Wheat diseases: an overview

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1 Introduction

On average, about 20% of the global wheat production is lost due to diseases and pests every year. In 2012, these losses amounted to about 140 million tons, equivalent to about $35 billion (Anonymous, 2014). Fungal pathogens like rusts (*Puccinia* ssp.), *Septoria* leaf blotch (*Septoria* spp.), powdery mildew (*Blumeria graminis*) and *Fusarium* species are ranked among the top ten of the most important fungal pathogens (Dean et al., 2012). Historical and current sources report epidemics leading to sometimes devastating yield losses in wheat caused by these pathogens. In regions with low productivity where no seed dressing is conducted, smuts and bunts are also important pathogens (Oerke and Dehne, 2004). Furthermore, in specific wheat-growing areas, fungal pathogens such as *Pyrenophora tritici-repentis* causing tan spot, *Oculimacula* spp. causing eyespot of wheat or *Cochliobolus sativus* are of importance. One option to avoid yield losses caused by these pathogens is the application of fungicides. However, the repeated use of fungicides induces a considerable selection pressure on respective pathogens resulting in fungicide resistance or tolerance, which has been detected already in *B. graminis*, *Septoria* spp. or *Fusarium* spp. (Becher and Wirsel, 2012; Cools and Fraaije, 2013) to azoles mediated by mutations of the sterol 14-demethylase P450 (*CYP51*) or to strobilurins due to mutations in the cytochrome b gene (Torriani et al., 2009). The application of fungicides also depends to some extent on grain prices, fungicide costs and the possibility of applying these at the right time (Lopez et al., 2015). Hence, cultivars carrying resistances are the

http://dx.doi.org/10.19103/AS.2016.0004.19

most environment-friendly and cost-effective way of preventing yield losses in wheat. In particular, resistances against leaf rust, stripe rust, stem rust and powdery mildew infections result in complete resistance, but are at risk to be overcome by virulent isolates due to the extensive spore production of these pathogens. At a moderate infection level, 1 trillion stem rust spores per acre are generated (Fetch et al., 2011), and therefore, the occurrence of a virulent pathotype is very likely. In contrast, quantitative, race non-specific resistances encoded by genes like *Lr34* or quantitative trait loci (QTLs) are more durable and today molecular markers facilitate their effective combination (Ellis et al., 2014). Besides fungal pathogens, viruses are important pathogens of wheat, as *Triticum* species are natural hosts of more than 40 different viruses of which some are important pathogens. As viruses cannot be combated by chemicals directly, the only way of avoiding yield losses are taking chemical measures against their vectors, which in case of soil-borne vectors, are inefficient, and growing of resistant cultivars is the only way of ensuring wheat cultivation in the growing area of infested fields. In the following sections, the most important fungal and viral diseases of wheat and the possibilities of reducing yield losses caused by these are briefly described (for details cf. Bockus et al., 2010).

2 Fungal diseases of wheat: rusts

2.1 Leaf rust

Puccinia triticina causes the most common rust disease of wheat (Bolton et al., 2008). The percentage of germination and the percentage of infection of uredospores are higher for *P. triticina* over a wider range of temperatures than for *P. striiformis,* resulting in a worldwide distribution of *P. triticina* (de Vallavieille-Pope et al., 1995). Alternate hosts, which are essential for sexual recombination, are *Thalictrum* spp. (Ranunculaceae family) and *Anchusa italica* (Boraginaceae family). Dissemination of developed spores from these alternative hosts to susceptible wheat plants initiates infection, wherein a germ tube grows towards stomata cells, generating an appressorium. The fungus utilizes the intercellular space of leaves to form a substomatal vesicle and generates infection hyphae and haustorial mother cells around mesophyll cells. Starting from the haustorial mother cells, haustoria are formed by acting as feeding structures. After haustorial formation, further haustorial mother cells and haustoria are generated and from the resulting network of fungal hyphae uredospores are generated first within seven to ten days (Fig. 1A, Bolton et al., 2008). Epidemics have been recently reported from many regions that are divided into epidemiological zones, i.e. Mexico, Canada and the United States; South Asia; West Asia, Eastern Europe and Egypt; Southern Africa; Northern Africa and Western Europe; the Far East; South-East Asia; South America, Australia and New Zealand (Huerta-Espino et al., 2011). Yield losses up to 50% were observed in Egypt in 1980. One reason for the occurrence of epidemics is the cultivation of a limited number of cultivars; for example, in 1976–7, the leaf rust-susceptible cultivar Jupateco J73 was grown on nearly 75% of the wheat acreage in Northwestern Mexico, resulting in 40% yield losses (Dubin and Torres, 1981). Nowadays, more than 70 genes, mostly race-specific resistance genes, are known, but only a few of these genes, for example, *Lr3, Lr10, Lr13, Lr20, Lr26* and *Lr37*, were used in cultivar breeding (Goyeau et al., 2006). Genes like *Lr1*, *Lr10* or *Lr37* code for nucleotide-binding sites (NBS) and for leucine-rich repeat (LRR) proteins and are principally vulnerable to the emergence of

virulent isolates, which were shown for *Lr37* for the first time in 2006. Today, isolates with virulence against *Lr37* are very common within the European rust population (Hanzalova and Bartos, 2014). Virulence in general increases rapidly in response to the growing area of leaf rust-resistant cultivars. Kolmer et al. (2012) identified 74 different leaf rust pathotypes with virulence to 19 of 20 *Lr* genes, with the exception of *Lr42*. Within Europe, no virulence to *Lr9* was observed, and only rarely virulence to *Lr19* and *Lr24* was noticed, whereas *Lr24* has already been overcome in South America (Huerta-Espino et al., 2011). The analysis of the leaf rust population on a differential set of genotypes carrying *Lr* genes within an epidemiological zone is the basis for the identification of effective resistances. Besides these, QTLs or genes conferring slow-rusting resistances, for example, *Lr34*, *Lr46*, *Lr67* or *Lr68*, confer resistance to all known leaf rust races (Singh et al., 2011a; Herrera-Foessel et al., 2012). Pyramiding of effective NBS-LRR resistances, for example, *Lr16* and *Lr23*, with race-independent quantitative resistances conferred for example by *Lr34* (Tsilo et al., 2014) or *Lr46* as well as by QTL, may be a cost-effective and environment-friendly

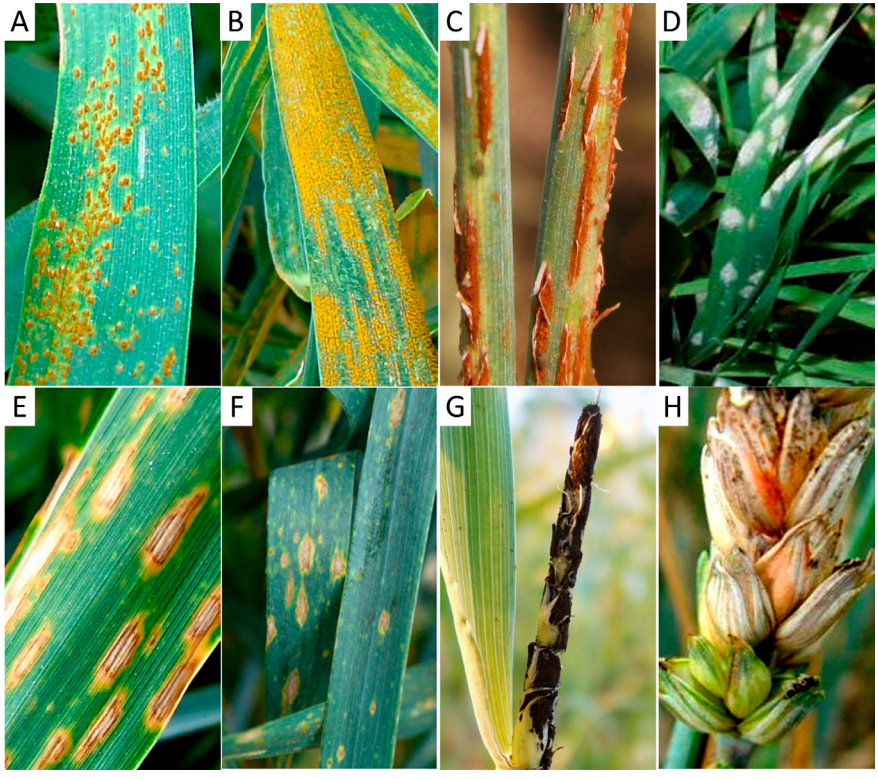

Figure 1 Wheat plants infected by different fungal pathogens. Stems are infected by leaf rust (*Puccinia triticina*, A), leaves by stripe rust (*Puccinia striiformis*, B), stem rust (*Puccinia graminis*, C) powdery mildew (*Blumeria graminis*, D), leaf blotch (*Septoria* spec., E) and tan spot (*Pyrenophora tritici-repentis*, F). Ears are infected by loose smut of wheat (*Ustilago tritici*, G) and Fusarium head blight of wheat (*Fusarium* spec., H).

approach to avoid epidemics and yield losses (Stuthman et al., 2007). However, as leaf rust-resistant cultivars are in general not grown on the complete wheat acreage; for example, in Germany, the three cultivars with the highest area of seed increase in 2015 (Anonymous, 2015) were moderately susceptible to leaf rust, fungicide protection is required. In contrast to other fungal pathogens, resistance of *P. triticina* to fungicides has not been observed up to now. However, shifts observed in the past with regard to the sensitivity of *P. triticina* to sterol 14α-demethylase inhibitors (DMIs) indicate that a moderate-to-low risk of fungicide resistance is present (Stammler et al., 2009). Several fungicide classes such as C3-quinone outside inhibitor fungicides (QoI, strobilurins), G1 DMIs and C2-succinate dehydrogenase inhibitors (SDHIs) are efficient against leaf rust.

2.2 Stripe rust

Puccinia striiformis is also of worldwide importance, but epidemics occur only under favourable weather conditions, which are described in detail by Coakley (1979). A minimum of three hours of free moisture exposure is required for the germination of uredospores and the spreading of infection. Fungal infections start at temperatures of 4°C with an optimum temperature at 7°C. In contrast to stem or leaf rust, stripe rust does not form appressoria on wheat so that the germ tubes penetrate the stomata directly. After the generation of haustorial mother cells, long hyphae grow in a longitudinal direction on leaves. This results in the streaky appearance of uredospore pustules on the leaves (Fig. 1B, Moldenhauer et al., 2006). Epidemics occur in particular if wheat crops of different maturity dates are grown and if an overlap between late crops and volunteer plants is present (Zeng and Luo, 2008), so that the pathogen can survive on living green tissue the whole year to complete its asexual life cycle (Zeng et al., 2014). However, as both *Berberis* spp. and *Mahonia* spp. are known as alternate hosts, they do not play a role in stripe rust epidemics under natural conditions (Chen et al., 2014). Ten epidemiological zones are structured on the basis of race patterns and their virulence composition (Ali et al., 2014). Epidemics with up to 80% yield losses are reported and frequently yield losses between 5% and 10% are observed. Breakdown of resistance genes has been documented in the past for example for *Yr2* and *Yr9*, which were widely used in most parts of the world in the 1990s (Bahri et al., 2011). Epidemics also evolved after the breakdown of *Yr17* in Europe and *Yr27* in India. Due to recombination and a high diversity within the population, new races emerge frequently; for example, in Europe, the so-called 'Warrior' race, which is characterized by a complex spectrum of virulences and a very high spore production, was detected in 2011 for the first time (Hovmøller et al., 2015). Hence, the combination of effective *Yr* genes with quantitative resistances is an option to avoid massive infections by stripe rust. Such effective *Yr* genes are for example *Yr5* and *Yr15* (Hovmøller, 2014), whereas *Yr18*, which is co-localizing with *Lr34*, *Yr29* and *Yr46*, confers a race-unspecific adult plant resistance. These slow-rusting genes are known to enhance the level of resistance when combined with additional QTLs (Herrera-Foessel et al., 2011), so that a wider use of slow-rusting genes in wheat breeding may result in increased resistance (Singh et al., 2000). However, stripe rust epidemics do not occur every year; therefore, resistance against this pathogen in some regions of the world is not the main focus of wheat breeders. Therefore, and because of rapid changes of isolates within the stripe rust populations, fungicide treatments are needed in many parts of the world. Plant protection according to the principles of integrated pest management (IPM) with a flexible handling of fungicide treatments and continuous monitoring of fields is essential in case of stripe rust as the pathogen sometimes infects seedlings in fall or

early spring, already (Jørgensen et al., 2008). As mentioned for leaf rust, no resistance to fungicides like DMIs, QoI or SDHIs was observed, but a shift to DMIs is documented.

2.3 Stem rust

Puccinia graminis is historically known as the most aggressive rust and its uredospores were already detected in lemma fragments from the late Bronze Age (Kislev, 1982). In addition to dew, which is essentially needed for germination and penetration, high temperatures of more than 30°C are optimal for fungal growth and sporulation. The process of infection resembles that of leaf rust, but under optimal conditions, more uredospores are generated than by leaf or stripe rust. Pustules, which consist of up to 10,000 spores, are formed on the entire plant (Fig. 1C). As mentioned for stripe rust, sexual recombination occurs on the alternate host *Berberis vulgaris* (common barberry). In contrast to leaf and stripe rust, high amounts of aecidiospores are produced on alternate hosts (Singh et al., 2015). The combination of virulences by sexual recombination often results in epidemics leading frequently to losses of up to 80% (Dubin and Brennan, 2009) causing famines, e.g. in Ethiopia (1993–4). Ever since the deployment of wheat stem rust adult plant resistance gene *Sr2* in the 1920s, which provided partial resistance to all stem rust races, more than 60, mostly race-specific, *Sr* genes have been identified, and some of these were introduced in cultivars. However, the break down of *Sr* genes resulted in devastating epidemics, e.g. in Australia in the 1940s due to the breakdown of *Sr6* and in the United States in the mid-1950s after the breakdown of *Sr9*, *Sr11* and *Sr16* (Singh et al., 2015). Stem rust was well controlled after the deployment of stem rust resistance genes *Sr26* and *Sr31* derived from rye in the 1970s. *Sr31* was used worldwide, and it provided the basis of resistance in spring, facultative and winter wheat varieties. However, strain *Ug99*, which is virulent against *Sr31*, was detected in Uganda in 1999 and has meanwhile spread out over Africa to Iran. Besides this, virulent strains against *Sr24* and *Sr36* were detected, rendering stem rust a most important pathogen (Jin and Singh, 2006; Singh et al., 2011b). The emergence of race *Ug99* was mainly due to the selection pressure imposed by the extensive use of a few *Sr* genes. Nevertheless, long-term control can be achieved by using stem rust resistance genes effectively against race *Ug99* (Yu et al., 2014) and by pyramiding these using information about effective resistance genes (Borlaug rust initiative (BGRI), http://www.globalrust.org/). Furthermore, Singh et al. (2015) point out that a high level of durable resistance can be achieved if slow-rusting genes leading to adult plant resistance are combined, for example, *Sr2*, *Sr55*, *Sr56*, *Sr57* and *Sr58*. Even if stem rust infections can be controlled by growing resistant cultivars, fungicide treatments should be conducted if a stem rust severity level higher than 5% is monitored, for example, by applying fungicides containing DMIs in combination with QoI (Wanyera et al., 2009).

3 Fungal diseases of wheat: powdery mildew, *Fusarium* diseases and *Septoria* diseases

3.1 Powdery mildew

In contrast to rusts, *B. graminis* f. sp. *tritici* to a larger extent depends on humid rain-fed conditions or irrigation for successful infection. The severity of powdery mildew infection is

strongly influenced by the amount of nitrogen fertilizer. Large single applications or excessive multiple applications of N fertilizers result in outbreaks of powdery mildew (Chen et al., 2007). In mild climatic conditions, *B. graminis* can replicate even during winter. Powdery mildew germinates best at a high relative humidity (>95%). The temperature range for germination is around 10–22°C. Disease development will decline rapidly at temperatures higher than 25°C. TeBeest et al. (2008) described favourable weather conditions which increase the probability of powdery mildew epidemics, that is, high relative humidity in the range of 95–100%, low radiation and sunshine hours and the above-mentioned temperature range. The powdery mildew infection process starts after conidiospores generate germ tubes. Next, an appressorium forms and initiates the penetration of the leaf cuticle and epidermal cell wall. Within epidermal cells, a haustorium is generated. After five to eight days, the asexual life cycle is completed by the formation and release of conidiospores (Schulze-Lefert and Vogel, 2000). Conidiophores and spores are visible as white pustules on all plant parts above ground (Fig. 1D). In correspondence with the above-mentioned optimal conditions, regions in which powdery mildew is an important pathogen are the cooler regions of China, Japan and humid regions in Asia, North and East Africa, Northern Europe and Northeastern America with mild winters where wheat is sown in autumn. Many race-specific resistance genes against powdery mildew (*Pm* genes) are deployed in wheat cultivars. Currently, 64 resistance genes conferring resistance to wheat powdery mildew are known (Alam et al., 2011). However, cultivars in general carry only one or very few *Pm* genes. This results in selective pressure on the pathogen population and therefore, resistances are of low durability (Parks et al., 2008). Most of these race-specific resistances are easily overcome by simple genetic changes in the pathogen so that, e.g. *Pm8* and *Pm17* were overcome already in the 1980s. *Pm4a* was overcome in some areas of China and *Pm21* was overcome after extensive use in Europe (Gao et al., 2012). Therefore, alternatively, the deployment of race-independent resistances, such as *Lr34/Yr18*, which is also effective against powdery mildew in combination with other resistance genes, could prevent the emergence of new virulent races. In addition, more than 100 QTLs have been identified and can be employed in marker-based selection procedures (Keller et al., 1999; Asad et al., 2014; Marone et al., 2013). Furthermore, Wang et al. (2014) were able to engineer mlo-driven resistance in wheat, which is known in barley as a durable powdery mildew resistance, by allele editing. In order to control powdery mildew infections, fungicides with specific activity against powdery mildew, such as quinoxyfen, are available. However, in contrast to rusts, some isolates of *B. graminis* f. sp. *tritici* have developed resistance to DMIs in the field (Wyand and Brown, 2005). Furthermore, resistance to QoI fungicides (Sierotzki et al., 2000), quinoxyfen, benzimidazoles (Felsenstein et al., 2010) or morpholines was detected (Godet and Limpert, 1998). No cross-resistance to fungicides has been described so far in field populations and therefore, a combination of SDHIs, azoles and strobilurins is still highly effective.

3.2 Fusarium head blight and *Fusarium* crown rot

Fusarium head blight (FHB) or scab is caused by several species of the genus *Fusarium* and is one of the most important diseases in wheat. FHB occurs in most parts of the world especially in the wheat-growing areas of the United States, Canada, Australia and Europe and primarily in regions with warm and humid conditions during flowering (Zhang et al., 2011a). The infection of the ear can cause high yield losses ranging from 10% to 70% during epidemics by reducing the number of grains per spike and thousand grain

weight (Matthies and Buchenauer, 2000). Also, germination is affected and baking quality is reduced. *Fusarium* infection may also change the content of gliadins and glutenins (Eggert et al., 2010). Nineteen different *Fusarium* species cause wheat FHB (Stepień and Chelkowski, 2010), and the most important of these species are *F. culmorum*, *F. avenaceum* (teleomorph *Gibberella avenacea*), and especially *F. graminearum* (teleomorph *G. zeae*; Buerstmayr et al., 2012). The *Fusarium* species grow and overwinter in the soil on plant debris of maize, wheat and other cereals (Parry et al., 1995). The saprophytic mycelium on stubble residues such as chlamydospores (*F. culmorum*), perithecia (*F. graminearum*) and macroconidia build the inoculum for the infection in the next growing season (Buerstmayr et al., 2012). Wind, rain and insects lead to the spread of spores, and warm, humid, weather conditions before, during and after flowering favour inoculum production as well as infection and infestation of developing grains (Schmale and Bergstrom, 2003). The fungal spores germinate on the surface of flowers and husks and the hyphae of the fungus enter either passively via the stomata or actively via the cell wall. The first symptoms of FHB occur shortly after flowering. Diseased spikelets exhibit premature bleaching within the head, and over time bleaching may progress throughout the entire ear (Fig. 1H, Schmale and Bergstrom, 2003). Infested grains are shrivelled, are smaller than normal and are white to pale pink in colour and contain different trichothecenes (Buerstmayr et al., 2012). The presence of these mycotoxins in food and feed is a problem worldwide, leading to the setting of thresholds for the use of wheat as food and feed (Buerstmayr et al., 2012). Effective fungicides for reducing FHB infections and deoxynivalenol (DON) levels are tebuconazole, metconazole, prothioconazole and bromuconazole (Freije and Wiese, 2015; Mesterházy et al., 2011). With respect to resistance, five types of resistance are known: type I, resistance to primary infection; type II, resistance to disease spread; type III, resistance to DON accumulation; type IV, resistance to kernel infection and type V, resistance to yield loss (Zhang et al., 2011a). The development and the use of resistant cultivars and IPM systems are essential for the control of FHB. Genetic variation for resistance to FHB is well documented in wheat and its relatives (Gilbert and Tekauz, 2000; Buerstmayr et al., 2012). FHB resistance is in general a quantitative trait and many QTLs have been identified (Zhang et al., 2011a; Buerstmayr et al., 2009). The Chinese cultivar 'Sumai 3' shows an extraordinary high level of type II resistance which is due to a gene located on chromosome 3BS. Two smaller-effect QTLs from 'Sumai 3' were mapped on chromosome 6BS. The so-called 'Sumai 3' resistance is used in breeding programmes worldwide (Zhang et al., 2011a; Buerstmayr et al., 2012). An overview on QTLs known for FHB resistance is given by Buerstmayr et al. (2009, 2012).

Besides FHB, *Fusarium* crown rot (FCR) is caused by several species of the genus *Fusarium*, of which *F. pseudograminearum* (teleomorph *Gibberella coronicola*) turned out to be most important, at least in Australia (Liu and Ogbonnaya, 2015). FCR is a severe disease causing yield losses up to 25–35% mainly in the semi-arid wheat-growing regions worldwide (Chakraborty et al., 2006). Symptoms, which are intensified by drought, are leaf sheath or stem base browning and whiteheads (Singh et al., 2009). Chlamydospores overwinter on crop residue, in the soil or on seeds and form asexual conidia in spring, which are spread by wind, and are the primary inoculum infecting wheat. Rarely, perithecia with asci and ascospores are formed (Chakraborty et al., 2006). *F. pseudograminearum* also produces DON, but *F. pseudograminearum* in general does not infect heads (Mudge et al., 2006). An integrated management to reduce the FCR infestation includes fungicide seed treatments (difenoconazole–mefenoxam fungicide), agronomic practices and growing of resistant cultivars (Moya-Elizondo and Jacobsen, 2016). No varieties showing

complete resistance are known (Chakraborty et al., 2006; Bentley et al., 2008), but quantitative differences have been observed (Liu and Ogbonnaya, 2015; Moya-Elizondo and Jacobsen, 2016), and several QTL involved in FCR resistance have been detected (Liu and Ogbonnaya, 2015).

3.3 Septoria diseases

Three pathogens of the genus *Septoria*, that is, *S. nodorum* (teleomorph *Leptosphaeria nodorum*) causing septoria glume blotch, *S. tritici* (teleomorph *Mycosphaerella graminicola*) and *S. avenae* f. sp. *triticea* (teleomorph *Leptosphaeria avenae* f. sp. *triticea*) causing septoria leaf blotch (Eyal, 1987; Wiese, 1987) can lead to yield losses ranging between 30% and 50% (Eyal, 1987). Before the 1960s they played only a minor role in wheat production, but since the introduction of semi-dwarf cultivars, their importance increased, since these cultivars are in general more susceptible due to the mode of spreading of the pathogens. Infection with *Septoria* spp. leads to a reduced number of seeds and shrivelled kernels.

Symptoms like chlorotic spots which get necrotic later appear initially on lower leaves and progress upward during the growing season (Eyal, 1987; Wiese, 1987). In the necrotic epidermal and mesophyll cells, pycnidia arranged in rows are formed on both sides of the leaves (Fig. 1E, Eyal, 1987; Palmer and Skinner, 2002). Lesions of leaf blotch are more rectangular, whereas lesions of glume blotch are lens-shaped. If the nodes are infected, distortion and bending of the straw, as well as lodging, occur. Symptoms become visible for leaf blotch 15 to 21 days and for glume blotch 7 to 14 days after inoculation and can be found on all plant parts, but glume blotch is mainly found on the glume and the nodes and contrary to leaf blotch it also infects seeds (Eyal, 1987).

Septoria spp. overwinter as conidia in pycnidia on infested plant debris or in infested seed, but pseudothecia also play an important role as primary inoculums in some regions. Under wet conditions for a period of at least 30 minutes, pycnidia release pycnidiospores, which are spread onto host plants by splashing rain, irrigation, farming tools, animals, etc. The pycnidiospores germinate by budding or elongation and enter the host tissue via stomata or directly through the cell wall of the epidermis. The hyphae colonize the mesophyll cells, grow intercellularly, obtaining nutrients from the apoplast. Eight to ten days after the initial infection, the fungus attacks the mesophyll cells leading to chloroses and necroses and forming pycnidia. Wet and windy conditions favour epidemics and moisture is required for all developmental stages of the pathogen (Eyal et al., 1987; Kema et al., 1996).

Cultural practices to control *Septoria* diseases are the use of healthy seeds, a two to three years´ crop rotation, tilling and the destruction of volunteer wheat (Wiese, 1987). Chemical control can be achieved through seed treatment and foliar fungicide application. Available fungicides are DMIs, for example, epiconazole, prothioconazole and the SDHIs fluxapyroxad, isopyrazam, bixafen and biscalid (Fraaije et al., 2012). *Septoria* spp. have developed resistance to several classes of fungicides and therefore, the preventive use of SDHI fungicides in mixtures with a maximum of two sprays per season is advised. Mixtures of the fungicidal compounds which have different mode of action, such as azoles with SDHI, reduce the occurrence of fungicide-resistant isolates (Fraaije et al., 2012). The most effective way of controlling *Septoria* diseases is by growing resistant cultivars. So far, 17 major resistance genes (*Stb* genes) and many QTLs have been identified (Goodwin, 2007; Orton et al., 2011; Ghaffary et al., 2012).

4 Fungal diseases of wheat: other important diseases

4.1 Tan spot

Tan spot of wheat, caused by the ascomycete *Pyrenophora tritici-repentis* (Ptr), has become increasingly important since the late 1970s and is now widespread in all wheat-growing areas (Moreno et al., 2012; Faris et al., 2013). *P. tritici-repentis* has the widest host range of all *Pyrenophora* species (Shoemaker, 1962). It infects numerous, mostly perennial, grasses, but the most economically important host plants are wheat (*Triticum aestivum* L.) and durum wheat (*Triticum turgidum* L.) (Strelkov and Lamari, 2003). The fungus on the one hand lives on its host plants and seeds as mycelium or in its saprophytic lifestyle on infected straw and crop residues on the soil surface (de Wolf et al., 1998). The main source of infection is wheat straw debris, and the most important cause for the increasing importance is reduced tillage (Faris et al., 2013). Typical for the disease are the secondary infections caused by conidia. After an infection, the first visible symptoms are small brown spots. Around these, yellowing occurs due to toxins (Strelkov and Lamari, 2003). Next, necrosis is visible, developing into oval or diamond-shaped, yellow brownish spots with a small dark brown or black centre (Fig. 1F; de Wolf et al., 1998; Strelkov and Lamari, 2003). The ability of *P. tritici-repentis* to induce necroses and chloroses in wheat is the result of several host-specific toxins, which occur in various combinations in the eight described Ptr-races (Lamari et al., 2003; Strelkov and Lamari, 2003). The fungus produces at least three host-selective toxins (HSTs), known as Ptr ToxA, Ptr ToxB and Ptr ToxC, which interact with proteins encoded by dominant host genes (Faris et al., 2013). Tan spot can cause significant yield losses by reducing the photosynthetic area, resulting in a lower thousand kernel weight and a lower number of kernels per ear (Faris et al., 2013). Under favourable conditions for disease development, yield losses of 30–50% were observed (Shabeer and Bockus, 1988). The disease can be controlled by growing resistant cultivars, fungicide application, crop rotation and a combination of all three approaches (Singh et al., 2012). Fungicides containing strobilurins, together with propiconazole, turned out to be very efficient in preventing the spread of tan spot (Jørgensen and Olsen, 2007) as well as triazole fungicides (Bankina and Priekule, 2011). Resistance to QoI fungicides in *Ptr* strains was detected in Northern and Western Europe, and molecular analysis revealed the presence of the F129L mutation in resistant isolates (Sierotzki et al., 2007; Patel et al., 2012).

Resistance based on single recessive tan spot HST insensitivity genes (*tsn1*, *tsc1*, *tsc2*) and tan spot resistance genes (*tsr2* to *tsr5*) (Faris et al., 2013) and QTLs conferring quantitative resistance to different races e.g located on chromosomes 1BS and 3BS and being effective against *Ptr* races 1, 2, 3 and 5 are known (Faris and Friesen, 2005). Furthermore, *tsn1* has been isolated (Faris et al., 2010). Additional sources of resistances were identified in wheat and in related wild species, wheat-alien species derivatives and synthetic hexaploid wheat lines (Faris et al., 2013).

4.2 Smuts

Bunt and smut diseases are caused by fungal pathogens belonging to the basidiomycetes. There are six pathogens associated with five bunt and smut diseases of worldwide importance in wheat. These are *Tilletia laevis* and *T. tritici* (common bunt), *T. controversa*

(dwarf bunt), *T. indica* (karnal bunt), *Ustilago tritici* (loose smut) and *Urocystis agropyri* (flag smut). Karnal bunt occurs currently only in India, Pakistan and Mexico. The other bunts are known worldwide, but are of minor importance, compared to other wheat diseases. Yield losses can be up to 50%, and complete crop failures were also observed in heavily infested fields (Knox and Menzies, 2012; Toor and Bariana, 2012; Wiese, 1987; Wilcoxson and Saari, 1996).

The teliospores get disseminated by wind or during threshing and overwinter on seeds, plant debris or in the soil, some also as mycelium in the seed or plants (Wilcoxson and Saari, 1996). Under favourable conditions, the teliospores germinate, produce dicaryotic mycelium and infect the ovaries or young seedlings. The hyphae grow inter- and intracellularly and produce haustoria (Batts, 1955; Bonde et al., 1997; Cashion and Luttrell, 1988; Wilcoxson and Saari, 1996). Plants infected with common bunt, dwarf bunt, karnal bunt and loose smut (Fig. 1G) develop bunt sori containing teliospores instead of kernels. Spikes infected with common bunt, dwarf bunt and karnal bunt have a foul odour due to the production of trimethylamine (Bonde et al., 1997; Wiese, 1987; Wilcoxson and Saari, 1996). Sori of flag smut can be seen on the stem and leaves (Nelson and Durán, 1984). Other symptoms can be stunting, increased tillering, reduced kernel weight, reduced number of spikes and reduced number of kernels per spike, twisted and bended leaves and leaf discolouration (Bonde et al., 1997; Cashion and Luttrell, 1988; Knox and Menzies, 2012; Nelson and Durán, 1984; Toor and Bariana, 2012; Wilcoxson and Saari, 1996).

Effective control of bunts and smuts can be achieved by chemical seed treatments. Active ingredients are triazoles, mancozeb, carboxin, oxycarboxin, triadimenol and triadimefon, which are systemic and offer mostly complete protection (Goel, 1992; Wiese, 1987; Wilcoxson and Saari, 1996). Besides this, sufficient control can be achieved by resistant cultivars. Resistance in wheat against common and dwarf bunt is due to the same resistance genes, designated *Bt1* to *Bt15* (Wiese, 1987; Wilcoxson and Saari, 1996). Resistance genes to karnal bunt have been detected on chromosomes 2A, 7A, 3B, 1D, 3D and 5D (Gill et al., 1993). Against loose smut eight resistance genes have been found and mapped till now and these are *Ut1* and *Ut2* on chromosome 6A; *Ut3*, *Ut4* on 7B; *Ut5* on 2B; *Ut6* on 5B; *Ut7* on 7A and *Ut8* on chromosome 3A (Kassa et al., 2014, 2015; Knox and Menzies, 2012; Knox et al., 2014). Many cultivars that are resistant against flag smut are available. QTLs for resistance have been mapped on chromosomes 3A, 6A, 1B and 5B (Toor and Bariana, 2012; Toor et al., 2013).

4.3 Blast

Magnaporthe grisea (anamorph *Pyricularia grisea*) is an ascomycete and the causal agent of wheat blight. *M. grisea* is a major pathogen in rice, but in 1985, it was detected in wheat in Paraná, Brazil, for the first time (Igarashi et al., 1986). Since then it spread to other states of Brazil within a few years and was also recently detected in Paraguay and Bolivia (Kohli et al., 2011; Maciel et al., 2014). Average yield losses range between 10% and 13% (Goulart et al., 2007), but complete crop failures have also been observed (Igarashi, 1991). After wheat and rice, barley, rye, triticale and some grasses are affected and can be important sources for inoculum (Kohli et al., 2011).

Symptoms are visible on all plant parts (Igarashi, 1991; TeBeest et al., 2007). A seedling infection can result in plant death. Foliar symptoms on young leaves are elliptical lesions which vary in shape and size on older leaves. The centres are whitish to light brown with reddish-brown to dark grey margins. On the lower side of the leaf the lesions are dark

grey due to sporulation. The awns can have brown to whitish discolourations. The spikes are straw-coloured. Depending on the growth stage at which infection takes place, the kernel formation can be zero to normal. Kernels of diseased plants are smaller, lighter and shrivelled and are of low quality (Urashima et al., 2009).

Pyricularia grisea is an air- and seed-borne disease. Seeds, grasses, volunteer plants and plant debris can be sources of inoculum. By wind and rain, conidia are distributed to host plants, where they germinate and generate an appressorium. Through a penetration peg the primary infection hyphae invade the plant tissue where they grow rapidly. In resistant cultivars the growth is inhibited. Secondary lesions on the leaves produce new inoculum resulting in many infection cycles per season. Towards the end of the growing season, the infection of the heads, mainly through airborne inoculum, takes place, leading to partial or total loss of the heads, depending on the point of infection, since *P. grisea* does not grow systemically in the plant like *F. graminearum* (TeBeest et al., 2007; Kohli et al., 2011).

Maciel et al. (2014) found different virulence groups in *M. grisea* isolates. They identified 14 seedling virulence groups (SVGs, A' to U') and eight detached-head virulence groups (HVGs, A' to T'). A mixed reproductive system in *M. grisea*, gene flow and dispersal across large spatial distances, as well as a high pathotype diversity, leads to a high evolutionary potential, making this pathogen a great threat to wheat growers.

Fungicides that provide complete control have not been discovered so far. Some strobilurins and triazoles can be applied during heading, but they provide no control in susceptible cultivars. The majority of available cultivars is susceptible, but some resistance genes like *Rmg1* (Takabayashi et al., 2002; Hirata et al., 2005), *Rmg2*, *Rmg3* (Zhan et al., 2008), *Rmg4*, *Rmg5* (Nga et al., 2009), *Rmg6* (Vy et al., 2014), *Rmg7* (Tagle et al., 2015) and *Rmg8* (Anh et al., 2015) have been found in the past few years, and they are located on chromosomes 1D, 7A, 6B, 4A, 6D, 2A, 1D and 2B, respectively.

4.4 Spot blotch

Spot blotch is a leaf disease of wheat caused by *Cochliobolus sativus* (anamorph *Bipolaris sorokiniana*), but *C. sativus* also infects other plant parts and causes seedling blight, seed rot and root rot. *C. sativus* is an important disease in warmer and humid wheat-growing regions (Acharya et al., 2011). A heavy infestation leads to a significant reduction in grain yield and quality. Average yield losses due to spot blotch range between 15% and 20% and can reach 40–70% in susceptible genotypes (Duveiller and Sharma, 2009; Aggarwal et al., 2004). Symptoms mainly develop on subcrown internodes, stems, leaves, awns, glumes and seeds. Early symptoms are characterized by small, dark brown lesions without chlorotic margins. In susceptible genotypes, these lesions extend very quickly in oval to elongated blotches, light brown to dark brown in colour. Diseased seedlings develop dark brown lesion on the coleoptiles, crowns, stems and roots (Acharya et al., 2011). *C. sativus* is seed-transmitted, but can also survive as thick-walled conidia and as mycelium in soil or crop debris. The sexual stage is not important in the disease cycle. Primary inoculum includes mycelium from infected seed, conidia in the soil, conidia on the kernel surface, volunteers and secondary hosts (Acharya et al., 2011; Duveiller and Sharma, 2009). Conidia grown on dead plant debris, spread by wind or rain, germinate under favourable conditions on the host surface and produce an appressorium from which infection hyphae are developed. The infection hyphae enter the host tissue where they grow rapidly along the intercellular spaces of the mesophyll tissue. Conidia of secondary lesions spread the infection. The disease is polycyclic (Acharya et al., 2011; Duveiller and Sharma, 2009).

Among the isolates of *C. sativus*, differences in virulence were observed and genetic studies provide hints regarding the presence of single major genes (Mironenko et al., 2013; Mahto et al., 2012). *C. sativus* can be controlled by growing resistant cultivars, foliar fungicide application and seed treatment. Besides this, the appearance of *C. sativus* is influenced by crop rotation and cultural practice (Acharya et al., 2011). Yield losses can be avoided by spraying fungicides of the triazole group (e.g. tebuconazole and propiconazole) at tillering and booting (Yadav et al., 2015; Singh et al., 2013) and by seed treatment (Mehta, 1993). Resistance to *C. sativus* has been identified in genotypes derived from Brazil, Zambia and the Yangtze River Valley, and resistance is controlled by dominant and recessive genes (Duveiller and Sharma, 2009). Besides this, two QTLs were located on chromosomes 2BS and 5BL (Suneel et al., 2015).

4.5 Eyespot

Eyespot is a major disease affecting winter wheat, caused by two fungal species *Oculimacula acuformis* and *O. yallundae*; this disease occurs in several European countries and other temperate regions worldwide. Winter wheat grown in areas of high rainfall and moderate winters, such as Northwestern Europe and the Pacific Northwest (PNW) region of the United States, are particularly vulnerable to infection, but the disease has also been detected in South Africa and New Zealand. Yield losses up to 50% are reported (Leonard et al., 2008; Burt et al., 2010). Symptoms caused by these two pathogens are macroscopically quite similar (Burnett and Hughes, 2014). After spore germination, appressoria are generated. Starting from these appressoria *O. acuformis* penetrates directly into coleoptile epidermal cells, with hyphae crossing cell walls, while *O. yallundae* follows an orientated hyphal growth pattern. Besides this, *O. yallundae* exhibits an asymptomatic biotrophic phase during coleoptile colonization before switching to a necrotrophic phase (Burt et al., 2010). Elliptical lesions on the lower portion of the stem become clearly visible and the stems are weakened, causing their collapse in all directions (Kwiatek et al., 2012). Fungicide treatments to control wheat eyespot are most effective when applied between the end of tillering and the second node stage. DMIs can be used to control eyespot, but they are effective especially against *O. yallundae,* whereas isolates of *O. acuformis* that are less sensitive to triazoles have already been detected (Leroux et al., 2012). Hence, fungicide mixtures of DMIs (e.g. epoxiconazole), aminopyrimidines (e.g. cyprodinil) as a systemic component, and strobilurins (e.g. picoxystrobin) should be used in order to avoid the selection of insensitive isolates (Ray et al., 2004). Resistances against *Oculimacula* species (*Pch1* and *Pch2* and a QTL on chromosome 5A) and respective markers (Meyer et al., 2011) are available in wheat, but neither of these genes provides complete disease control. Besides this, resistance has been detected in wild relatives (e.g. Yildirim et al., 1998).

5 Virus diseases of wheat

Triticum species are natural hosts of more than 40 different viruses. But only some of them cause relevant yield losses worldwide, or in some geographic regions. These are viruses belonging to the genus *Bymovirus* (family *Potyviridae*) or the genus *Furovirus* (family *Virgaviridae*) transmitted by the root-infecting plasmodiophorid *Polymyxa*

graminis Ledingham (Rao and Brakke, 1969) as well as insect-transmitted viruses. Insect transmitted viruses are the aphid-borne viruses of the family *Luteoviridae* causing Barley yellow dwarf disease, and the leafhopper-transmitted *Wheat dwarf virus* (WDV), a member of the genus *Mastrevirus* within the family *Geminiviridae*. Furthermore, the mite-transmitted *Wheat streak mosaic virus* (WSMV) belonging to the genus *Tritimovirus* within the family *Potyviridae* is an important viral pathogen of wheat.

For further information on viruses that have been reported only in some countries, e.g. Aubian wheat mosaic virus, *Barley yellow striate mosaic virus*, *Barley stripe mosaic virus*, Brazilian wheat spike virus, *Brome mosaic virus*, Chinese wheat mosaic virus, European wheat striate mosaic virus, High Plains disease virus, *Indian peanut clump* virus, *Maize dwarf mosaic virus*, Mal de Rio Cuarto virus, Nariño dwarf virus, *Peanut clump virus*, Triticum mosaic virus, Winter wheat Russian mosaic virus, confer to e.g. Lapierre and Signoret (2004).

5.1 Soil-borne viruses

In the case of soil-borne viruses, breeding of resistant cultivars is the only method of controlling these diseases. Because of the long-term survival of the resting spores of their vector, *Polymyxa graminis*, long crop rotations have only a limited impact and the application of fungicides is not effective as *P. graminis* is found infectious up to a soil depth of 70 cm (Richard-Molard, 1985; Brakke and Langenberg, 1988; Adams, 1990).

5.1.1 *Soil-borne wheat mosaic virus* (family: *Virgaviridae*, genus: *Furovirus*)

Soil-borne wheat mosaic virus (SBWMV, syn.: Wheat soil-borne mosaic virus, Wheat mosaic virus) was first reported in 1919 in the United States (McKinney, 1925) and is now one of the most important diseases in winter wheat, especially in the Central and Eastern US (Putman et al., 1994). But it was also detected in Argentina, Brazil, China, Japan and Germany (Ziegler et al., 2013). Considerable yield losses up to 50% or 80% were estimated in infected wheat fields in the United States and Brazil, respectively (Myers et al., 1993; Prestes and Wietholter, 1993). Infection of wheat roots takes place by zoospores of *Polymyxa graminis* in autumn stimulated by lower temperature and higher soil moisture. In the following spring, leaves of younger plants show light green or yellow mosaics or stripes (Fig. 2A). Older plants may be stunted or form rosettes with excessive tillering or a reduced root system. Symptom expression depends on the virus isolate, the wheat cultivar grown and also on the plant developmental stage when infection takes place. Virions of SBWMV are rigid rods with a length of approximately 140 to 160 and 260 to 300 nm and a diameter of about 20 nm (King et al., 2011; www.ictvonline.org/virustaxonomy.asp).

To reduce virus infection late sowing of wheat is recommended. But more effective is the cultivation of resistant cultivars. Genetic resources, breeding lines and cultivars carrying resistance to SBWMV are mainly known from the United States, but also from Japan and Brazil (Hall et al., 2009). Narasimhamoorthy et al. (2006) located a single resistance gene on chromosome 5DL of the winter wheat cv. Karl 92. On the same chromosome, Hall et al. (2009) detected a resistance gene in a germplasm with *Aegilops tauschii* as resistance donor. In addition, Zhang et al. (2011b) identified a further locus on chromosome 4DS.

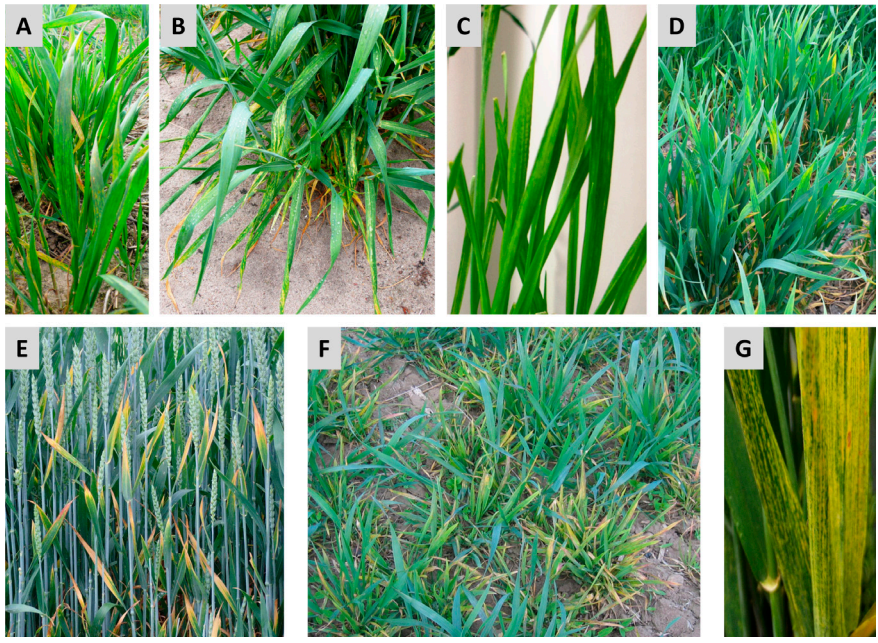

Figure 2 Virus symptoms on *Triticum* caused by (A) *Soil-borne wheat mosaic virus*, (B) *Soil-borne cereal mosaic virus*, (C) *Wheat spindle streak mosaic virus*, (D) *Wheat yellow mosaic virus*, (E) *Barley yellow dwarf virus*, (F) *Wheat dwarf virus*, (G) *Wheat streak mosaic virus*.

Photos: A-C Kastirr, U.; D-F Habekuß, A.; G Rabenstein, F. (JKI)

The mechanism of resistance to SBWMV is still unclear. In investigations of Myers et al. (1993) the virus was detected in the roots of all cultivars independent of their level of resistance, but in the leaves it was found only in susceptible cultivars, signalling a translocation resistance. But at higher temperature (23°C) the virus was also detected in the leaves of resistant cultivars. The results of Pennington et al. (1993) who observed a reduced virus titre in the leaves of resistant cultivars support the hypothesis that resistance is due to an inhibition of virus movement.

5.1.2 *Soil-borne cereal mosaic virus* (family: *Virgaviridae*, genus: *Furovirus*)

Soil-borne cereal mosaic virus (SBCMV) infects mainly wheat and triticale in Western and Southern Europe causing yield losses up to 30–70% (Rubies-Autonell et al., 2003), and mainly rye in Central and Northeastern Europe. In the last decades an increase in the area infected with SBCMV has been detected. On virus-contaminated fields plants are infected by *Polymyxa graminis* in autumn. In March and April the virus initially causes pale mosaic symptoms on leaves and on sheaths which develop to chlorotic streaks on stunted plants until May and June (Fig. 2B). The virus particles are rod-shaped with 18 to 20 nm in diameter and with modal lengths of 120 to 130 and 200 to 230 nm (King et al., 2011; www.ictvonline.org/virustaxonomy.asp).

Resistance to SBCMV has been identified in *Triticum aestivum* and *T. durum* cultivars mainly grown in France, the United Kingdom and Italy (Hariri et al., 2001; Budge et al., 2002; Rubies-Autonell et al., 2006) and also in genetic resources (Kastirr et al., 2002). In resistant cultivars virus can be found in the roots but not or only in small amounts, in the leaves (Driskel et al., 2002; Rubies-Autonell et al., 2003). The resistance derived from cvs. Cadenza, Tremie and Claire, that is, the resistance gene *Sbm1* (Kanyuka et al., 2004; Perovic et al., 2005), is located on chromosome 5DL (Bass et al., 2006) and a highly diagnostic marker (*Xgwm469*-5D) was developed (Perovic et al., 2009). A second locus, *Sbm2*, for resistance in common wheat has been mapped on chromosome 2BS (Bayles et al., 2007). Furthermore, resistance has been detected in *Thinopyrum intermedium* addition lines (Rumjaun et al., 1996) and *Triticum monococcum* (Kanyuka et al., 2004). In durum wheat cultivars, a major QTL was located on chromosome 2BS and several minor QTLs were identified (Maccaferri et al., 2011; Russo et al., 2012).

5.1.3 *Wheat spindle streak mosaic virus* (family: *Potyviridae*, genus: *Bymovirus*)

Wheat spindle streak mosaic virus (WSSMV), first reported in Canada (Slykhuis, 1960), was later detected in the United States, in different European countries and in India (Kühne, 2009). Besides wheat, the virus affects triticale and rye and can cause yield losses about 30% in winter wheat (Cunfer et al., 1988; Miller et al., 1992). Mixed infections with SBWMV or SBCMV are often detected in Europe. Above 17°C, leaf symptoms disappear. The virus causes chlorotic to necrotic streaks parallel to the leaf veins (Fig. 2C), slight stunting and reduced tillering (Berger et al., 2005). Virions of WSSMV are slightly flexuous, filamentous particles, 13 nm in diameter and in modal lengths of 250 to 300 nm and 500 to 600 nm (King et al., 2011).

Resistance to WSSMV has been reported in several wheat cultivars and experimental lines. In some lines the virus could not be detected in leaves, but virus-bearing resting spores of *Polymyxa graminis* were found in the roots of susceptible and resistant lines giving again hint to a translocation resistance (Haufler and Fulbright, 1986). In two germplasms, that is, KS92WGRC21 and KS92WGRC22, resistance to a combined infection of *wheat spindle streak* and *soil-borne wheat mosaic viruses* was detected (Cox et al., 1994). This resistance originated from two closely related accessions of *Aegilops tauschii* (Coss) Schmal. (TA 2567 and TA 2570) derived from Armenia. Initial results on the genetics of WSSMV resistance indicate the presence of two dominant loci responsible for resistance (Van Koevering et al., 1987). Results of Yao et al. (1999) suggested a dominant mode of inheritance and the presence of one or two genes encoding this resistance. One strong QTL explaining more than 70% of the phenotypic variance in the cv. Geneva has been located on chromosome 2D (Khan et al., 2000). A major gene designated *Wss1* has been transferred from *Haynaldia villosa* by a translocation of the short arm of chromosome 4V to the long arm of chromosome 4D (Zhang et al., 2005). Furthermore, resistance has been detected in rye, which may serve as a source of resistance for wheat (Li et al., 2007).

5.1.4 *Wheat yellow mosaic virus* (family: *Potyviridae*, genus: *Bymovirus*)

Yellow mosaic of wheat was first described in Japan (Sawada, 1927) and renamed to *Wheat yellow mosaic virus* (WYMV) by Inouye (1969). Later the *Polymyxa graminis*

transmitted virus was also detected in China (Chen, 1993; Hen et al., 2000) and it can cause serious yield losses in winter wheat, the only natural host, up to 70%. WYMV-infected plants are dwarfed and show irregular chlorotic streaks or yellowing on the leaves (Fig. 2D). In Japan 3 pathotypes are distinguished on the basis of their infectivity of different cultivars and on differences in their RNA sequences (Ohki et al., 2014). The virions are slightly flexuous filaments with a diameter of 13 nm and two modal lengths of 550 and 275 nm (King et al., 2011).

Resistance to WYMV has been detected in European and Japanese winter wheat cultivars (Chen et al., 2000). The resistance detected in the Chinese wheat cultivar Yangfu 9311 and the European cv. Ibis has been mapped to chromosome 2DL (Liu et al., 2005; Nishio et al., 2010). Furthermore, in the Chinese cv. Xifeng QTLs were detected on chromosome 3BS, 5AL and 7BS (Zhu et al., 2012). In Japan, cv. Yumechikara is resistant to pathotype I and II of the virus (Netsu et al., 2011) and a single major QTL for resistance to pathotype I was mapped to chromosome 2D (Kojima et al., 2015). Suzuki et al. (2015) identified 2 major QTLs on chromosome 2DL and 3BS originated from cv. Madsen and 1 QTL on chromosome 4D originated from cv. Hokushin.

5.2 Insect-transmitted viruses

The aphid-transmitted *Barley yellow dwarf virus* (BYDV) and *Cereal yellow dwarf virus* (CYDV) and the leafhopper-transmitted *Wheat dwarf virus* (WDV) can cause serious yield losses depending on the weather conditions and the incidence of their vectors. BYDV and CYDV are of major importance in nearly all wheat-growing areas of the world, while WDV has been mainly detected in Europe and Asia so far. However, on account of global warming, it is expected that insect-transmitted viruses will acquire more importance in the future.

5.2.1 *Barley yellow dwarf virus* (BYDV, family: *Luteoviridae*, genus: *Luteovirus*) and *Cereal yellow dwarf virus* (CYDV, family: *Luteoviridae*, genus: *Polerovirus*)

BYDV was detected as early as the 1950s (Oswald and Houston, 1951) and is now known worldwide. The viruses can affect the yield of all cereals and grasses, for instance, in wheat, yield losses of about 50% and more are reported (Riedell et al., 1999). The virus is most important in winter wheat, but in some geographic regions spring wheat can also be affected. The disease incidence is dependent on the temperature, the presence of virus reservoirs and aphid vectors in autumn after sowing. The main symptoms in wheat are dwarfing of shoots, leaf yellowing and reddening (Fig. 2E), reduced number and sterility of ears, delay in heading and reduced winter hardiness.

The BYDVs are transmitted by different aphid species in a persistent and circulative manner. In the actual virus taxonomy, five BYDVs (BYDV-MAV, -PAV, -PAS, -kerII and -kerIII) have been assigned to the genus *Luteovirus* and RPV, RPS (*Rhopalosiphum padi* Severe) and RMV are assigned as CYDV-RPV, CYDV-RPS and *Maize yellow dwarf virus* (MYDV-RMV) within the genus *Polerovirus*. In addition, there are some species (BYDV-GPV and -SGV) that are unassigned till now (King et al., 2011; Krueger et al., 2013; www.ictvonline.org/virustaxonomy.asp). Further species have been identified in China, for example, BYDV-GAV, BYDV-PAV-CN and Wheat yellow dwarf virus (WYDV). BYDV-PAV with its

vectors *Rhopalosiphum padi* and *Sitobion avenae* is the most prevalent BYDV serotype worldwide, followed by BYDV-MAV (Domier, 2008). The virions are isometric with about 25 nm in diameter (King et al., 2011).

Besides agronomic measures, like optimal sowing dates and elimination of volunteer plants, insecticide spraying and seed treatment are used to reduce the virus incidence. But the effectiveness of these control methods is strongly dependent on the weather conditions after sowing and furthermore in some countries the seed treatment with neonicotinoides as an active component is no longer allowed. Therefore, growing of resistant cultivars is the most effective and environment-friendly method of control. However, against BYD, no complete resistance is known in common wheat and related *Aegilops* species. A partially effective tolerance gene inherited in an incomplete dominant manner, designated *Bdv1* and located on chromosome 7DS, was detected in the wheat cultivar Anza and in nine additional wheat lines (Qualset et al., 1973, 1984; Singh et al., 1993). Besides this, seven QTLs were identified in the Brazilian cultivar Frontana, which may also be the source of *Bdv1* (Ayala et al., 2002).

In the past 15 years, many efforts have been made to transfer the high level of BYDV resistance found in several perennial wild relatives (tertiary gene pool), for example, in *Thinopyrum intermedium*, *Th. ponticum*, *Leymus multicaulis* and *Roegneria ciliaris*, into wheat (cf. e.g. to Zhang et al., 2009). Three resistance genes, namely *Bdv2*, *Bdv3* and *Bdv4*, were identified in *Th. intermedium* and transferred to cultivars in Australia. The various resistance genes are differently effective against BYDV, CYDV and MYDV (Barloy et al., 2003; Chain et al., 2005). Several SSR- and STS markers (Ayala et al., 2001, 2007; Zhang et al., 2004; Gao et al., 2009; Kong et al., 2009) were developed to identify the different *Thinopyrum* chromosome segments and facilitate efficient marker-based selection for BYDV resistance in wheat.

5.2.2 *Wheat dwarf virus* (family: *Geminiviridae*, genus: *Mastrevirus*)

WDV was first detected in the former Czechoslovakia (Vacke, 1961), and is now widely distributed in Europe. The virus is also present in Asia (Xie et al., 2007; Wu et al., 2008) and Africa (Najar et al., 2000; Kapooria and Ndunguru, 2004). WDV is transmitted persistently by the leafhopper species *Psammotettix alienus*. Furthermore, Ekzayes et al. (2011) detected *P. provincialis* as a vector in Syria. The virus attacks wheat, barley, oat, rye, triticale and different species of grasses. Symptoms of WDV infection are chlorosis, reddening and streaking of leaves and strong dwarfing of the whole plant (Fig. 2F). Local epidemics can cause high yield losses in winter wheat (Lindblad and Waern, 2002; Širlová et al., 2005). In Hungary, WDV is the most important viral pathogen of winter wheat (Pribék et al., 2006). The virions of WDV are isometric twinned particles of about 20 × 30 nm (King et al., 2011). Three strains, namely a wheat, a barley and a oat strain of the virus, were described, which can be distinguished by their host range and sequence polymorphisms (Lindsten and Vacke, 1991; Commandeur and Huth, 1999; Ramsell, 2007; Schubert et al., 2007).

Because of the high mobility of the leafhoppers, chemical control of the virus vectors by insecticides is not effective. Therefore, growing of resistant cultivars is the most effective method to control WDV. But information about the sources of resistance have been very rare. In investigations of Vacke and Čibulka (2000), some genotypes showing a reduced virus titre were identified and also some are known showing only a slight reduction in yield

after infection (Bartoš et al., 2002). Besides this, Benkovics et al. (2010) detected partial resistance in the cultivars Mv Vekni and Mv Dalma characterized by a strong reduction in symptom expression, lower infection rates and reduced viral DNA accumulation, and recently *Aegilops tauschii* was identified as a possible source of resistance (Nygren et al., 2015). A comprehensive overview on WDV is given by Abt and Jacquot (2015).

5.3 Mite-transmitted viruses

5.3.1 *Wheat streak mosaic virus* (family: *Potyviridae*, genus: *Tritimovirus*)

WSMV, first recognized in the Great Plains of the United States in the 1920s (McKinney, 1937), is one of the most important viral diseases of wheat (*Triticum aestivum*). The virus causes periodically epidemics in Canada and the United States with dramatic yield losses ranging from 30% to 95% (Bockus et al., 2001). The virus was also detected in Europe, Russia, Mexico and Australia. Leaves of infected plants show yellow-green streaks (Fig. 2G) occurring at a temperature higher than 10°C. The stunted plants often develop a rosette. Besides yield losses, the milling properties of grains from infected plants are also negatively affected (Atkinson and Grant, 1967). WSMV is transmitted by the wheat leaf curl mite *Aceria tosichella* Keifer (syn. *Aceria tulipae* Keifer), which is spread by wind. Also, seed transmission at a rate of up to 1.5% was reported (Lanoiselet et al., 2008). Maize and various wild grass species are also natural hosts of the WSMV. The virus consists of flexuous rod-shaped particles with 15 nm in diameter and 700 nm in length (King et al., 2011). Disease control measures include the limitation of virus-infected volunteer plants and of grasses, as well as delaying the sowing time in autumn. The efficiency of the currently registered acaricides (pesticides) is economically not sufficient (Velandia et al., 2010). Potential sources for resistance are present in wide relatives of wheat, not only to the virus but also to the vector; for example, in several *Thinopyrum* species and numerous wheat-*Thinopyrum*, partial amphiploids have been developed and have turned out to be resistant to WSMV (Cox et al., 2002; Li et al., 2004). Three temperature-sensitive resistance genes, namely *Wsm1*, present in cv. Mace (Graybosch et al., 2009), *Wsm2* (Lu et al., 2011) and *Wsm3*, present in cv. RonL (Zhang et al., 2014), have been detected. PCR-based markers for the detection of these genes (Talbert et al., 1996) and for the detection of additional introgressions from *Th. intermedium* have been developed (Chen et al., 2003). The lines containing the gene *Wsm1* and all partial amphiploid lines, except cv. Agrotana, turned out to be susceptible to the wheat curl mite (Chen et al., 1998; Li et al., 2004). Vector resistance has been transferred into wheat from rye (*Secale cereale* L., e.g. Martin et al., 1984), *Aegilops tauschii* Coss. (syn. *A. squarrosa* L., e.g. Malik et al., 2003), *Th. ponticum* (Whelan and Hart, 1988), *Triticum timopheevii* var. *araraticum* (Brown-Guedira et al., 1996) and *Haynaldia villosa* (Li et al., 2002). However, it has to be taken into account that the resistance in some *Th. intermedium*-derived germplasms is temperature sensitive (Seifers et al., 2006, 2007) and is not effective to different strains of *Aceria tosichella* (Harvey et al., 1999). In parallel to the efforts to introgress WSMV resistance from different wild species into *Triticum aestivum*, the evaluation of winter and spring wheat cultivars was done and WSMV-tolerant accessions and cultivars were identified (Bottacin and Nassuth, 1990). Lehnhoff et al. (2015) demonstrated that the expression of WSMV resistance or tolerance in *Triticum aestivum* is influenced by environmental conditions and by the tested virus isolate.

6 Conclusions

Fungal and viral pathogens are reported to be causing high yield losses in wheat on a global scale, and due to climate change there will be a shift in the importance of these pathogens, e.g. it may be assumed that in the northern wheat-growing areas, insect-transmitted viruses will become more important. Modifications in agricultural practices, for example, reduced or no tillage and shorter crop rotations, have fostered the importance of fungal pathogens like *Fusarium* species. Furthermore, it has to be taken into account that pathogens develop resistance to pesticides (e.g. *B. graminis* to azoles) and are more or less continuously overcoming wheat resistance genes, as recently shown for the stem rust race *Ug99*. Consequently, there is a continual need (1) to adapt wheat production systems to the changing importance of pathogens, (2) to develop new effective fungicides and insecticides and especially (3) to improve resistance of wheat. Resistant cultivars are the most cost-effective and environment-friendly method of plant protection and are a prerequisite for any sustainable wheat production system. To reduce yield losses caused by biotic stresses, an efficient collaboration between phytopathologists, geneticists, breeders and specialists on wheat production is needed. As all wheat production starts with seed, breeding for resistance is of prime importance in this respect. Improving resistance in the first step requires identification of genetic resources in wheat and its wild relatives, followed by the analyses of genetics of resistance and the development of molecular markers. The availability of genomic tools in wheat will speed up the isolation of resistance genes which, in turn, will facilitate the detection of new and, perhaps more efficient, alleles e.g. in large gene bank collections, and the creation of new alleles by site-directed mutagenesis. In the future, breeding for resistance may take place at the allele level and therefore the process may become more effective. Cultivars with a high level of resistance to the most prevalent pathogens in their growing area will ensure yield and facilitate the development of sustainable wheat production systems by allowing soil-conserving production systems with minimal input of pesticides, thereby reducing site effects on non-target organisms, environment and consumers.

7 Where to look for further information

Besides information provided in this chapter, recent information on wheat and also to some extent on wheat diseases can be found on the homepage of Wheat Initiative (http://www.wheatinitiative.org/). The website is developing a WheatVIVO portal, which will provide access to information on wheat researchers and projects globally, and an International Wheat Information System, which will provide a single-access web-based system to access the available data resources. An index of fungal diseases of wheat and other cereals is available on the web page of the American Phytopathological Society (APS, www.apsnet.org/EDCENTER/INTROPP/LESSONS/FUNGI/Pages/default.aspx). Different authors present information on symptoms, pathogen biology, disease cycle and methods to biologically or chemically control pathogens. Furthermore, North Dakota State University provides an overview of symptoms caused by pathogens (https://www.ag.ndsu.edu/pubs/plantsci/smgrains/pp1552.pdf), and Agriculture and Horticulture Development Board provides management strategies (https://cereals.ahdb.org.uk/). Detailed information on rusts can be found at the homepage of

Borlaugh Global Rust Initiative (http://www.globalrust.org/). In addition to this, CIMMYT (International Maize and Wheat Improvement Center) offers much more information and also the downloadable book Wheat Diseases and Pests: A Guide for Field Identification at http://repository.cimmyt.org/xmlui/handle/10883/1115 (Wheat diseases and pests: a guide for field identification. 2012. Duveiller, E., Singh, P. K., Mezzalama, M., Singh, R. P., Dababat, A. A. 138p. Mexico, DF (Mexico). CIMMYT).

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