European Journal of Plant Pathology **105:** 629–641, 1999.

Mini review

The septoria tritici and stagonospora nodorum blotch diseases of wheat

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Accepted 20 April 1999

Key words: wheat, *Triticum* spp., *Septoria tritici*, septoria tritici blotch of wheat *Stagonospora nodorum*, stagonospora nodorum blotch of wheat

Introduction

Septoria tritici and stagonospora nodorum blotch of wheat are regarded as major diseases because of their impact on crop management and wheat production (Bayles, 1991; Eyal et al., 1987; King et al., 1983; Lovell et al., 1997; Meien-Vogeler et al., 1994). The wheat pathogens, *Septoria tritici* Roberge in Desmaz. (teleomorph *Mycosphaerella graminicola* (Fuckel) J. Schrot in Cohn and *Stagonospora nodorum* (Berk.) E. Castellani and E.G. Germano syn. *Septoria nodorum* (Berk.) in Berk and Broome (teleomorph *Phaeosphaeria nodorum* (E. Muller) Hedjaroude, syn. *Leptosphaeria nodorum* E. Muller) (Cunfer, 1997) are distinct from each other in many ways (Eyal, 1999a). *S. tritici* is considered to be more confined to Mediterranean-type climates (wet winters with temperate temperatures), whereas *S. nodorum* is more common in northern latitudes (Leath et al., 1993). The pathogens have been reported from all continents except for most parts of South-west Asia (Eyal et al., 1987). A shift in prevalence and economic importance from *S. nodorum* to *S. tritici* has occurred in the UK (Jones, 1985; Polley and Thomas, 1991; Royle et al., 1986; Shaw, 1999) and probably in Germany, where the two pathogens co-existed together in the wheat management systems. This was attributed to the widespread growing of susceptible cultivars, early sowing, increased nitrogen fertilizer, high summer rainfall and probable differential response to certain fungicides (Bayles, 1991; Lovell et al., 1997; Meien-Vogeler et al., 1994; Polley and Thomas, 1991; Shaw, 1999). A reverse shift from *S. tritici* to *S. nodorum* was reported from Morocco (Farih and Ezzahiri, 1996) where *S. tritici* predominated in the population (Saadaoui, 1987).

Host range

A wide range of graminaceous hosts in nature and using artificial inoculations are susceptible to both *S. tritici* and *S. nodorum*. Isolates of *S. nodorum* from 11 different species of infected leaves of *Agropyron* spp., *Elymus* spp., *Hordeum jubatum* and *Leymus* spp. all caused symptoms on seedlings of six wheat cultivars (Krupinsky, 1989). Isolates from alternative grass hosts showed stable, low aggressiveness on wheat and probably would not adapt easily to wheat; thus they do not pose a threat to wheat (Krupinsky, 1997a). The existence of wheat- and barley-adapted types which were considered as biotypes or *formae speciales* was reported in *S. nodorum* (Cunfer and Youmans, 1983; Smedegard-Peterson, 1974). Differences between the wheat- and barley-types of *S. nodorum* suggested that there are genetically distinct populations within the species (Newton and Caten, 1991).

Agropyron spp., *Agrostis* spp., *Brachypodium* spp., *Bromus* spp., *Dactylis* spp., *Festuca* spp., *Hordeum* spp., *Glyceria* spp., *Poa* spp., *Secale cereale* and *Triticum* spp. were all suggested as possible alternative hosts to *S. tritici* (Eyal, 1999a). The ability to infect and sporulate on artificially inoculated leaves of alternative hosts does not necessarily indicate their role in

†We have learned with great sadness that Professor Zahir Eyal died on 31 July 1999 (Ed.).

the epidemiology of this pathogen. Pycnidia of *S. tritici* can be found on plants of the indigenous wild-emmer wheat (*Triticum turgidum* var. *dicoccoides*) in natural stands in Israel. The role of wild-emmer as a source of primary inoculum in inciting epidemics on commercial durum (*T. durum*) or common wheat (*T. aestivum*) and its contribution to the virulence spectrum of *S. tritici* is not known.

Onset of epidemics

The primary sources of inoculum in the disease-cycle of both pathogens are airborne ascospores, rain-splashed pycnidiospores from infected plant debris and possibly seed-borne infections. A principal role for airborne ascospores discharged from mature pseudothecia is the onset of and probably perpetuation of epidemics. The environmental factors associated with ascospore discharge (rain duration, intensity, wind and temperature) and the resulting infection intensity on wheat genotypes have been used in formulating disease models with implications for forecasting and disease control (Royle, 1994; Parker et al., 1999). Uniform spatial distribution of infection by *S. tritici* found throughout the growing season by Shaw and Royle (1989) may be indicative of a distant airborne inoculum source. Upward vertical transport of rain-splashed pycnidiospores from the base of the crop to the upper leaves is affected by penetrating raindrops and canopy structure (distance between leaves, leaf length and posture). Hunter et al. (1999) and Zhan et al. (1998) reported that a continuous within-crop supply of ascospores from lower to upper leaves is operative. The within-crop ascospore distribution may have bearings on both short and long distance spore dissemination and on the possible availability of greater genetic variability in pathogenicity factors in the ascospore-operated system (Hunter et al., 1999; Royle et al., 1986; Shaw and Royle, 1989), presumably unmatched by an epidemic generated solely by pycnidiospores (Eyal et al., 1987; Lovell et al., 1997).

In Mediterranean-type countries, rain, which serves as a major driving force of the epidemic, remains a limiting factor and severe epidemics are therefore sporadic, unless sprinkler irrigation is implemented (Eyal, 1971). Long rainless intervals may limit both horizontal spread and vertical progression to upper leaves, which are responsible for grain filling (Eyal et al., 1987). Such factors often interfere with assessments of host response of wheat germplasm under field conditions, where the pathogen may remain on the lower leaves, a situation which can be interpreted as a resistant host response rather than a consequence of no rain-driven disease progression. Tall plant stature and late maturity often contribute to lower disease coverage on the upper leaves, which can also be interpreted as resistance (Scott et al., 1982; Eyal et al., 1987; Van Beuningen and Kohli, 1990).

Seedling infection from infected wheat kernels is a primary source of inoculum for *S. nodorum*. Cockerell and Rennie (1996) reported that in 1992–93 more than 40% of the certified seed sampled in Britain had at least 20% seeds infected with *S. nodorum*.

Seeds infected with *S. tritici* were reported by Brokenshire (1975) for artificially inoculated seeds of *T. dicoccum*. Brokenshire (1975) stated however that 'infecting seedlings from infected untreated seed samples has proved unsuccessful with *T. dicoccum*'. The reports on possible seed transmission of *S. tritici* were used in suggesting this transmission mode as a driving force for gene-flow on a global scale (McDonald et al., 1995). The epidemiological implications of naturally infected grasses and seeds for *S. tritici* in short dissemination and long-distance transport, warrant additional investigations.

The infection process

Information on ascospore liberation, dissemination, viability, and long-distance movement is lacking. Ascospores of the sexual states, *M. graminicola* (2-celled spore) and *P. nodorum* (4-celled spore) serve as the primary source of inoculum in the initiation of epidemics of the two pathogens. The two pathogens also have distinctly different pycnidiospores, though both germinate by elongation of the apical cells or by budding. The liberation of pycnidiospores from the pycnidium is gradual with the great majority exuded during the first wetting, while the rest are released on subsequent wettings. The number of liberated pycnidiospores of *S. tritici* from a pycnidium may range between 5 and 10×10^3 (Eyal, 1971). Under a scaled pycnidial coverage of 90%, the actual observed pycnidial coverage was 20.6% per unit leaf area (Eyal and Brown, 1976). In a 10 cm^2 leaf with such a pycnidial density, 3×10^6 pycnidiospores are available for the infection process, depending on pycnidial size, age and maturity. It is assumed that the same relationship holds true for wheat heavily infected with *S. nodorum*. The large number

of available pycnidiospores compensates for the inefficient, rain-dependent splash dispersal mechanism. The relationship between the number of incoming ascospores to the infection court and the establishment of lesions bearing pycnidia, and thereafter the progression of the epidemic from additional showers of ascospores, from airborne ascospores within the crop, and from horizontal and vertical distribution of rain-splashed pycnidiospores, warrants more attention.

The two pathogens differ in the mode of penetration of the germinating pycnidiospores. The almost exclusive penetration by *S. tritici* through stomata (Cohen and Eyal, 1993; Kema et al., 1996c) distinguishes this pathogen from *S. nodorum* which was shown to penetrate directly through the cuticle (Karjalainen and Lounatmaa, 1986). Formation of pycnidia in *S. tritici*-infected leaves occurs solely in conjunction with stomata; the linear arrangement of pycnidia follows the linear pattern of stomata along the veins. Although pycnidial shape appears to be determined by the contours of the sub-stomatal chamber in which it is formed (Cohen and Eyal, 1993; Kema et al., 1996c), the relationship seems tenuous since similarly shaped pycnidia were produced on artificial medium (Zelikovitch and Eyal, 1989). Pycnidial formation on seedlings of susceptible cultivars grown under certain environmental conditions seems to occur 14–21 days after inoculation for *S. tritici* and 7–14 days for *S. nodorum*. Royle et al. (1986) proposed that because of the shorter incubation period, attacks by *S. nodorum* (2–4 weeks) will be more damaging than those of *S. tritici* (3–5 weeks).

The factors triggering and contributing to pycnidial formation (whether they be independent of or dependent on mycelial biomass or physiological stresses), need elucidation. Kema *et al*. (1996c) reported that mycelial biomass was a determinant in pycnidial formation in *S. tritici*. The use of reporter gene(s) (e.g. GUS, GFP) in genetically transformed *S. tritici* and *S. nodorum* isolates and immunological assays can greatly contribute to the understanding of *in planta* qualitative and quantitative post-inoculation events (Cooley et al., 1988; Kema et al., 1996c; Payne et al., 1997; Pnini-Cohen et al., 1997b, 1998).

The direct penetration of wheat tissue by germinating pycnidiospores of *S. nodorum* through the periclinal wall into the lumen of epidermal cells, is accompanied by the formation of a penetration peg and possibly by enzymatic degradation of the wheat cuticle (Karjalainen and Lounatmaa, 1986). Polygalacturonase, xylanase and cellulase were produced in mineral medium supplemented with wheat cell walls and in wheat leaves inoculated with *S. nodorum*, with an apparent lysis of the cutin layer (Lehtinen, 1993).

Colonization of host tissue, cell collapse, and pycnidial formation were associated with the reaction of wheat cultivars to the two pathogens (Eyal et al., 1987; King et al., 1983). Unsuccessful penetrations by *S. nodorum* were associated in part with papilla formation followed by lignification which consequently reduced infection and colonization, though this phenomenon was not restricted to resistant cultivars (Zinkernagel et al., 1987). There is no report that substantiates the formation of papillae in wheat tissue infected by *S. tritici.* It is likely that fluorescing products at the invaded stomata do not provide a sufficient barrier to halt infection by the invading pathogen, but rather affect the rate of mycelial development *in planta* at the onset of infection (Pnini-Cohen et al., 1997b). In resistant wheat cultivars inoculated with *S. tritici* which harbored none-to-few pycnidia, mycelium was more restricted to the sub-stomatal cavity (Kema et al., 1996c). Fungal mycelium was limited to the intercellular spaces between epidermal and mesophyll plant cells. Disruption in chloroplast integrity (size and shape) and their compartmentalization within the host cells was reported by Karjalainen and Lounatama (1986) for *S. nodorum* and by Kema et al. (1996c) for *S. tritici*. Disorientation of mesophyll cells and the collapse of host tissue several days after inoculation were ascribed to the production of soluble phytotoxic compounds. Two families of phytotoxic compounds are produced by *S. nodorum*: mellein (= ochracin, -4-hydroxymellein, -5-hyroxymellein and -7-hydroxymellein) and septorin (*N*-methoxyseptorine and *N*-methoxyseptorinol) which cause the uncoupling of mitochondria in wheat coleoptiles. Information on the direct involvement of mellein and septorin phytotoxins in the infection process and in the specificity of *S. nodorum* is lacking. Despite circumstantial evidence (disorientation of cell compartmentalization, collapse of host tissue, and the necrophytic response) which strongly suggests the involvement of phytotoxin(s) in the infection of wheat by *S. tritici*, no biologically active, pathogen-produced compound has been isolated. It is possible that such toxic compounds and their cumulative effect provides a stressed environment within the host tissue that is associated with the formation of symptoms (necrosis, pycnidia) (Zelikovitch et al., 1992).

Speciation and specificity

The issue of specificity in the wheat–*S. tritici* and wheat–*S. nodorum* pathosystems has been discussed extensively in the literature (Allingham and Jackson, 1981; Eyal et al., 1973; Eyal et al., 1985; Kema et al., 1996a,b; Krupinsky, 1997a; Saadaoui, 1987; Scharen et al., 1985). The issue remains somewhat unsolved, partly because of the difficulties in interpreting the quantitative host responses in the wheat–*S. tritici* and wheat–*S. nodorum* pathosystems which govern the interaction.

Septoria tritici

Eyal et al. (1973) reported the existence of physiologic specialization in *S. tritici* and stressed that in the *S. tritici*–wheat pathosystem, host response is quantitative (% necrosis, % pycnidia coverage) rather than qualitative. Possible specialization on *Triticum* species and statistically significant isolate × cultivar interaction terms were calculated. *S. tritici* isolates obtained from cultivars of durum wheat (*T. durum* Desf.) produced low levels of pycnidia on most common wheat cultivars (*T. aestivum* L.) tested, but high coverage on the durum wheat Etit 38. Conversely, isolates derived from common wheat produced high levels of pycnidia on all *T. aestivum* cultivars, except the resistant winter wheat cv. Racine, and low levels on Etit 38. In countries where durum wheat cultivars predominate, the pathogen's population is skewed towards durum-adapted virulence (Eyal et al., 1985; Saadaoui, 1987; Jlibene et al., 1995; Kema et al., 1996a). However, in these populations it is also possible to find *T. aestivum*- or *T. aestivum/ T. durum*- adapted isolates. Kema et al. (1996a,b) proposed the designation of two varieties in *M. graminicola*, common wheat- and durum wheat-adapted isolates to the respective *Triticum* species. In Israel, where durum and common wheat are grown, *S. tritici* isolates can be categorized into distinct groups on the basis of their origin and ability to infect *Triticum* species: (a) isolates originating from *T. aestivum* that cause symptoms on both common and durum wheat or those that are restricted to common wheat, and (b) isolates originating from *T. durum* that exclusively infect durum wheat and those that infect both wheat species.

Variation in virulence patterns within and between populations of *S. tritici* was shown by assessing host response (mostly % pycnidial coverage) on a selected set of cultivars, with little commonality apparently between results obtained wheat genotypes composing the differential set of cultivars (Eyal, 1995). Evidence for specificity was also confirmed on adult plants of field-grown wheat cultivars inoculated with specific isolates (the same isolates and cultivars as used in corresponding seedling studies) (Danon and Eyal, 1990; Ezrati et al., 1997; Kema and van Silfhout, 1997a).

The sexual state may have an impact on the virulence spectrum in regions where pseudothecia were found and ascospore dispersal coincided with the wheat growing cycle (Royle et al., 1986; Shaw and Royle, 1989; Lovell et al., 1997). Using RFLPs and DNA fingerprints, Chen and McDonald (1996) concluded that where sexual reproduction in *M. graminicola* is playing a role in the epidemic, new virulence combinations can be selected by corresponding host resistance genes. They further suggested that new recombinations of matching virulence genes will overcome pyramided resistance genes. Zhan et al. (1998) monitored the frequency of *S. tritici* isolate mixtures originating from two wheat cultivars by nine individual RFLPs and one DNA fingerprint. The frequency of novel isolates detected in the inoculated plots increased from 0.03 to 0.34 over the course of the season, of which 43% were immigrants and 57% resulted from recombination between the 10 inoculated isolates. The combined effects of ascospore distribution, long distance and within-crop dissemination are likely to increase the chances for the appearance of new recombinants. The continuous development of the sexual phase infers that there is genetic exchange throughout the growing season (Hunter et al., 1999). As a consequence, the pathogen may respond over time to selection exerted either by the introduction of new cultivars or by the application of site-specific fungicides. The question of whether recombinants with new virulence combinations are also being produced has great implications for population structure and breeding for disease resistance. The high genetic diversity in nuDNA and mtDNA (nuclear and mitochondrial DNA, respectively) recorded by McDonald et al. (1999) in Nahal-Oz, Israel, strongly implied the presence of the sexual state. It was further suggested that the Middle East could be the centre of diversity and probably the centre of origin for this fungus. It should be noted that isolates of *S. tritici* sampled from California and Oregon (where the sexual state is present) differed significantly in their virulence (Ahmed et al., 1996), yet they manifested a very high degree of similarity between the two

regions for the frequency of selectively neutral alleles as measured by RFLPs markers (McDonald et al., 1996). This discrepancy suggests that the selectively used neutral DNA markers may not be closely related to the expressed *S. tritici* virulence patterns.

The heterothallism reported for both *S. tritici* (Kema et al., 1996d) and *S. nodorum* (Newton and Caten, 1991; Halama, 1999) and the ability of isolates of different mating types and probably avirulence to cross provide the needed tools for a range of genetic studies.

Crosses between *S. tritici* isolate IPO323 (MAT1-1), avirulent on wheat cvs Kavkaz, Shafir and Veranopolis and IPO94269 (MAT1-2), virulent on these cultivars, indicated that avirulence segregated as a monogenic character and that the two isolates carry the same avirulence factor(s) for Kavkaz/K4500 L.6.A.4 (Kema et al., 1999). The avirulence locus segregated independently from the mating-type locus (Kema et al., 1998, 1999). A linkage map of *M. graminicola* was produced using the AFLP technique (Kema et al., 1998). AFLP analysis yielded 24 linkage groups, whereas pulsed field gel electrophoresis (PFGE) used for electrophoretic karyotyping resulted in 16–18 chromosomes. The difference was attributed to possible co-migration of chromosomes in the PFGE (Kema et al., 1999). The avirulence and mating-type loci were mapped on small linkage groups. It is hoped that these findings will serve as the basis for more detailed mapping of the genome and increased genetic knowledge of *M. graminicola.*

The presence of a gene-for-gene interaction in the wheat-*S. tritici* pathosystem was inferred from the statistical variance of the interaction term (cultivar × isolate) of host response (pycnidial coverage) (Eyal et al., 1985; Kema et al., 1996a,b). The expression of virulence of certain well-studied *S. tritici* isolates on specific wheat cultivars is distinct, repeatable, and highly stable, despite more than 30 years of sub-culturing on artificial media and re-isolation from pycnidia on infected leaves (Eyal, 1992; Zilberstein et al., 1993). However, no attempt was made to assign race connotation to isolates with distinct virulence patterns on a set of differential cultivars. Mundt et al. (1999) showed that isolates collected from different cultivars in the field demonstrated adaptation of the pathogen to each respective cultivar, namely, quantitative variation in virulence. In addition, isolates were found to be more adapted to cultivars from the same region of origin in comparison to cultivars from other regions.

Septoria tritici isolates were genetically transformed with the selectable marker gene *hph* (hygromycin phosphotransferase) which confers resistance to hygromycin B and the reporter gene *uid*A which encodes the enzyme β-glucuronidase (GUS) reporter gene (Pnini-Cohen et al., 1997a,b). Mutants of *S. tritici* were recovered with an acquisition of virulence or with a loss in virulence (Pnini-Cohen et al., 1997a). The expression of virulence of the two *S. tritici* mutants was highly stable in culture and following repeated passages through pycnidia in inoculated wheat tissue. These results strengthen the notion that specificity and a gene-for-gene scheme is operative in the wheat–*S. tritici* pathosystem.

Marked suppression in pycnidial coverage was recorded on seedlings and in field-grown adult plants of specific cultivars when inoculated with a mixture of avirulent and virulent *S. tritici* isolates (Zelikovitch and Eyal, 1991; Eyal, 1992), or after sequential inoculation by these isolates (Halperin et al., 1996). The divergence from the expected host response on Seri 82 after inoculation with a mixture of *S. tritici* isolates was explained by the induction of resistance by the avirulent isolate in the mixture, where host product(s) resulting from the interaction suppress *in planta* mycelial development and the initiation and/or production of pycnidia by the virulent isolate in the mixture (Halperin et al., 1996; Pnini-Cohen et al., 1997b). Suppression in pycnidial coverage was confirmed by field trials for the same *S. tritici* isolates and the same wheat cultivars (Ezrati et al., 1997). In natural populations of *S. tritici*, cultivar × isolate interactions, aggressiveness and isolate–isolate interactions operate in unison to determine the structure of the pathogen population (Ezrati et al., 1997).

Stagnospora nodorum

Unlike *S. tritici*, specificity in the wheat–*S. nodorum* pathosystem is much less distinct. Allingham and Jackson (1981) tested cultivar × isolate relationships of 282 isolates of *S. nodorum* originating from Northern Florida. Despite the recorded differential interactions, the 253 response patterns were not categorized into conventional race classification. Virulence frequencies of 33 *S. nodorum* isolates from eight countries were assessed by Scharen et al. (1985) on 38 wheat and triticale accessions based on percent leaf necrosis. Significant main effects of isolates and cultivars and a non-significant interaction term were recorded in this study indicating a lack of clear specificity in the wheat–*S. nodorum* pathosystem. Isolates of

S. nodorum obtained from Montana, North Dakota and South Dakota exhibited low levels of specificity unless extreme isolates were tested (Krupinsky, 1997a). Isolates with high and low aggressiveness were detected. It appears that in *S. nodorum*, physiologic specialization much less pronounced outside a specific experimental situation. Moderate levels of specificity were reported for populations of *S. nodorum* in Switzerland where sexual reproduction contributed to high levels of genotypic variation (Keller et al., 1997); earlier studies had shown limited interactions in the wheat–*S. nodorum* pathosystem in this country (Fried and Mister, 1987). *S. nodorum* isolates from barley and wheat, which were highly virulent to their original hosts, proved to be weakly virulent to alternate species in reciprocal inoculations (Cunfer and Youmans, 1983). Similar results were recorded by Krupinsky (1989, 1997a,b) for *S. nodorum* originating from wheat and several grass species.

Control measures

The economic impact of both pathogens on wheat production (yield and quality) has become more pronounced in part due to the increased genetic resistance of wheat to other foliar pathogens, such as rusts and powdery mildew. This increased resistance to biotrophic pathogens was not accompanied by a control strategy for other 'minor' wheat diseases. Consequently, control measures geared to contain septoria tritici and stagonospora nodorum blotch pathogens of wheat had to be adjusted to their rather different life-cycles and modes of host resistance.

The fact that the distribution of these pathogens is dependent on wheat refuse management and the involvement of both airborne and splash-dispersal mechanisms should have dictated measures to reduce the amount of infected debris in the wheat cropping system. Moreover, conditions which influence dispersal of airborne inoculum and rain-splash from infected plant refuse and from within the crop are not necessarily the same. In most countries, wheat is grown under rain-fed conditions and under rather short crop rotation intervals between wheat cropping. Even in *S. tritici* and *S. nodorum*-prone countries, severe epidemics are sporadic, introducing a degree of uncertainty into designing control strategies. This uncertainty tilts the protection strategy of the crop towards short-term, stop-gap measures rather than long-term measures. Chemical control is more routinely used in high wheat productivity systems than under low crop productivity. Under high productivity systems and high septoria tritici/stagonospora nodorum blotch risks, yielding capacity can be more readily attained by combining pre-planting measures (management practices) with genetical protection (disease resistance), with post-planting measures designated to protect the carbohydrate assimilation and grain-filling machinery (chemical control). Under low productivity systems, growing conditions, environmental factors, and abiotic and biotic stresses may strongly affect the economics of yields and, as a consequence, reduce demands for high-energy inputs (irrigation, fertilizers, disease and weed control) and thus influence control strategy (Jordan and Hutcheon, 1999).

Cultural practices and chemical control

In management systems where wheat is planted in short consecutive yearly intervals, discarding infected stubble by ploughing, and/or burning can drastically reduce primary inoculum. Under growing conditions where the lower wheat leaves are in direct contact with infected wheat debris (e.g. mulching) the chances for the early onset of an epidemic are high. Where primary infection is caused by airborne ascospores and horizontal spread, and vertical progression is subsequently caused by splashing pycnidiospores or, as recently reported by Hunter et al. (1999), by within-crop ascospore dispersal, cultural control practices are more difficult to implement and the chances of disease avoidance by such practices are low. In less-intensive arable production regimes, minimizing the risk of disease can be achieved, according to Jordan and Hutcheon (1999), by multi-function crop rotation, combined with resistance, and manipulation of crop structure by adjustments in sowing date and nitrogen application (amount and timing), with a consequent decreased rate of disease development and a reduction in fungicide requirement. Bannon and Cooke (1998) reported that in wheat–clover intercrops, clover significantly reduced the horizontal and vertical movement of *S. tritici* pycnidiospores compared to a wheat monocrop. In field trials, however, both increases and decreases in the level of septoria tritici blotch in wheat–clover intercrops were recorded.

The increase in importance in recent years of septoria tritici blotch and the limitations it imposes on yield in winter wheat in the UK has intensified the research on disease prediction, disease risk and forecasting for the

optimization of fungicide application (Royle, 1994). The sporadic occurrence of septoria and stagonospora blotch diseases in the UK and other European countries within and between seasons and economic considerations allow for manageable disease control. The intensive epidemiological research conducted in the UK has contributed to our fundamental understanding of the interaction between the wheat crop and populations of the pathogen and to the practical knowledge required in designing an efficient and cost-effective disease control program (Royle, 1994).

Since the reviews of King et al. (1983) and Eyal et al. (1987) a variety of new fungicides have been recommended and used in controlling both pathogens. A wide variety (>20) of 14-demethylase inhibitors (DMI) of sterol biosynthesis, based on N-substituted 1,2,4-triazole and imidazole were introduced by the agrochemical industry, some of which are effective in controlling septoria and stagonospora blotch diseases of wheat (Jordan and Hutcheon, 1999; Lockley, 1997). Some of these compounds are effective against several other foliar pathogens of wheat. Resistance risk baselines are required to be established for key pathogens as legal requirements for registration of new active ingredients in Europe and the United States. Broad spectrum, systemic fungicidal natural products, which belong to the strobilurins (e.g. azoxystrobin) are being introduced (Godwin et al., 1999). Strobilurin A and Oudemansin A are natural products found in the Basidiomycete fungi *Strobilurus tenacellus* and *Oudemansiella mucida*, respectively. They share a common mode of action, the inhibition of mitochondrial respiration. Oxidation of ubiquinol is blocked at the Q_0-site of the cytochrome bc_1 located in the inner mitochondrial membrane of fungi. Resistance risk assessments indicated that the likelihood of *S. tritici* or *S. nodorum* populations developing resistance to strobilurins is moderate (Godwin et al., 1999).

Future development of antifungal compounds will rely more on the isolation and identification of genes that are essential to host infection and are expressed during the infection processes. Such studies will require a detailed understanding of mechanisms and processes associated with infection. *Septoria/Stagonospora* spp. have been chosen by several agrochemical companies as models for identification of novel fungicides through both biochemical (rational) design and high-throughput biochemical screening (Dancer et al., 1999). Utilizing molecular biology technologies, several fungicide targets were identified for *S. tritici*, including 4-Hydroxyphenylpyruvate dioxygenase (HPPD) an enzyme of the tyrosine catabolic pathway (Hargreaves and Keon, 1997). The gene encoding the iron–sulfur subunit of succinate dehydrogenase (Sdh) thus determining resistance to carboxin (Cbx) has been isolated from *M. graminicola* (Skinner et al., 1998). Using *S. nodorum* as a model, Bailey et al. (1997) used ornithine decarboxylase (ODC) which is involved in polyamine synthesis by catalyzing the conversion of ornithine to putrescine. The nitrate reductase gene (NIA1) that was cloned from *S. nodorum* by gene disruption was used as a method of fungicide target validation (Howard et al., 1997). The NIA1 gene was disrupted by co-transformation and gene replacement methods. The tested mutants all retained full pathogenicity thereby invalidating nitrate reductase as a fungicide target. The same approach was used in disrupting resident *S. nodorum* chitin synthase and assessing disruptants for reduced ability to infect wheat.

Breeding for disease resistance

The enhancement of yield stability of modern wheat cultivars through the incorporation of multiple resistance to plant pathogens is one of the major strategies used for increasing crop productivity. The marked decline in the level of resistance to *S. tritici* in commercial winter wheat cultivars in the UK during the late 1970s and early 1980s, reaching a plateau in 1982 and 1984, contributed to the increased importance of this pathogen (Bayles and Clarkson, 1997). Dubin and Rajaram (1996) have reported a gradual but steady increase in septoria tritici blotch resistance in spring wheat since the 1970s, prior to the introduction by CIMMYT of resistance from South American, Russian and Chinese sources.

Seedling resistance to *S. nodorum* and to *S. tritici* was identified in the diploid wheats *Aegilops longissima* (genome S^1S^1), *Ae. speltoides* (genome SS) and in *Ae. squarrosa* (= *Triticum tauschi*, genome DD) (Ma and Hughes, 1993). High levels of resistance to *S. tritici* were attained in wheat cultivars by incorporating resistance from *Thinopyrum* (*Agropyron*) *curvifolium* (Dubin and Rajaram, 1996).

Septoria tritici blotch

There are very few examples where resistance controlled by major genes was recognized and incorporated into specifically designated septoria tritici blotch

breeding schemes. The irregular, sporadic epidemics, the lack of knowledge on the virulence spectrum in this pathogen, the relevance of specificity (magnitude, durability) to breeding and the scarcity in resistance sources have complicated the design of a reliable incorporation scheme. Multi-location testing revealed differential responses in symptoms on genotypes classified as being resistant under certain conditions and identified new sources of resistance. The magnitude of national and global virulence patterns revealed a rather wide virulence spectrum on seedlings of tested wheat differentials with no defined spectrum assigned to a particular geographic region (Eyal, 1999b).

Most sources of resistance were first screened under natural and/or artificially inoculated field trials to a badly-defined (magnitude, relevance) virulence spectrum. Genetic studies designed to elucidate the mode of inheritance of resistance were conducted at the seedling stage, or in field trials inoculated in most cases with selected isolates of *S. tritici*. Seedling studies under controlled conditions provide accuracy and repeatability, may recognize specific resistance, but do not take into consideration possible differences in host response at the adult stage and other types of resistance, phenology and agronomic characteristics (Eyal, 1999b).

Resistance to *S. tritici* controlled by a single dominant gene was assigned to the winter wheat cv. Bulgaria 88 (Rillo and Caldwell, 1966) and to its derivative Oasis (CI 15929) (Shaner and Buechley, 1989); the latter expressed only moderate resistance in Israel. The expression of dominance in Bulgaria 88 can be modified by other genes, and by the physiological stage of the wheat plant. A single dominant gene was assigned to the Brazilian accessions IAS20-IASSUL (Colonias/Frontana/Kenya 58) and to Veranopolis (Trintecinco/Frontana) by Wilson (1979) in Australia, where the latter was inherited independently of the gene in Bulgaria 88. Resistance to *S. tritici* in the highly resistant spring wheat accessions Bobwhite 'S' (BOW) and Kavkaz/K4500 L.6.A.4 (KK) is controlled by several genes (mostly recessive), with predominantly additive effects (Jlibene et al., 1992; Matus et al., 1993). It is likely that the resistance of these highly resistant accessions and others is based on germplasm from the South American wheat accessions Frontiera (Polyssu/Alfredo Chavez 6.21) or Frontana (Frontiera/Mentana) and not on the Aurora-Bezostaya 1-Kavkaz winter wheat germplasm (Danon and Eyal, 1990; Eyal, 1995). Dubin and Rajaram (1996) suggested combining several genes (2–3) may impart an acceptable level of resistance and consequently lower the impact of the disease on yield. Information is still lacking on the adaptation of suitable incorporation and selection strategies, and the lasting effectiveness of recognized resistance sources upon increasing wheat hectarage and the distribution of improved cultivars.

Evidence for a decline in the effectiveness of resistance to *S. tritici* was recently reported by Ahmed et al. (1996) and Mundt et al. (1999) for the winter wheat cv. Gene. Mundt et al. (1999) suggested that host susceptibility selects for increased pathogen aggressiveness compared to host genotypes of lesser susceptibility. He proposed that this may be caused by differences in variance for aggressiveness of the pathogen on susceptible as compared to less susceptible hosts. Aggressiveness of an isolate in the population and its spatial ability to compete with less fit isolates combined with a broad virulence spectrum, may give such isolates an adaptive advantage in *S. tritici* populations in successive disease-cycles (Ezrati et al., 1997).

Wheat cultivars which express a consistent host response over time not associated with plant height and maturity (days to heading), combined with low-moderate pycnidial coverage (<20%) are likely to possess a more stable type of resistance (Eyal and Talpaz, 1990). Under low disease pressure, such cultivars would exhibit low disease coverage, while under high disease pressure a moderate pycnidial coverage could be expected. These cultivars would therefore show stable resistance to variable populations of the pathogen. A similar approach for selecting germplasm with non-specific resistance was suggested by Van Beuningen and Kohli (1990). It remains to be seen whether this type of protection is heritable and can be selected for inbreeding populations.

Stagonospora nodorum blotch

Resistance to *S. nodorum* on leaves was reported to be independent of resistance on the spikes, and therefore seedling tests cannot replace field evaluations (Fried and Meister, 1987). The inheritance of both leaf and spike resistance was explained by Fried and Meister (1987) by the additive-dominance mode. The inheritance of resistance to stagonospora nodorum blotch was studied by Bostwick et al. (1993) in the Brazilian wheat cv. Cotipora (Veranopolis∗2/Egypt NA101) which expresses a low percentage of diseased tissue on both leaves and spikes. These authors suggested that combining the flag leaf resistance of Coker 84-27

with Cotipora's leaf and spike resistance may greatly improve the overall resistance level. Monosomic analysis of resistance in Cotipora suggests that common genes are found on chromosomes 3A, 4A and 3B for flag leaf and spike resistance, but their magnitude may be different (Hu et al., 1996). Assuming one gene per chromosome, a minimum of four genes, on chromosomes 3A, 4A, 7A and 3B, are responsible for spike resistance. This gene number correlates well with the 3.16 gene number estimated by Bostwick et al. (1993).

Disease severity (on leaf and spike), plant height, days to anthesis and ear morphology were analyzed in recombinant F_5 inbred lines (RILs) derived from the cross between the winter spelt wheat (*T. spelta* L., genome AABBDD) Oberkulmer and the winter bread wheat Forno (Messmer et al., 1997). The 121 RFLP clones and microsatellite markers tested on 226 recombinant lines yielded 156 segregating loci. The investigators reported up to seven QTLs (Quantitative Trait Loci) for resistance to leaf infection, and seven others associated with spike resistance, with one QTL common to both leaf and spike resistance. QTL analysis is aimed at the development of tools that will be useful in marker-assisted selection for resistance to stagonospora nodorum blotch of wheat. Alignment of QTL with candidate-cloned genes or characterized sequences based on anchored markers may have great potential in the identification of the actual underlying mechanisms of the QTL. Placement of cloned genes and characterized sequences on comparative maps, as suggested by Messmer et al. (1) for *S. nodorum,* can facilitate the identification and selection of direct markers for QTL. This still does not however exclude the possibility that the candidate gene is not the target gene but rather a tightly linked locus.

Conclusions and future developments

Research on *S. tritici* and *S. nodorum* has greatly expanded over the last 30 years, and may continue at a faster pace in the future due to the economic impact of these pathogens on grain production in high-input wheat management systems (such as those found in Europe). Despite the long-recognized economic impact of these pathogens on wheat production, there are some basic issues which deserve more research effort, especially the biology of these pathogens and host–pathogen interactions. Issues associated with the ability to infect wheat and processes related to the infection-cycle (biochemical processes, toxin production, tissue necrosis, pycnidial formation and production), virulence (genotypic diversity and population structure), epidemiology and disease management (initial sexual or asexual inoculum, pathogen migration, effect of genotypic diversity in the pathogen), genetic host resistance (specific, non-specific, effect of host growth stage and phenology, biochemical and molecular studies) and resistance management (at national and international levels using novel technologies) need more attention. Considerable efforts are currently invested in chemical protection measures and associated disciplines but less in resolving long-term solutions which may provide lasting protection. It is likely that new molecular, genetical, and biochemical technologies will enable us to revive and explore biological issues associated with the two pathogens, their host, and the host–pathogen interaction which have been neglected because of the lack of appropriate research tools. Harnessing genetical crosses in *M. graminicola* (Kema et al., 1996d) and *P. nodorum* (Halama, 1999), together with defined molecular markers, will greatly contribute to the elucidation of the genetics of virulence and aggressiveness in these pathogens and will help us to understand the potential of genetic recombination on pathogen structure and thereafter on resistance management (Caten, 1999; Pnini-Cohen et al., 1998). The combined use of crosses with mapping technologies have resulted in the creation of a linkage map of the *M. graminicola* genome (Kema et al., 1998). Genetical and molecular analyses of mutants with altered expressions of virulence may contribute to the identification, mapping and possibly cloning of genes and their products associated with the infection process.

The use of genetically transformed isolates of both *S. nodorum* (Cooley et al., 1988, 1991) and *S. tritici* (Pnini-Cohen et al., 1996, 1998; Payne, 1997) and the incorporation of traceable reporter genes open up new avenues for detailed analyses (qualitative and quantitative) of events associated with the infection process. This approach and others (e.g. induced resistance) may be useful in identifying phases which are unique in the interaction between fungal isolates and wheat genotypes (e.g. symptom development), and may aid in identifying wheat genotypes with a similar response (e.g. loci) to such mutants and generate molecular and genetical research on genes associated with resistance. This approach may later call for the adoption of proper measures (e.g. QTL analyses, genomics) to elucidate loci associated with host resistance. Such

measures may be incorporated into breeding for resistance schemes provided they are easily manipulated and economically feasible. It is possible that these and other measures will contribute to a fuller understanding of lasting disease resistance and tolerance to septoria tritici blotch (Jlibene and El Bouami, 1995; Zuckerman et al., 1997) and stagonospora nodorum blotch (Broennimann, 1982; Jeger et al., 1983; Jones, 1985) in the wheat host. The economic importance of both pathogens is likely to generate more interest and expand research activities with the aim of better understanding the wheat–*S. tritici* and *S. nodorum* pathosystems; this will, in turn, contribute to increased protection of the wheat crop and security of grain production.

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