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REVIEW

Practical breeding strategies to improve resistance to Septoria tritici blotch of wheat

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Abstract Septoria tritici blotch (STB), caused by fungal agent *Zymoseptoria tritici* (previously known as *Mycosphaerella graminicola*) is a devastative foliar wheat diseases globally. Importance and potential threat of STB have been discussed historically and geographically. This paper reviews information on the *Z. tritici*—wheat pathosystem and proposes approaches to identify resistance genes and to advance in breeding for STB resistance. Screening of resistant lines/cultivars, QTL mapping analysis within genetic populations derived from crosses, detection of new resistance gene(s) and finally application of *Stb* gene carrier line/cultivar in crosses are the major stages of a practical wheat-breeding program against STB of wheat. Phenotyping and genotyping outputs on the top of each other should confirm each other, so it needs to expose a resistance gene carrier line/cultivar in the epidemic condition at seedling/adult plant stage to confirm resistance performance of detected gene(s) in the real condition. On the other word, detecting an associated QTL to resistance should not be considered as the end of investigation. Climate change resulted geographical disease pattern conversion where some diseases became more important in some area where they had not been serious in the past and vice versa. Hence, a reconsideration of wheat disease importance zone is necessary to predict regions where STB is and will be a limitation for wheat yield improvement.

Keywords Wheat · Breeding · Resistance · Septoria tritici blotch

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Introduction and importance

Septoria tritici blotch (STB) is one of the most destructive wheat diseases described first by Desmazières in 1842 and later by Sprague (1938). The causal agent is the ascomycete *Zymoseptoria tritici* (Quaedvlieg et al. 2011), which was first observed in 1894 but the association between this fungus and STB was only discovered almost 80 years later in New

Zealand (Sanderson 1976). STB gained importance in the early 1970s possibly due to a combination of improved genetic control of wheat rusts and the promotion of conservation agriculture that supported the over-summering of many pathogens, including *Z. tritici* (Mergoum et al. 2007). Moreover, industrialization and climate change also influenced the incidence of *Z. tritici* and *Stagonospora nodorum* (Bearchell et al. 2005; Shaw et al. 2008). Current forecasts project a geographically variable but steady increase of STB (Roos et al. 2010).

Zymoseptoria tritici has an asexual (Quaedvlieg et al. 2011) as well as a sexual life cycle that is driven by its heterothallic bipolar mating system (Kema et al. 1996c) resulting in rain-splash-dispersed pycnidiospores and airborne ascospores, respectively. Ascospores are known to be the initial infection sourced by previous crop wheat debris. Disease progress during the growing season is largely driven by the rain splash-borne pycnidiospores, although ascospores can be formed year-round (Linde et al. 2002; Ponomarenko et al. 2011).

Temperature and relative humidity (RH %) have long been considered as the two most critical factors for *Z. tritici* establishment. A range of temperatures (12–25 °C) was tested and 22 °C was determined as the optimal temperature for disease development. During incubation, a leaf wetness period of at least 48 h post inoculation is required for penetration and the initialization of colonization (Kema et al. 1996a). After incubation, the relative humidity should be $\geq$ 85% for optimal disease development. In the field, pycnidia exude cyrrhi (Fig. 1) with conidia release maximized at 100% RH and reduced to half at 98% (Gough and Lee 1985). A positive correlation has been reported between post-harvest sunlight hours and STB incidence in the following year. This might be due to the reduced reproduction of saprotrophic organisms, suggesting a greater quantity of nutrients in the wheat residue and available for *Z. tritici* pseudothecia development (Shaw et al. 2008). Greenhouse experiments and host–pathogen interactions of related wheat pathogens indicate that light is a crucial environmental factor for disease development (Kema et al. 1996b).

Suboptimal field conditions significantly prolong the latency period of *Z. tritici* and hence delay the appearance of disease symptoms, but rarely reduce the damage on susceptible cultivars (Henze et al. 2007).

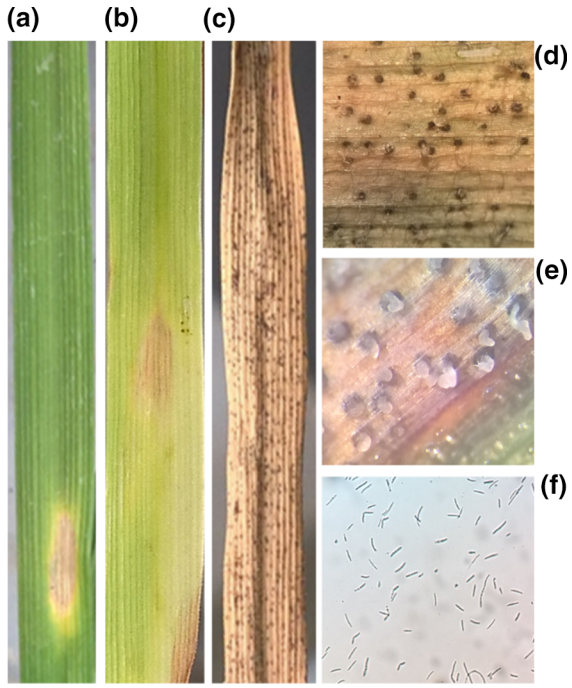

Fig. 1 Macroscopic and microscopic (**a**–**f**) disease symptoms of *Zymoseptoria tritici* on wheat. **a** Chlorosis around necrotic area; **b** Necrosis; **c**, **d** Pycnidia distributed over necrotic leaf area; **e** Fructification: Exuding pycnidiospores in high relative humidity condition; **f** Pycnidiospores

The mega-environment classification of the International Maize and Wheat Improvement Center (CIMMYT) has identified STB as the main breeding target in at least one-third of the total spring wheat growing areas of developing countries in Central and Western Asia, North Africa (CWANA) and Latin America (Duveiller et al. 2007). The incidence of STB on winter wheat is particularly high in colder climates with high rainfall at higher altitudes. Europe, Russia, Australia and New Zealand are also classified as high-risk regions for STB (Chawade et al. 2018; Odilbekov et al. 2018; Pastircak 2005; Polley and Thomas 1991; Sanderson 1976).

Both spring and winter cultivars suffer variable yield losses depending on seasonal and regional conditions, cultivar susceptibility, crop history and management (Hardwick et al. 2001). Linear and exponential regression analysis models showed that yield loss was highly correlated with STB severity on the flag and flag-1 leaf at GS 75 in winter wheat (King et al. 1983). The combined yield penalty of *Z. tritici* and *S. nodorum* is reported to be 35% per year (Jenkins

and Morgan 1969). Comparative fungicide experiments indicate that STB damage alone ranged from 8 to 18% in spring wheat and 10–25% in winter wheat, and can easily increase to 50% during epidemics at field level (King et al. 1983). During 1985–1989, total yield losses in England and Wales were estimated at 329 million tons per year amounting to an economic loss of 40 million Euros per year (Cook et al. 1991). This was confirmed for the entire UK in 1998, a year with a unique and dramatic disease incidence primarily due to STB (Hardwick et al. 2001). Deployment of susceptible varieties in UK recorded 20% of yield losses in average in 2012–2013, while planting resistant cultivars and spraying fungicide reduced yield losses in the range of 5–10% (Fones and Gurr 2015).

Disease control

Management of STB has been mainly done through chemical control, but the host resistance is increasingly considered as a crucial management strategy to minimize STB yield penalties. Fungicides have been used for over 200 years to protect small grain cereals, but the demand has significantly increased after the Second World War, due to the availability of a greater variety of crops and fungicides (Morton and Staub 2008).

The copper and sulfur-based fungicide formulations controlled the disease from 1940s to 1980s. Sterol demethylation-inhibiting (DMIs) fungicides replaced these until the early 1990s (Fraaije et al. 2003). STB and glume blotch (caused by *S. nodorum*) control commenced in 1964 in Western Europe. Overtime, STB increased in importance and is currently the main target of the agrochemical and breeding industry (Goodwin et al. 2011). In 1997, Quinine outside Inhibitors (QoI) were introduced and largely replaced DMIs for STB management. However, contrary to the expectations, fungicide resistance rapidly developed and disseminated over Europe (Torriani et al. 2009). Therefore, STB management is currently virtually entirely azole based (imidazoles and triazoles; DMIs), with imminent risks on fungicide resistance development and consequently reduced efficacy of STB control (Cools and Fraaije 2008). *Boscalid* was the first carboxamide succinate dehydrogenase inhibitor (SDHI) that registered for application in 2003 (Hahn 2014). This type of fungicides prevent succinate dehydrogenase (Sdh) respiratory chain (complex II) of mitocondri and SDHI fungicides resistance has not been reported yet (Fraaije et al. 2012). Integrated pest management programs enabled the development of decision support systems that optimized fungicide applications, thus responding to increasing economic and environmental demands (te Beest et al. 2009; Wiik and Rosenqvist 2010). Currently, national pesticide reduction programs and European legislation further delimit fungicide applications (Sande et al. 2010). This contributed to priority setting for the cereal market with increasing emphasis on the identification and deployment of breeding of host resistance to control STB (Jorgensen 2008). Fungicide application during the flag leaf stage increased grain yield around 1.5% in association with green leaf area extension in UK. Although, average of grain crude protein concentration had a negative response particularly by applying fungicide against *Z. tritici* (Gooding 2007).

Breeding strategies

Plant disease epidemic will occur when there is a susceptible cultivar, a virulent pathogen, favorable environmental conditions and adequate time for pathogen growth and activity, which is known as a disease epidemic pyramid model (Madden 1987). To protect plants from abiotic and biotic stresses; avoidance, escape, tolerance and resistance are the four main breeding strategies. In plant-pathogen interaction, avoidance is a passive resistance mechanism to genetically control plant traits to reduce host and pathogen contact (Alexander 1992; Bowers et al. 2001), while disease escape occurs whenever the epidemic pyramid factors do not coincide and interact appropriately (Agrios 2005). Tolerance describes the ability of an infected cultivar to maintain economic yield production (Agrios 2005). Resistance can be characterized either by non-host resistance known as microbial- or pathogen-associated molecular patterns (MAMPs/PAMPs) or by the gene-for- gene concept, when a host resistance gene product interacts with its corresponding avirulent gene product from the pathogen, resulting in a minimal or no disease symptoms on the plant by controlling pathogen growth in or on the plant tissue surface (Flor 1971; Jones and Dangl

2006). Grain yield reduction in tolerance approach is inevitable as the pathogen organs can penetrate inside the plant and contribute in assimilate consumption, and incidence of disease escape is conditional by breeding early or late mature germplasm to manage mismatch of epidemic pyramid factors resulting losses a part of optimum growing season and potential yield. Also, plant architectural modification to avoid or even minimize contact between host and pathogen needs a multi-trait breeding program, therefore breeding for resistance is the most efficient fast forward approach to reach the expected yield potential in the locations where *Z. tritici* is a major concern.

Gene for gene concept in wheat-*Zymoseptoria tritici* interaction

Gene for gene concepts in host–pathogen interactions are basal for co-evolutionary resistance gene and pathogenicity effector evolvement. *Z. tritici* is a high-risk pathogen due to its biology. It frequently undergoes sexual/asexual reproduction (Ponomarenko et al. 2011), it has spore dissemination strategies that favor gene flow and therefore easily circumvents resistance genes (Linde et al. 2002). The wheat-*Z. tritici* pathosystem mainly adapts with the gene-for-gene concept that is known as pathogen effector and host target gene interaction (Brading et al. 2002). So, natural *Z. tritici* populations can circumvent new *Stb* genes under disease pressure (Linde et al. 2002). This calls for a continuous effort to unveil new or wide range resistance genes to control this disease.

Reported *Stb* genes, application and limitation

Thus far, in contrast to the hundreds of resistance genes identified for other cereal diseases and pests, only 21 resistance genes (*Stb*) have been identified for STB (Table 1) (Brown et al. 2015). These genes have been mapped mainly in bread wheat (except *TmStb1* that sourced by *Triticum monococcum*), but dramatic severity of STB on durum wheat, particularly in the Mediterranean region, resulted in identification of new resistance sources in durum wheat germplasm (Ferjaoui et al. 2015).

Narvaez and Caldwell (1957) published the first genetic study of wheat resistance to STB. Subsequently, resistance genes *Stb1–Stb4* were identified and later mapped (Adhikari et al. 2004a, b, c; Wilson 1979, 1985). Arraiano et al. (2001a, b) characterized *Stb5* in a synthetic hexaploid line that provided broad resistance to at least 12 *Z. tritici* isolates. The discovery of the mating system in *Z. tritici* (Waalwijk et al. 2002) resulted in the formal genetic proof of an operational gene-for-gene interaction in the wheat-*Z. tritici* pathosystem. This further enabled the identification of a range of additional *Stb* genes, including *Stb6* (Brading et al. 2002) which is predominant among European wheat cultivars (Arraiano and Brown 2006). In the period of 2003–2011, a total of 12 additional resistance genes (*Stb7–Stb18*) have been characterized and mapped in spring and winter wheat cultivars (Table 1), but unfortunately, the efficacy of the above mentioned *Stb* genes is generally narrow (Ghaffary et al. 2012).

Resistance gene *Stb1* from the winter wheat cv. Bulgaria 88 is the first resistance gene that was commercially deployed in cvs. Oasis and Sullivan; providing long-lasting resistance to STB in the Midwest of the United States (Goodwin 2007). The Brazilian cv. Veranopolis carries *Stb2* and was released in 1950 and deployed as a progenitor of other wheat cultivars such as cvs. Cotipora, Lagoa-Vermelha, Nova Prata and Vacaria (Kohli and Skovmand 1997; Prestes and Hendrix 1975; Wilson 1979). The breeding line Israel 493 carries *Stb3* (Wilson 1979), but there is no official report on its commercial deployment (Goodwin 2007). Cultivar Tadinia carries the resistance gene *Stb4*, and is a derivative of a cross between the Dutch cv. Tadorna and Inia 66. It was introduced as a commercial cultivar in 1985 in California with adequate resistance to STB that lasted almost 15 years (Jackson et al. 2000). *Stb5* was described in the Chinese Spring/Synthetic hexaploid substitution line of chromosome 7D that presented resistance to 12 of the 13 tested *Z. tritici* isolates (Arraiano et al. 2001b), providing a relatively broad resistance that is however, not yet commercially applied. *Stb6* was firstly described in the cvs. Shafir and Flame and was later identified in a range of cultivars suggesting that it is among the most widespread *Stb* genes in the contemporary wheat breeding programs (Arraiano and Brown 2006; Chartrain et al. 2005b; Kema et al. 2000; Kema and van Silfhout 1997). Another predominant gene *Stb7* derived from the cross EHRO (Estanzuela-Horenero (Novafen/

Table 1 Genes for resistance to Septoria tritici blotch (STB) of wheat that have been reported in winter and spring wheat cultivars along with their chromosomal positions and associated molecular markers

Stb genes	Cultivars source	Chromosomal position	Closest(Flanking‡) marker	Marker type	Resistance allele size (in base pairs)	Distance between gene and markers (cM)	References
Stb1	Bulgarai 88 *	5BL	*Xbarc74*	SSR	188	2.8	Adhikari et al. (2004c)
Stb2	Veranopolis *	1Bs	Xwmc406 Xwmc230	SSR		6 5	Adhikari et al. (2004b) and Liu et al. (2013)
Stb3	Israel 493 *	7As	Xwmc83	SSR			Brown et al. (2015), Goodwin and Thompson (2011)
Stb4	Tadinia *	7Ds	*Xgwm111*	SSR	210	0.7	Adhikari et al. (2004a)
Stb5	Cs Synthetic 6X (7D)*	7Ds	*Xgwm44*	SSR		7.2	Arraiano et al. (2001b)
Stb6	Shafir Flam	3As	*Xgwm369*	SSR	197	2	Brading et al. (2002)
Stb7	Estanzuela Federal	4AL	*Xwmc313*	SSR	206	0.5	McCartney et al. (2003)
Stb8	W7984 ‡	7BL	*Xgwm146* *Xgwm577*	SSR	200 160 and 200	3.5 5.3	Adhikari et al. (2003)
Stb9	Courtot‡	2B	*XksuF1* *Xfbb226*	RFLP	1550 7350	9 3.6	Chartrain et al. (2009)
Stb10	KK4500 †	1D	*Xgwm848*	SSR			Chartrain et al. (2005a)
Stb11	TE9111 †	1Bs	*Xbarc008*	SSR	275		Chartrain et al. (2005c)
Stb12	KK4500 †	4AL	*Xwmc313; Xwmc219*	SSR			Chartrain et al. (2005a)
Stb13	Salamouni	7BL	*Xwmc396*				USDA-Annual wheat news letter volume 53
Stb14	Salamouni	3Bs	*Xwmc500*				USDA-Annual wheat news letter volume 53
Stb15	Arina *‡	6As	*Xpsr563a* *Xpsr904*	RFLP		22 14	Arraiano et al. (2007)
StbSm3	Salamouni	3As	*Xbarc321*	SSR		1.9	Cuthbert (2011)
Stb16	M3	3DL	*Xgwm494* *Xbarc125*	SSR		2 8.8	Ghaffary et al. (2012)
Stb17	M3	5AL	*Xhbg247*	SSR		3.1	Ghaffary et al. (2012)
Stb18	Balance*‡	6Ds	*Xgpw5176* *Xgpw3087*	SSR		5 3.6	Ghaffary et al. (2011)
StbWW	WW1842, WW2449, WW2451	1Bs	*Xbarc119b*	SSR		(0.9–4.1)	Raman et al. (2009)

Table 1 continued

Stb genes	Cultivars source	Chromosomal position	Closest(Flanking‡) marker	Marker type	Resistance allele size (in base pairs)	Distance between gene and markers (cM)	References
TmStb1	MDR043 (*T. monococcum*)	7Ams	*Xbac174*	SSR		23.5	Jing et al. (2008)

Klein-Impacto)/CNT8 (IAS 20/ND 81) (GRIPI) and selected from cv. Estanzuela Federal, was firstly identified in the Uruguayan line ST6 (McCartney et al. 2003), and later in cvs. KK4500 and TE9111 (Chartrain et al. 2005a, c). The International Triticeae Mapping Initiative (ITMI) population is developed from a cross between cv. Opata85 and the synthetic hexaploid derived line W7984, which carries *Stb8* (Adhikari et al. 2003; Roder et al. 1998). Hence, W7984 has been deployed in the development of marker assisted selection (MAS) programs (Varshney et al. 2007), but thus far not in commercial wheat breeding for resistance to STB. The gene *Stb9* is discovered in the French winter wheat cv. Courtot as well as the British spring wheat cv. Tonic (Chartrain et al. 2009). The breeding line Kavkaz-K4500 L.6.A.4 (KK4500) developed at CIMMYT and is derived from winter wheat cvs. Kavkaz and Frontana, which originated from Russia and Brazil, respectively (Eyal 1999). KK4500 is an important international source of resistance to STB and genetic analysis indicates that it carries *Stb6*, *Stb7*, *Stb10* and *Stb12* (Chartrain et al. 2005a), suggesting that gene pyramiding is an effective strategy for STB resistance breeding. STB resistance in the Portuguese line TE9111 was studied and carries resistance genes *Stb11*, *Stb7* and *Stb6* (Chartrain et al. 2005c). Four adult plant resistant STB QTLs were reported on chromosomes 1BS, 3AL, 5AL and 7AS, and two of them 1BS and 7AS, are likely associated with *Stb3* and *Stb11* genes, respectively (Dreisigacker et al. 2015). *Stb13* and *Stb14* are described in cv. Salamouni (USDA-Annual wheat newsletter volume 53) and *Stb15* was reported in the Swiss cv. Arina and could also be present in the British cv. Riband (Arraiano et al. 2007).

Stb16 and *Stb17* are two reported resistance genes to STB, derived from the synthetic hexaploid wheat line M3. The former widely protects wheat in both seedling and adult plant stage, while the later only expresses in adult plant stage (Ghaffary et al. 2012). The broad resistance spectrum of *Stb16* was investigated and recently unraveled a new class of R gene in plant pathogen interaction (Saintenac et al. 2017). *Stb17* originating from a tetraploid durum wheat line was used in the development of M3 (Cando/R143//Mexi'S'/3/*Ae. tauschii* (C122)). *Stb17* is the first specifically adult plant resistance gene reported by Ghaffary et al. (2012) for wheat-*Z. tritici* pathosystem. This complies with APR genes that are common to other cereal diseases such as rusts (White and Frommer 2015).

Stb18 has been detected in the French winter wheat cv. Balance, flanked by the SSR markers *Xgpw3087* and *Xgpw5176*, and is located on chromosome 6DS. This is an isolate specific resistance gene that was detected with the French *Z. tritici* isolates IPO98022 and IPO98046 and with the Dutch isolates IPO89011 and IPO323. Isolate IPO89011 detected *Stb18* at the seedling stage, whereas IPO323 identified it in both the seedling and adult plant stages (Ghaffary et al. 2011).

Generally, application of resistance sources of wheat germplasm in breeding program is narrow, mainly because it has emerged as a major foliar wheat disease since 1970 when other diseases like rust and powdery mildew were dominant (Forrer and Zadoks 1983). Hence, the knowledge of *Stb* genes and their applications in wheat breeding is relatively limited. Moreover, due to the lack of near isogenic lines with individual *Stb* genes, the differential set of cultivars with single or multiple mapped *Stb* genes (Table 1) have been replaced in phenotypic evaluation. Based on gene-for-gene concept in *Z. tritici*-wheat interaction, each single *Stb* gene can resist against the strain carrying avirulent factor in a specific differential cultivar x *Z. tritici* isolate interaction, however, conclusion of effectiveness of each *Stb* gene remains unclear in a multi *Stb* gene cultivar (Ghaffary et al.

unpublished data). *Stb* gene interaction with additive and epistatic effects also complicate the role of each gene in the resistance expression against a given set of isolates (Ghaffary et al. 2011). Although, the clear message in applied breeding refer to *Stb* gene stacking, which results in durable and effective STB management, the identification of new *Stb* genes and their accumulation in germplasm will significantly contribute to STB management. This is also illustrated by the fact that the majority of differential cultivars contain a broad resistance spectrum (Chartrain et al. 2005a; Ghaffary et al. 2011). This can strongly benefit the development of cultivars with durable resistance.

Effective breeding strategies to identify more *Stb* genes

To identify new sources of resistance to STB, breeders need a comprehensive breeding strategy. Here we provide a step-by-step standard breeding approach to discover new resistance *Stb* genes, including evaluation of parental lines using a broad range of isolates, validation of map using wheat map databases and QTL analysis using as many as possible polymorphic isolates.

Screening parental lines along with a differential set of cultivars

For screening purposes, it is essential that *Z. tritici* isolates are well characterized. The best procedure is to phenotype a *Z. tritici* strain on a set of isogenic lines. These are, however, not available and thus the next best option is to screen isolates on wheat cultivars with mapped *Stb* genes. After initial analyses, 21 *Stb* genes were identified and mapped with well-characterized *Z. tritici* isolates (Table 1). An analysis by careful characterization of the pathogenicity patterns of 50 isolates on 98 wheat accessions the differential set provided a unique informative outcome of the virulence of the isolates on known *Stb* genes (Ghaffary et al. unpublished data). These isolates and their virulence expression on known *Stb* genes were later used to test recombinant inbred line (RIL) or double haploid (DH) mapping populations that resulted in the identification of three new *Stb* genes (Ghaffary et al. 2011, 2012). This effort should be continued in order to monitor the emergence of new pathogenic *Z. tritici* variants.

Mapping

A valid QTL analysis depends on accurate mapping and phenotyping process. It needs to validate both the position and orientation of markers on the linkage group. Polyploid wheat species (tetra and hexaploid) originated from inter-specific hybridization of wild diploid wheat progenitors (Dubcovsky and Dvorak 2007) and resulted in a high similarity of gene content and order in the A, B and D hexaploid wheat genomes (Dvorak et al. 2006). Such similarity can be genetically mapped to a portion of molecular markers in more than one position over wheat genomes (Song et al. 2005). To avoid any problem in the mapping procedure, the reported mapped location of known SSR, DArT and other type of markers should be linked to the marker's name prior to linkage mapping in new projects. This approach would facilitate to choose the right LOD value in grouping tree navigation and increase accuracy of constructed linkage groups by monitoring the chromosomal location of markers. It is observed that the mapping software users simply choose LOD 3 to construct a linkage group, while the aligned markers belong to different chromosomes. False positive mapping is precedent in wheat-*Z. tritici* pathosystem (Goodwin 2007; Liu et al. 2013). Here we suggest tagging each marker to its position to facilitate choosing the correct LOD value for linkage group construction when at least the majority-linked markers are considered being on the same chromosome. Moreover, we strongly suggest comparing the constructed genetic map and publicly available map databases in order to validate the map orientation of constructed linkage group (Ghaffary et al. 2011).

Phenotyping for QTL

Various qualitative and quantitative phenotyping scales were used over the years. In some reports both necrosis (*N*) and pycnidia (*P*) were quantitatively scored (Kema et al. 1996a), while others only scored *P* (Arraiano et al. 2001a; Chartrain et al. 2009). A combined qualitative/quantitative assessment method evaluating disease severity as the leaf area with pycnidia bearing necrosis along with the level of sporulation (a variation on the earliest qualitative 0–5

Necrosis %	100	80	75	65	60	60	45	40	25	20	5	0
Pycnidia %	100	40	70	30	50	15	20	0	0	0	0	0

Fig. 2 Scoring pattern of estimating the leaf area bearing necrosis and pycnidia

scale for STB phenotyping) has also been used (Adhikari et al. 2003; McCartney et al. 2003). In fact, all reported *Stb* genes were identified by different scoring methods in either attached or detached leaf assays (Arraiano et al. 2001a; Kema et al. 1996a). A combination of the attached/detached leaf technique was also applied to induce sporulation in overall symptomless responses of the diploid *T. monococcum* (Jing et al. 2008). This evidently is far from ideal and hampers effective introgression of *Stb* genes into breeding programs, particularly as these programs most often rely on field studies using specific isolates and accompanying marker assisted approaches (Goodwin 2007). Ghaffary et al. (unpublished data) evaluated a vast array of interactions using *Stb* differentials to validate *Stb* efficacy, providing a new starting point for *Stb* gene and extending previous knowledge that tested *Stb1–15* differentials in an attached leaf assay for both *N* and *P* using one scale. The *Stb16*, *17* and *18* genes in 'M3' and the French wheat cv. Balance were successfully characterized by exploiting *N* and *P* criteria in detailed mapping studies (Ghaffary et al. 2011, 2012). This confirmed the value of deep screening studies using percentage of leaf area bearing necrosis (*N*) and pycnidia (*P*) along with application of synthetic wheat and biological inducers to identify new sources of resistance (Fig. 2).

Pre-screening of the RIL and DH populations for QTL analysis

With the aim of increasing the possibility of detecting new *Stb* genes, even in modern breeding lines and cultivars, we propose to screen the RIL or DH populations using as many isolates as possible. Hence to identify distinct response of parental lines, we should screen them using a broad spectrum of isolates to find the best candidate stain for phenotyping of populations. The best example refers to the phenotyping of Apache/Balance DH and Kulm/M3 RIL populations, which were tested using eight and four *Z. tritici* isolates, respectively. These isolates were candidate by screening of 30 against Apache-Balance as well as 20 isolates against Kulm M3 parental lines. A pre-screening (phenotyping) of populations for QTL analysis indicated overlapping detection of QTL in which a QTL detected by more than one isolate. Hence, to avoid repetitive screening, the number of isolates were narrowed down based on QTL/linkage group position for final QTL analysis to five and two,

respectively. This process maximized QTL identification possibility with flexible selection of isolates that can induce the highest LOD per QTL (Ghaffary et al. 2011, 2012). This is a novel approach with a worthwhile message for practical breeding programming not only for resistance to *Z. tritici* but also for other biotic stresses.

Marker assistant selection (MAS) for resistance to STB

Associated *Stb* gene markers can be used for characterizing new breeding materials. Resistance to standard isolate IPO323 (avirulent on *Stb6*) is common within wheat germplasm with broad allelic variation (143–299 base pair) (Chartrain et al. 2005b). Ghaffary et al. (unpublished data) reported 12 alleles varying between 143 and 212 base pair for the *Stb6* associated SSR marker *Xgwm369*. The resistant check Shafir and other resistant cultivars and lines were categorized in the group of 196–212 bp allelic sizes. Similar results were observed for *Stb4* SSR marker *Xgwm111* with 11 alleles varying between 161 And 232 bp in length. Standard *Stb4* carrier cultivar (Tadinia), categorized with a group of 222 or 222/224 bp. Maximum phenotypic/genotypic match for SSR marker *Xgwm111*, was detected in the plant material, with allelic size of 204–230 bp. Allelic variation was reported previously for the markers associated to *Stb6* and *Stb4* (Adhikari et al. 2004a; Chartrain et al. 2005b). Allelic variation for the markers associated with the *Stb* genes and distance between a *Stb* gene and the closest marker (Table 1) calls for a specific strategy integrating MAS (application of the primer pairs of marker) and phenotyping screening with avirulent *Z. tritici* isolate specific to each *Stb* gene. This strategy can narrow down the breeding material first based on potential resistance genes by MAS, and then by resistance screening for the specific *Z. tritici* isolates.

Omics

Genome sequencing of wheat and *Z. tritici*

Availability of the genome sequence has expedited identification of key genes and alleles. The draft genome sequence of the 17-gigabase hexaploid wheat (*Triticum aestivum*) was released in 2012 using whole-genome shotgun sequencing and between 94,000 and 96,000 genes were identified in the assembly (Brenchley et al. 2012). In 2014, sequencing of individual chromosome arms was done by the International Wheat Genome Sequencing Consortium (IWGSC) and annotated 124,201 genes across the three genomes (Mayer et al. 2014). Reference sequence of 1-gigabase chromosome 3B was done using bacterial artificial chromosomes and released in 2014 with annotations for 5326 protein-coding genes (Choulet et al. 2014). Genome sequencing of the tetraploid wheat (*T. turgidum*) cultivars Cappelli and Strongfield is also available from the IWGSC sequence repository at URGI. Higher depth reference sequencing of individual chromosomes is currently ongoing and the status is regularly updated on the IWGSC homepage (www.wheatgenome.org). Together, these genomic resources will enable fast tracking of identification of the key resistance genes for STB resistance in tetraploid and hexaploid wheat.

The 39.7-Mb genome of *Z. tritici* has 21 chromosomes and was sequenced from the isolate IPO323 and released in 2011. In total, 19.933 genes were identified and 6111 were annotated. These resources will facilitate fine mapping of key resistance genes in the host and avirulence effector genes in the pathogen.

Discoveries from omics studies

Z. tritici is a hemibiotroph with symptomless invasion and growth in the wheat apoplastic regions during the initial phases of the invasion. Around 10–13 days post infection (dpi) (Keon et al. 2007), chlorotic followed by necrotic symptoms appear on the wheat leaves (Fig. 1). In order to understand the wheat-*Z. tritici* molecular interaction under various stages of infection, transcriptomics, metabolomics or proteomics approaches were adapted in different studies. Rudd et al. (2015) performed transcriptomics by RNAseq and metabolomics analysis of the *Z. tritici* invasion of the wheat and identified over 3000 *Z. tritici* genes, 7000 wheat genes and 300 metabolites differentially expressed during the course of the invasion. An Increased number of pathogen transcripts were identified in the samples during the course of the infection, indicating colonization of the host by the pathogen and at 21 dpi, over 80% of the identified transcripts belonged to the pathogen.

Given the fact that *Z. tritici* is localized in the apoplastic region during the asymptomatic phase, identifying secreted proteins in the apoplastic region could improve our understanding of the interactions during the early phase of infection. Yang et al. (2015) performed mass spectrometry based proteomics to identify key secreted proteins in the early phase of infection. The results from the work demonstrated that the plant resistance to *Z. tritici* is correlated with cell wall remodeling, changes in carbohydrate metabolism and an increase of PR proteins in the apoplast. The pathogen overcomes the host defenses by detoxifying reactive oxygen species (ROS) to colonize the plant cell.

Comparative transcriptomics analysis of virulent strains can reveal core virulence factors and strain-specific genes underlying quantitative virulence. Recently, comparative transcriptomics was performed on four strains differing in virulence to a single susceptible wheat cultivar Drifter (Palma-Guerrero et al. 2017). Conserved transcription profiles were identified among strains for proteases and lipases while significant differences in the expressions of genes for small secreted proteins and secreted peroxidases. Overall, the comparative transcriptomics study revealed core genes that are important for virulence in all strains and genes that explained the differences in the virulence of different strains. Recently, by combining QTL analysis and GWAS, the avirulence gene corresponding to the major wheat resistance gene *Stb6* was identified (Zhong et al. 2017). The avirulence gene was named *AvrStb6* and was found to be highly polymorphic given the selection pressure from the wheat cultivars carrying *Stb6* resistance gene. Thus, various omics techniques have shown a tremendous promise in identifying novel resistance genes, susceptibility factors and avirulence genes from the pathogens that are interacting with the resistance genes. Further developments in the next generation techniques, data processing pipelines (Chawade et al. 2015) and multivariate analysis (Chawade et al. 2016) is expected to fast track these novel discoveries leading to improved resistance of wheat cultivars to STB.

From genomics to the field

Resistance to *Z. tritici*, and *Stb* genes expression is not always similar at the seedling and adult plant stage. In contrast to *Stb16*, which is effective in both seedling and mature plants, *Stb17* is only functional at the adult plant stage (Ghaffary et al. 2012). Partial resistance and contribution of disease escape (plant earliness that affects the coincidence of epidemic factors) and specific resistance have already been suggested as two breeding strategies to control STB on adult plants (Arraiano et al. 2009; Chartrain et al. 2004). Moreover, crop structure, canopy architecture and position of inoculum in crop canopy, which is controlled by plant genome (avoidance), affect the risk of STB epidemics in wheat.

A resistance QTL was identified on chromosome 2DS with the SSR marker *Xgpw332* and was exclusively and consistently detected throughout all adult plant tests in 2007 and 2008 at two trial locations in the north of France (Ghaffary et al. 2011). It was also significantly correlated with earliness (− 0.48 and − 0.25, $P = 0.05$ in Florimond Desprez and Serasem, respectively), tallness (− 0.36, $P = 0.05$ at Serasem) and resistance to FHB. Subsequent regression analyses that fitted means of logit transformed STB values on earliness and tallness left no residual STB resistance effect for the 2D locus ($p = 0.359$) (Ghaffary et al. 2011). Therefore, based on this information, it was not possible to assign 2D QTL as a new STB resistance gene and it was suggested that it indirectly influences STB resistance by regulating earliness and tallness that are known to affect STB severity (Arraiano et al. 2009). The associated SSR marker *Xgpw332* is also associated with *Rht8* and *Ppd-D1* that are involved in the regulation of wheat tallness and earliness (Korzun et al. 1998). Previously, Handa et al. (2008) identified a possible multidrug resistance associated protein (MRP) at this 2D chromosomal location that is involved in the wheat-Fusarium interaction.

Climate change and future STB impact

Given the present status on food security, it becomes increasingly important to close the gap between the potential yield (PY) and farmer yield (FY). Main factors responsible for this are the biotic and abiotic stresses (Fischer and Edmeades 2010). Heat stress is one of the major abiotic stresses that is expected to be more severe by the end of the current century, exceeding the extreme seasonal temperatures recorded from 1900 to 2006 with high probability that it would

damage the food system and security (Battisti 2009). Damage would be directly linked to abiotic stresses or indirectly because of changes in pathogen, insect and weeds pattern. An investigation during 1988–1990 estimated almost 243 billion US$ annual financial losses caused by pathogens, insects and weeds on the eight most important agricultural crops all over the world or 72% of total production value (Oerke et al. 1995). Temperature, light and water are the major factors controlling growth, development, the proliferation of biotic stress agents and their temporal/spatial distribution (Rosenzweig et al. 2001). Most analyses illustrated that in a warmer climate, pathogens, insects and weeds will have a major impact on crop production resulted by more activity in higher temperature as well as wider range of geographically distribution of pathogens, insects and weeds (Roos et al. 2010). The climate change also alter the disease pattern as well.

Mega-environments are wide, usually noncontiguous or transcontinental areas with similar biotic or abiotic stresses, cropping pattern and consumer habits were proposed by CIMMYT to characterize major wheat breeding objectives. Comparison between this classification overtime illustrated a conversion in the disease resistance breeding objects within each mega-environment (Lantican et al. 2005). Analysis of disease combined model on wheat samples, over 160 years indicated periodical shifting within *Septoria* species (*Z. tritici* and *S. nodorum*). The ratio of the pathogens varied and highly correlated with SO_2 emissions measured as the atmospheric pollution factor (Bearchell et al. 2005). The reviewed reports indicated dynamic ability of the pathogen to overcome environmental effects or being replaced by other pathogens. Pathogen adaptation usually happens under environmental pressure on pathogen resulted point mutation or adapted recombinant selection within progeny derived from sexual reproduction (Zhan and McDonald 2013). In addition, lateral transfer between pathogens resulted switching disease agents from one to another host (Stukenbrock and McDonald 2008). By these strategies, pathogens can live in diverse environments over the globe (Zhan and McDonald 2011). In a symmetrical defense strategy, breeders should follow and focus on the offensive smart approaches of pathogen i.e. increasing genetic diversity within host genome. Currently, narrow genetic diversity in wheat has been found as a consequence of green revolution. Breeding for mega-environment leads to an intensive line and cultivar selection with wide general adaptation. This approach, however, results in erosion within wheat germplasm and dramatically dropped genetic diversity. Going back to breeding for nano-environments and exclusive area specific adaptation with higher authority for local breeders perhaps will be the near future breeding strategy to support food security all over the world.

Conclusion

STB incidence will continue to increase due to the switch from the conventional to the conservation agriculture with emphasize on keeping crop residual and reduction in tillage (Mergoum et al. 2007), increase in pathogen virulence spectrum of recombinant strains derived from sexual reproduction (Zhan et al. 2007) and social and political demand for limitation on fungicide application (Gullino and Kuijpers 1994; Ragsdale and Sisler 1994). In addition, emerging of fungicide resistant isolates and potential impact of climate change on regional as well as global disease models can significantly modify geographical disease distribution including STB in near future (Juroszek and von Tiedemann 2013).

Holistic management is emphasizing on host resistance either using wide range resistance gene like *Stb16* or *Stb* gene staking to extend resistance spectrum of breeding materials against STB (Ghaffary et al. 2011, 2012). Integrating disease management also needs appropriate agronomic interventions and efficient use of fungicides (Jørgensen et al. 2014), for instance, mixing and deployment different fungicide calsses (Qols,SDHIs and DMIs) to maximize disruptive selection of new fungicide resistant mutatnt race. Here we standardized genetic studies in a few efficient steps (1) working with wide genetic and geographical range of isolates, (2) testing the resistance spectrum of individual RILs or DH lines to a broad(er) set of isolates and (3) validate marker positions with publicly available wheat maps. The latter is much more important and is an obligation to avoid erroneous *Stb* positions for polyploid wheat species originated from interspecific hybridization of wild diploid wheat progenitors (Dubcovsky and Dvorak 2007) that resulted in a greatly similar gene order and content of the A, B and D homeologous chromosomes

(Dvorak et al. 2006). This may practically even result in multiple marker positions on the wheat genomes (Song et al. 2005). To ascertain map positions, the reported positions of SSR and DArT markers should be extracted from publicly accessible wheat map databases such as INRA-Genoplant (2011), Triticart wheat map (2011) and GrainGenes (2011)—and before using the mapping software each polymorphic marker should be labeled with its position. This approach facilitates the choice of appropriate LOD values and increases the accuracy of constructed linkage groups by monitoring the map alignment and chromosomal location of the markers. Hence the constructed map confidently can apply in QTL analysis to report detected associated QTLs. Embracing these guidelines enables the selection of lines with individual *Stb* genes and will greatly contribute to a sound characterization of *Z. tritici* isolates and in turn to improved QTL analyses in wheat which will greatly support practical breeding for STB resistance (Ghaffary et al. 2011).

Due to the fact that most studies have addressed bread wheat cultivars, there is an urgent need to launch a similar program for durum wheat. It can be broadly stated that the majority of the well-characterized *Z. tritici* strains with specific virulence for mapped *Stb* genes are useless in durum wheat screens as the far majority is avirulent on these tetraploids (Kema et al. 1996b). Hence, durum wheat breeding for STB resistance has to start from scratch, unless we are able to translate the advanced know-how from the bread wheat pathosystem to durum wheat by designing new phenotyping protocols. For any analyses, it is essential to study biparental mapping populations with such a suite of isolates rather than single isolates to verify the efficacy of individual resistance factors to STB. This then also contributes to effective isolation of individual *Stb*genes in segregating DH or RIL populations that can be used as differential lines and eventually can replace the current *Stb* 'differentials'. This would strongly contribute to improved phenotyping of *Z. tritici* strains, certainly with an eye on the massive investment in such tools in cereal rusts research (Ferjaoui et al. 2015; Goodwin 2007; White and Frommer 2015).

Throughout the history of wheat research aiming at cereal disease improvement, wild relatives have been considered as very valuable resources for new resistance genes. Within reported resistance genes, *Stb5* and *Stb16*, on the D genome in the synthetic hexaploid wheat lines have much broader resistance spectra than other *Stb* genes to the set of tested isolates (Arraiano et al. 2001b; Ghaffary et al. 2012). Recently, Wittenberg et al. (2009) and earlier Ware et al. (2007) reported sexual reproduction as a unique ability for genetic recombination in *Z. tritici* that simply results in massive genetically diverse progeny to cope with resistant cultivars and host specificity. However, tetraploid wheat are known as resistant to bread wheat derived *Z. tritici* isolates and vice versa (Kema et al. 1996a). This was confirmed by Ghaffary et al. (2011, 2012) in multiple phenotypic trials where none of the durum wheat-derived isolates were virulent on the tested bread wheat accessions including the susceptible check cv. Taichung 29. Hence, Synthetic hexaploid (SH) lines are predicted to be resistant to the adapted bread wheat *Z. tritici* isolates, the D genome component, however, can affect the resistance expression, which has previously been shown for rust diseases (Kema et al. 1995). Introgression of D genome to the tetraploid wheat and synthetic breeding approach seems much more efficient to breed resistance to STB than other wheat diseases. Broad-spectrum resistance to *Z. tritici* (99% of 194 accessions) in seven *Aegilops* species was reported by Assefa and Fehrmann (1998), in contrast only 8, 11, 16 and 24% of the evaluated germplasm was resistant to stem rust, leaf rust, eyespot and powdery mildew, respectively. Similar wide range resistance was detected in phenotypic screens of the diploid wheat *T. monococcum*, which resulted in the identification of the resistance locus *TmStb1* linked to *Xbarc174*SSR marker on chromosome $7A^m$ (Jing et al. 2008). SHs derived from tetraploid and diploid genome combination of wheat progenitors and relatives (Yang et al. 2009), hence, they may have an arsenal of novel undetected genes for resistance to *Z. tritici* and other biotic stresses. Despite the value of *Stb5* and *Stb16* resistance genes that originated from SH line, *Z. tritici* populations exposure may potentially enable the fungus to finally break them down (Linde et al. 2002; Ware et al. 2007; Wittenberg et al. 2009; Zhang et al. 2007). Thus, their commercial deployment should consider their maximum efficacy in practical breeding programs using gene-pyramiding approach.

Ever since the elucidation of wheat evolution and domestication, breeders started to introgress material from wild relatives (Valkoun 2001; Zhang et al. 2009).

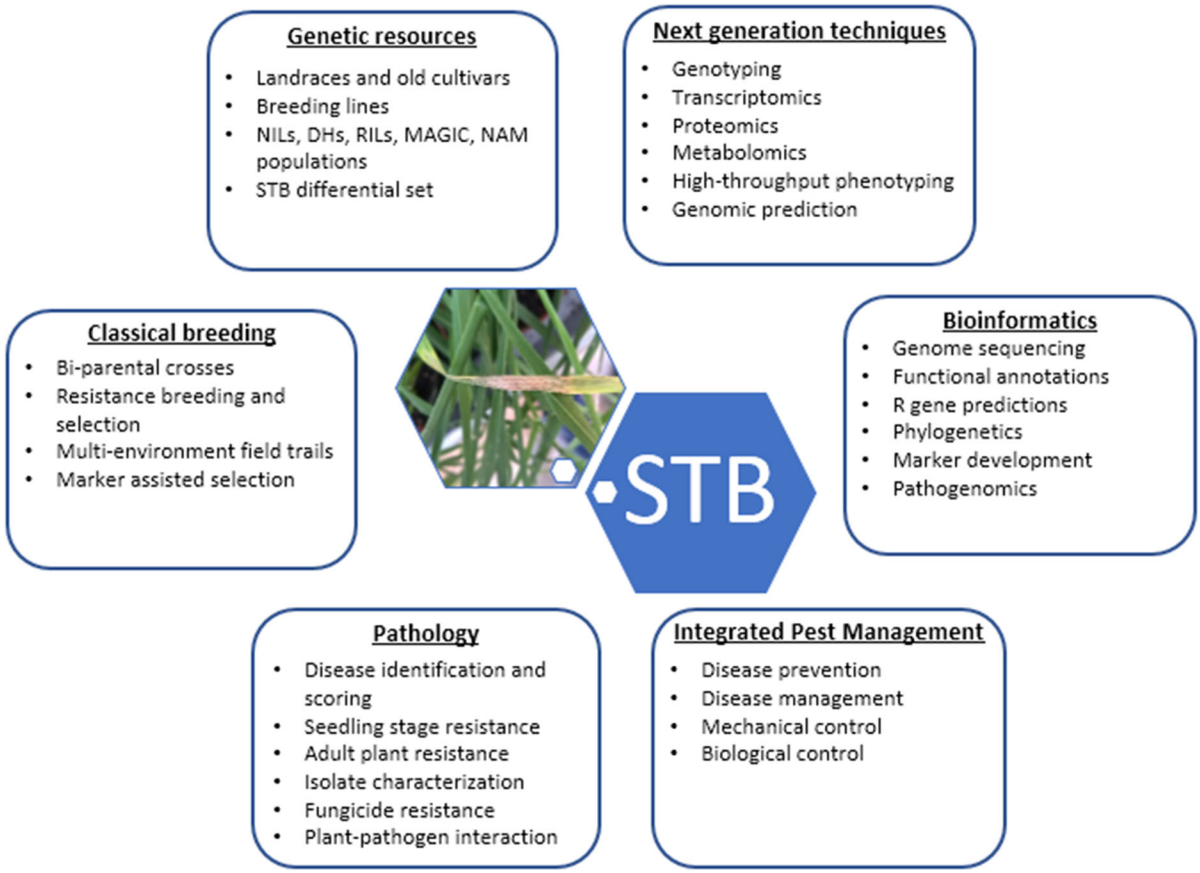

Fig. 3 Integrating various strategies to manage Septoria tritici blotch in wheat

Programs were started that crossed wild relatives and related grasses to bread wheat cultivars for gene transfer (Hajjar and Hodgkin 2007). Alternatively, synthetic hexaploids were developed that avoided structural chromosomal rearrangements and fertility problems in such gene enrichment programs (Mujeeb-Kazi et al. 2000, 2006, 2007; van Ginkel and Ogbonnaya 2007; Yang et al. 2009). This latter strategy has been increasingly and widely adopted since it enables the rapid transfer of genes from a broad gene pool by direct crosses with common wheat and, hence, such lines directly and significantly contribute to commercial breeding programs (Ogbonnaya et al. 2008; Warburton et al. 2006).

Interactions between QTLs, let alone QTL stacking as a strategy to develop broad resistance, particularly when marker assisted selection cannot be considered for all *Stb* genes as some of them map on the same position, like *Stb12* and *Stb7* (Chartrain et al. 2005a; McCartney et al. 2003) or too close to each other, such as *Stb4* and *Stb5* (Adhikari et al. 2004a; Arraiano et al. 2001b), but future studies should also address this issue that will serve the community.

Another important aspect of wheat-*Z. tritici* interaction refers to phenotyping and threshold between resistance and susceptibility. Too many times it is just an arbitrary threshold, which is not objective. Compared to the rust diseases, where agreed scales are being used, based on scientific evidence (McIntosh et al. 1995; McNeal et al. 1971), the threshold between compatibility and incompatibility in the wheat-*Z. tritici* pathosystem is hardly addressed (Kema et al. 1996d; Shetty et al. 2003, 2007, 2009). In general, the separation of resistant and susceptible plants in segregating populations was not transparent and only a few reports proposed arbitrary thresholds in different scales (Adhikari et al. 2003; Chartrain et al. 2005b; McCartney et al. 2003). It is urgently required to introduce an agreed methodology to phenotype populations, but it is even more difficult to propose

decisive methodologies for screening germplasm, which are not stable over geographical and temporal scales (Kema and vanSilfhout 1997; Shetty et al. 2009). In segregating populations, validation of QTLs can be easily addressed by defining (in)-compatibility by the extreme STB severity levels of plants with and without the co-segregating markers. This clearly depends on environmental situations and may differ over laboratories, but is founded in genetic facts (Ghaffary et al. 2012).

More research collaboration is needed to understand the global STB population structure and its ability to develop resistance to fungicide and increase its virulence. Enhanced efforts on developing genetic resources, applying current and on coming high-tech approaches, needed to develop durable STB resistant cultivars especially concerning adult plant resistance (Dreisigacker et al. 2015) (Fig. 3).

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Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

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