack) is the intergeneric hybrid between the female parent wheat (*Triticum* spp.) and the male parent rye (*Secale* spp.). What makes the history and evolution of triticale as a species so unique compared to other cereals like wheat or rye, is that its evolution occurred during the last 130 years and it is almost all directed by humans (Mergoum et al. 2009). The origin of triticale dates back to 1873 when Scottish scientist, A. Stephen Wilson, made the first cross between wheat and rye (Oettler 2005). During the subsequent years, many publications in the 1800s on wheat-rye hybrids were recorded; most notably the spontaneous doubling of chromosomes in the partially fertile hybrids grown by Rimpau in 1888 (Oettler 2005). The use of colchicines to double the chromosome number followed by the development of improved techniques of embryo culture, to rescue the aborting embryo, initiated commercial scale triticale breeding. Triticale exhibits amphiploidy with respect to wheat (AABBDD) and rye (RR) genomes (Ammar et al. 2004). Stable hexaploid (AABBR/D) and octoploid (AABBDDRR) triticale cultivars have been bred, resulting from a cross between tetraploid or hexaploid *Triticum* spp. and rye although the octoploid cultivars displayed partial sterility. Primary triticale cultivars result from crossing wheat and rye; secondary triticale cultivars result from crossing two triticale cultivars or a triticale cultivar with a wheat or rye cultivar (McGoverin et al. 2011). Even crosses between triticale cultivars bearing different ploidy levels are possible.

The first commercially available cultivars of triticale were released in 1968 from a Hungarian breeding program, the result of an octoploid–hexaploid cross (Ammar et al. 2004). Triticale breeding programs were initiated in Mexico (CIMMYT), Poland and France in the 1960s, and in Brazil, Portugal and Australia in the 1970s (Müntzing 1979; Oettler 2005; McGoverin et al. 2011). Today, two types of secondary hexaploid triticale are the most commercially grown triticale worldwide; complete triticale, which carry all seven pairs of unchanged chromosomes from rye, and substituted triticale, which have one or more of the rye chromosomes replaced with D-genome chromosomes from hexaploid wheat (Fox et al. 1990). There is some evidence that triticale varieties vary in their resistance to abiotic and biotic stress depending on the number of rye chromosomes present, with varieties that have a greater number of rye chromosomes having greater resistance

(Mergoum et al. 2009). Triticale breeding strategies utilised to ameliorate modern triticale cultivars for various uses have recently been reviewed by triticale breeders (Mergoum et al. 2009). They comprise yield, lodging resistance, winterhardiness, sprouting resistance and disease resistance as major breeding objectives (Haesaert and De Baets 1994).

Resistance breeding in triticale

Breeding for resistance: qualitative versus quantitative resistance

At present, the genetic diversity in current programs is extremely narrow (Mergoum et al. 2004). Moreover, it is known that triticale germplasm is widely shared among breeding centres from around the world (Kuleung et al. 2006). Recently, a genome-wide evaluation of genetic diversity based on a set of 161 diverse triticale lines of worldwide origin suggested that only few genetically similar rye lines have been used for the establishment of primary spring type triticale (Alheit et al. 2012).

The ability to adapt triticale to withstand multiple biotic stresses is critical to its future growth as a crop. An effective and environmentally sensitive approach to disease control involves breeding crop plants for resistance (Dodds and Rathjen 2010). Disease resistance is particularly important for low-input farming, which is common practice in triticale production, typically grown as forage for livestock feed.

Two general categories of host resistance have long been reported in plants: qualitative resistance conferred by a single resistance (*R*) gene and quantitative resistance mediated by multiple genes with each providing a partial increase in resistance (Poland et al. 2009; Kou and Wang 2010). However, it is important to note that host resistance often cannot be described simply as either qualitative or quantitative, and in some cases a gray zone between the two may exist (Poland et al. 2009; St.Clair 2010).

In qualitative resistance, known as gene-for-gene resistance or effector-triggered immunity (ETI) (Jones and Dangl 2006), the outcome of infection is based on the interaction of dominant resistance (*R*) genes in the host and dominant avirulence (*AVR*) genes in the pathogen. This type of resistance is specific to pathogen race and is lifetime limited in a particular cultivar due to strong selection pressure against rapid evolution of the pathogen (McDonald and Linde 2002).

Resistance genes for the most important pathogens have been compiled in parent crop wheat and to a lesser extent in rye (Table 1).

McDonald and Linde (2002) suggest that breeding efforts should concentrate on quantitative resistance, controlled by multiple genes each with small effects. This is often more durable than race-specific resistance because pathogens often adapt more slowly to it if at all (Brown and Tellier 2011). The quantitative nature of this resistance makes it, however, more complicated to handle in a breeding program compared to race-specific resistance because identifying these multiple genes with small phenotypic effects is a cumbersome task. The selection for quantitative resistance could, therefore, be more efficient with the aid of molecular markers linked to the genes that underlie a quantitative trait. Examples in practical wheat breeding in which marker-assisted selection (MAS) has been successfully applied are the wheat rust resistance gene *Lr34* and *Yr36* and two QTL for resistance to *Fusarium* head blight (Miedaner and Korzun 2012).

Breeding for resistance against powdery mildew

Pm resistance genes from wheat can relatively easily be transferred from wheat to triticale by crossing triticale cultivars with wheat cultivars. Kowalczyk et al. (2011) transferred *Pm4b* and *Pm6* from common wheat to triticale cultivars Fideloi, Magnat and Lamberto which resulted in an increased field resistance. Molecular insights into the presence of race-specific resistance genes in commercial triticale cultivars are fragmentary and often difficult to interpret. Using molecular markers, race-specific resistance genes *Pm17 and Pm3f* were frequently encountered in a set of 15 European commercial cultivars (Troch et al. 2013a). This study highlighted the narrow genetic base of triticale in relation to powdery mildew resistance, underscoring the need to deploy new sources of resistance in triticale. In addition, this study highlighted a common problem in interpreting the presence of resistance genes in triticale. Indeed, results from gene postulation multipathotype tests and molecular marker studies do not always match. Gene postulation can be complicated by interactions between R genes. This was recently found for the rye-derived powdery mildew R gene *Pm8* in wheat, which is suppressed by translated alleles at the *Pm3* locus (McIntosh

Table 1 Resistance genes, QTLs and suppressor genes in wheat and rye adapted from Tyrka and Chelkowski 2004 and McIntosh et al. (2008)

		Wheat	Rye
Powdery mildew	Designated genes	Pm1a-e, Pm2, Pm3a-g, Pm4a-b, Pm5a- e, Pm6, Pm7, Pm8, Pm9, Pm10, Pm11, Pm12, Pm13, Pm14, Pm15, Pm16, Pm17, Pm18, Pm19, Pm20, Pm21, Pm22, Pm23, Pm24, Pm25, Pm26, Pm27, Pm28, Pm29, Pm30, Pm31, Pm32, Pm33, Pm34, Pm35, Pm36, Pm37, Pm38, Pm39	Pm, Pm1, Pm2, Pm3, Pm4, Pm5, Pm6, Pm7, Pm?, Pm8
	Temporarily designated genes	PmLK906, PmPs5A, PmY39, Ml-Ad, Ml-Br, Mld, Ml-Ga, Mlm3033, Mlm80, mljy, mlsy, mlRd30, Mlre, Mlxbd, MlTd1055, Mlzec1	
	Suppressor genes	SuPm8	
	QTLs	QPm.vt-1B, QPm.vt-2A, QPm.vt-2B, QPm.sfr-1A, QPm.sfr-1B, QPm.sfr-1D, QPm.sfr-2A, QPm.sfr-2D, QPm.sfr-3A, QPm.sfr-3D, QPm.sfr-4A.1, QPm.sfr-4A.2, QPm.sfr-4B, QPm.sfr-4D, QPm.sfr-5A.1, QPm.sfr-5A.2, QPm.sfr-5B, QPm.sfr-6B, QPm.sfr-7B.1, QPm.sfr-7B.2, QPm.ipk-2B, QPm.ipk-4B, QPm.ipk-7D	
Yellow/stripe rust	Designated genes	Yr1, Yr2, Yr3a-c, Yr4a-b, Yr5, y6, Yè, Y8, Y9, Y10, Y11, Y12, Y13, Y14, Y15, Y16, Y17, Y18, Y19, Y20, Y21, Y22, Y23, Y24, Y25, Y26, Y27, Y28, Y29, Y30, Y31, Y32, Y33, Y34, Y35, Y36, Y37, Y38, Y39, Y40, Y41	Yr1, Yr2, Yr3, YrBl
	Temporarily designated genes	YrA, YrAlp, YrCle, YrCK, YrD, YrDa1, YrDa2, YrDru, YrDru2, YrH46, YrH52, YrHVII, YrMin, YrMor, YrND, YrS, YrSte, YrSte2, YrSP, YrSp, YrTye, YrTr1, YrTr2, YrYam, YrZH84, YrV23, Yrns-B1	
	QTLs	QYr.inra-2BL, Qyr.inra-2AL, QYr.inra-2BL, QYr.inra-2DS, Qyr.inra-5BL.1, QYr.inra-5BL.2, QYr.sgi-7D, QYr.sgi.2B.1	
Brown/leaf rust	Designated genes	Lr1, Lr2a-c, Lr3a-c, Lr4, Lr5, Lr6, Lr7, Lr8, Lr9, Lr10, Lr11, Lr12, Lr13, Lr14a-b, Lr14ab, Lr15, Lr16, Lr17a-b, Lr18, Lr19, Lr20, Lr21, Lr22a-b, Lr23, Lr24, Lr25, Lr26, Lr27, Lr28, Lr29, Lr30, Lr31, Lr32, Lr33, Lr34, Lr35, Lr36, Lr37, Lr38, Lr39, Lr40, Lr41, Lr42, Lr43, Lr44, Lr45, Lr46, Lr47, Lr48, Lr49, Lr50, Lr51, Lr52, Lr53, Lr54, Lr55, Lr56, Lr57, Lr58, Lr59, Lr60, Lr61, LrKr1, LrKr2, LrMq1, LrTb, LrTù, LrTr, LrTt1, LrPVM, LrW2	Lr1, Lr2, Lr3, Lr4, Lr5, Lr6, Lr7, Lr25, Lr26, Lra-c, Lrg-h, LrSatu
	Suppressor genes	SuLr23	
	QTLs	QLr.sfr-1B, QLr.sfr-2B, QLr.sfr-3A, QLr.sfr-4B, QLr.sfr-4D, QLr.sfr-5D, QLr.sfr-7B.1, QLr.sfr-7B.2	Qlr.pser.1BL
Black/stem rust	Designated genes	Sr2, Sr3, Sr4, Sr5, Sr7a-b,Sr8a-b, Sr9a-g, Sr10; Sr11, Sr12, Sr13, Sr14, Sr15, Sr16, Sr17, Sr18, Sr19, Sr20, Sr21, Sr22, Sr23, Sr24, Sr25, Sr26, Sr27, Sr28, Sr29, Sr30, Sr31, Sr32, Sr33, Sr34, Sr35, Sr36, Sr37, Sr38, Sr39, Sr40, Sr41, Sr42, Sr43, Sr44, Sr45, Sr46, SrA, SrR, SrTmp, SrWild, SrZdar	Sr1, Sr2, Sr+, SrNin, SrBj, SrVent, SrSatu, SrLa1, SrLa2
Fusarium head blight	Designated genes	Fhb1, Fhb2, Fhb3, Fhs1, Fhs2	
	QTLs	QFhs.inra-2A, QFhs.inra-2B, QFhs.inra-3A, QFhs.inra-3B, QFhs.inra-5A.1, QFhs.inra-5A.2, QFhs.inra-5A.3, QFhs.inra-5D, QFhs.inra-6D, QFhs.ndsu-2A, QFhs.ndsu-3AS, QFhs.ndsu-3BS, Qfhi.nau-4B, Qfhi.nau-5B, Qfhs.umc- 2B, Qfhs.umc-3B, Qfhs.umc-4B, QFhs.pur-2D, QFhs.pur-7El, Qfhs.ifa-5A, Qfhs.crc-2BL, Qfhs.ndsu-3AS, Qfhs.crc- 6BS, Qfhs.fcu-7AL, QFhs.fal-6DL, QFhs.fal-5BL.1, QFhs.fal.4AL, QFhs.whs-1BS, QFhs.whs-3B, QFhs.whs-3DL, QFhs.lfl-6AL, QFhs.lfl-7BS, Frontana QTL, QFhs.ndsu-3BS, QFhs.nau- 2DL, QFhs.nau-1AS, QFhs.nau-7BS, QFhs.ndsu-3BS, QFhs.nau-2DL, QFhs.nau-5AS QFhs.whs-5BL, QFhs.whs-7AL, QFhs.whs-7BL	

et al. 2011). The presence of the rye-derived R gene *Pm17* in most of the current triticale cultivars seems highly likely, because this gene might have been present in one of the rye ancestors. *Pm17* is located on the same chromosome arm as *Pm8* but was translocated to wheat chromosome 1AL instead of 1BL. Transferring gene *Pm17* into variety Amigo from a T1AL.1RS to a T1BL.1RS translocation in 'Helami-105' and subsequent crosses with *Pm8* varieties have shown that the two genes are allelic.

A study in Poland showed that hybrids derived from triticale crosses with common wheat cultivars carrying race-specific resistance genes (*Pm4b* and *Pm6*) displayed increased field resistance, suggesting the potential of introducing wheat genes to improve powdery mildew resistance in triticale (Kowalczyk et al. 2011). However, by genetic mapping of race-specific resistance genes, a dominant monogenic inheritance was identified in triticale lines, indicating that the use of major gene resistance seems to have a restricted durability only (Flath 2011; Klocke et al. 2013). Fortunately, cultivars highly resistant at the adult plant growth stage were identified in field studies (Flath 2011; Troch et al. 2013a), which could be of high value for durable powdery mildew resistance breeding. Additionally, a recent cytological study underscores the importance of non-penetrated papillae formation in the resistance response of triticale to powdery mildew, which is related to more durable resistance compared to programmed cell death (Troch et al. 2013b).

Breeding for resistance against rusts

Genetic resistance is the preferred method to reduce losses from leaf rust. Evaluation of seedling resistance to leaf rust in international triticale germplasm distributed by CIMMYT in 2005 indicated good resistance among the entries, which could represent potentially useful sources of seedling resistance in developing new triticale cultivars (Zhang et al. 2010). In resistance tests of winter triticale cultivars from Central Europe, differences in reaction patterns were observed, indicating that the tested cultivars possessed different genes for leaf rust resistance (Hanzalová and Bartoš 2011). By introducing leaf rust resistance into introgressive triticale lines with *Triticum monococcum* (diploid wheat) genes, it was found that some lines comprised partial resistance at the adult plant stage and complete resistance at the seedling stage (Sodekiewicz and Strzembicka 2004). By studying the chromosomal location of leaf rust resistance genes in these introgressive triticale lines, the transfer of two complementary resistance genes was reported (Sodekiewicz et al. 2008). Compared to wheat there is little information available on the inheritance of leaf rust resistance in rye, the number of resistance genes, their genomic location and their effectiveness.

Leaf rust Genetic origin of leaf rust resistance in triticale has been studied by several authors. Quinones et al. (1972) reported monogenic resistance in five triticale genotypes. Singh and McIntosh (1990) identified a resistance gene denominated as *LrSatu* in CIMMYT lines. Wilson and Shaner (1989) described genes for hypersensitive resistance in triticale which was confirmed by Grzesik and Strzembicka (2003).

It was shown that two resistance genes *LrTM16* and another not defined gene could be introduced from *T. monococcum*. These genes have a synergistic effect with respect to their ability to induce resistance (Sodekiewicz et al. 2008). In addition, it appeared that *LrTM16* also increased the resistance to stripe rust (Sodekiewicz et al. 2009).

Stripe rust In stripe rust tests, 93 % of the lines were postulated to carry *Yr9* (Zhang et al. 2010). Numerous sources of stripe rust resistance were characterised among western Canadian triticale varieties (Randhawa et al. 2012). Several introgressive triticale lines with stripe and leaf rust resistance introduced from *Triticum monococcum* showed resistance both at seedling and adult plant stage, which could be of great importance for triticale breeding (Sodekiewicz et al. 2009).

Stem rust In triticale, severe losses due to the development of virulence in wheat stem rust for a commonly used resistance gene in triticale were observed in the early 1980s in Australia and in 1988 in South Africa (Park 2007; Pretorius et al. 2007). Current commercial triticale cultivars display high levels of stem rust resistance, likely as a consequence of the release of cultivars with gene combinations. Genetic studies led to the identification of several stem rust resistance genes in triticale (McIntosh et al. 1995). Adhikari and McIntosh (1998) demonstrated that chromosomes 2R and 3R are important carriers of stem rust resistance genes in hexaploid triticaleand hypothesized that several genes of rye (*Sr27, SrNin, SrSatu, SrBj, and SrVen*) are present in triticale genotypes. According to Singh and McIntosh (1990)

approximately 50 % of the screening nursery lines possessed *SrSatu* on chromosome 3R. Other resistance genes were depicted amongst others: *SrLa1, SrLa2, SrBj, SrJ, SrVen, SrBj, SrNin and RM4*. A more recent study investigating seedling resistance to stem rust, leaf rust and stripe rust showed that 15 % and 85 % of entries were postulated to carry *Sr27* and *SrSatu*, respectively (Zhang et al. 2010). When compared with previous studies the results suggest a lack of expansion in the diversity of stem rust resistance.

In a recent study including entries from European breeders, stem rust resistance to Ug99 in triticale was identified, and this resistance was conferred mostly by single genes (*Sr27, SrSatu, and SrKw*) with dominant effects (Olivera et al. 2013). Therefore, the authors concluded that triticale can provide novel genes to increase the diversity of stem rust resistance in wheat. However, virulence to *Sr27*, *SrSatu*, and *SrKw* has been reported; therefore, use of these genes should be limited to areas where virulence to these genes is absent in the rust population.

Breeding for resistance against *Fusarium*

Resistance to FHB in small grain cereals is quantitatively inherited and comprises many QTL described especially in wheat (Table 1). Pedigrees of European winter wheat varieties showing moderate resistance to FHB have been compiled (Kosova et al. 2009). In the triticale parental crops wheat and rye, mainly additive effects were responsible for the large genetic variation found in triticale (Oettler et al. 2004; Mesterhazy 1995; Miedaner et al. 1996). This high entry-mean heritabilities of FHB rating and the expected gains from selection make it feasible to improve the FHB resistance level by recurrent selection (Miedaner et al. 2006).

A major issue remains the correlation between crop resistance and presence of mycotoxins. Indeed, correlation between FHB symptoms and concomitant DON content varies with genotype and environment (Landschoot et al. 2012). In addition high correlation were depicted in wheat and triticale, while correlations were lower in rye (Miedaner et al. 2004).

New approaches and concerns for triticale breeding

Interspecific hybridization is the most common method used by breeders to transfer disease resistance genes from *Triticum* and *Secale* species into triticale. In this way resistance genes used in wheat and rye breeding can be incorporated in triticale although genes can be genetically suppressed in triticale genetic background. Many resistance genes have already been transferred successfully from *Triticum* and *Secale* species (or their relatives) to triticale (e.g. *Pm, Yr, Lr, Sr* genes). A complete overview of wheat and rye resistance genes usable in triticale breeding is given in Table 1. At the R genome level *Secale montanum* and *S. africanum* are reported to carry excellent disease resistance genes (Lei et al. 2013).

Several methods such as the creation of new primary triticales or crosses between hexaploid triticale and wheat are used to increase genetic diversity of triticale and to introgress new resistance genes in triticale. Crosses of triticale and rye results in tetraploid triticale which can be used to introduce D chromosomes in hexaploid triticale leaving the R genome complete. To extend the diversity of resistance and to obtain a durable resistance, attention should be paid to the incorporation of the resistance genes in both wheat and rye parents prior to the synthesis of primary triticales. Crossing triticale directly with relatives of wheat and rye has been attempted, but in most cases the F_1 plants showed sever meiotic disturbances which prevented the utilisation of this type of crosses (Gruszecka et al. 2004).

Conventional triticale breeding is a slow process which typically requires 8–12 years from initiation to varietal release. Therefore, conventional and modern breeding approaches, involving shuttle breeding, hybrid breeding, double haploid technology, marker-assisted selection (MAS), and genetic transformations, are combined in most triticale-breeding programs (Mergoum et al. 2009; Gowda et al. 2013). The availability of a high-density linkage map in hexaploid triticale can serve as a useful tool to facilitate mapping and introgression of resistance genes (Alheit et al. 2011; Badea et al. 2011; Tyrka et al. 2011).

In addition, one should use the well-defined MAS strategies implied in wheat. Protocols for MAS in wheat include protocols for genes of resistance to leaf rust, stripe rust and FHB. This information is also, to a lesser extent available in rye (Tyrka and Chelkowski 2004). Genetic maps of rye have been published and a number of resistance genes have been localised.

Additionally, next-generation sequencing technologies may have the power and potential to assess complex polyploid genomes, like triticale, facilitating novel approaches and possibilities for genomics-assisted

breeding (Edwards et al. 2013). Karyotyping of the rye chromosomes in triticale cultivars through FISH (Fluorescent in situ hybridization) might help to estimate variability among triticale cultivars which is important to reduce the risk of chromosome irregularities in future crosses (Fradkin et al. 2013).

Recently, improved efficiency of doubled haploid generation in hexaploid triticale was observed by *in vitro* chromosome doubling (Würschum et al. 2012), a tool from which e.g. QTL mapping could benefit greatly. Future breeding strategies should focus at broadening the genetic background of new varieties that are being developed, through introgression and deployment of new sources of disease resistance. not only from the A and B genome but also from the D genome. The hexaploid wheat gene *Lr34* located on chromosome 7D, which encodes an ATP-binding cassette (ABC) transporter, confers durable field resistance against rusts and powdery mildew. Despite its extensive use in breeding and agriculture, no increase in virulence towards *Lr34* has been described over the last century and is therefore a promising gene to exploit in sustainable broad-spectrum resistance (Krattinger et al. 2009, 2013). The functionality of this gene has already been demonstrated in barley (Risk et al. 2013). D-genome species such as *Aegilops tauschii* and *Aegilops ventricosa* are highly valuable gene resources. 31 resistance genes have been transferred from *Aegilops* spp. to wheat. Exploiting those species for creation of new primary triticale might be a good breeding strategy in view of the progressive loss of effective resistance genes in triticale and the widening of the genetic diversity of triticale (Tyrka and Chelkowski 2004; Kwiatek et al. 2013). Importantly, they should focus on both quantitative and multipathogen sources of resistance.

However, there are some key concerns in triticale breeding. The expression of resistance genes in the new genetic background of triticale remains a major issue. An improvement of our understanding of how key traits behave in triticale is important to imply predictive breeding. An accumulating amount of evidence has affirmed that genetic diversity in wheat and rye used for primary triticale is very narrow. It is therefore unlikely that continued reshuffling of genes will be sufficient to meet the challenges of the plasticity of biotic stresses. A proliferated and well thought-out use of the extensive reservoir of genes in parent rye and wheat plants will be necessary to guarantee durable resistance to multiple pathogens.

Conclusions and future perspectives

The expansion of the triticale growing area is likely to continue globally. As we enter an era of drier and warmer climates in which more unfavourable soils will be included in farming systems, triticale may be well placed to take advantage on other cereals due to its adaptability to be grown in more marginal environments. Additionally, with the increasing desire for renewable energy sources, triticale straw residue is an excellent candidate for biofuel production as it has a high biomass production and amylase activity is higher compared to wheat. However, this expansion of triticale production is threatened by the disease emergence of powdery mildew, rusts and *Fusarium* head blight. *Vice versa*, this expansion may favour the emergence of new diseases and new pathogen genotypes. Therefore, to keep triticale suitable for low input farming, it is a challenge for plant breeders and researchers to achieve durable disease resistance in triticale. Recent advances in genetic analysis will support breeders' efforts to do this. Altogether, resistance breeding efforts, along with international pathogen surveillance, and research efforts covering triticale genetics, pathogen biology, epidemiology and evolutionary genetics, could provide a sustainable solution for triticale production.

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