

REVIEW

## Early stages of septoria tritici blotch epidemics of winter wheat: build-up, overseasoning, and release of primary inoculum

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Septoria tritici blotch (STB), caused by *Mycosphaerella graminicola*, is the most prevalent disease of wheat worldwide. Primary inoculum and the early stages of STB epidemics are still not fully understood and deserve attention for improving management strategies. The inoculum build-up and overseasoning involves various fungal structures (ascospores, pycnidiospores, mycelium) and plant material (wheat seeds, stubble and debris; wheat volunteers; other grasses). Their respective importance is assessed in this review. Among the mechanisms involved in the early stages of epidemics and in the year-to-year disease transmission, infection by ascospores wind-dispersed from either distant or local infected wheat debris is the most significant. Nevertheless, infection by pycnidiospores splash-dispersed either from neighbouring wheat debris or from senescent basal leaves has also been inferred from indirect evidence. *Mycosphaerella graminicola* has rarely been isolated from seeds so that infected seed, although suspected as a source of primary inoculum for a long time, is considered as an epidemiologically anecdotal source. *Mycosphaerella graminicola* can infect a few grasses other than wheat but the function of these grasses as alternative hosts in natural conditions remains unclear. Additionally, wheat volunteers are suspected to be sources of STB inoculum for new crops. This body of evidence is summarized in a spatio-temporal representation of a STB epidemic aimed at highlighting the nature, sources and release of inoculum in the early stages of the epidemic.

**Keywords:** alternative host, ascospore, *Mycosphaerella graminicola*, plant debris, pycnidiospore, *Septoria tritici*, volunteer plants

### Introduction

The epidemic phase of plant diseases is generally well studied and correctly represented in simulation and forecast models (Madden *et al.*, 2007). In contrast, inoculum overseasoning and the development of initial infections remain poorly described. As pointed out by Lucas (2006), “inoculum dispersal has received little attention, especially that occurring between the harvesting of one crop and the sowing of the subsequent crop”. For instance, local overwintering of *Puccinia triticina* (Zadoks & Bouwman, 1985), a very common wheat pathogen in Europe, is still poorly understood. When several survival strategies (and then several initial inoculum sources) coexist for the same species, their relative importance is

often controversial and may change with local conditions. This can be the case when the pathogen is able to survive between epidemic seasons in the form of both asexual and sexual survival structures, as in *Erysiphe necator* (Coriot-Costet, 2007). There are, however, very few comprehensive descriptions of cryptic epidemic stages in plant pathogens, except, to some extent, for seedborne diseases (Rennie & Cockerell, 2006). The best candidate for such a review is probably the wheat pathogen *Mycosphaerella graminicola*.

Septoria tritici blotch (STB), one of the most damaging diseases of wheat (*Triticum aestivum*) is caused by the ascomycete fungus *M. graminicola* (anamorph, *Septoria tritici*). STB is a well documented plant disease and a fairly large body of published papers addresses the cryptic epidemic stages and the nature of initial inoculum. Nevertheless, when considered separately, these studies remain fragmentary. Perhaps because a global picture of *M. graminicola* survival and dispersal during cryptic epidemic stages is still lacking, even the most recent models dealing with this

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Published online 11 October 2010

disease (Eriksen *et al.*, 2001; Audsley *et al.*, 2005; Robert *et al.*, 2008) do not include an explicit description of initial inoculum and epidemic onset.

In plant disease epidemiology, the nature and source(s) of the inoculum responsible for the first infections are embedded in the concept of primary inoculum, defined as “propagules or vegetative structures of a pathogen, usually from an overwintering source, that cause initial rather than secondary outbreaks of disease” (Shurtleff & Averre, 1997) or “the overwintering or oversummering pathogen, or its spores that cause primary infection” (Agrios, 2005). In conventional winter wheat cropping systems the crop is harvested in early summer and the next crop emerges as seedlings in mid autumn. The epidemic stage of STB, as it is usually observed, occurs between March and July. To develop epidemics, the pathogen has to produce primary inoculum at seedling emergence and thus has to survive the preceding intercrop period. Moreover, the pathogen has to remain present during winter either on the plants or in the form of survival structures. Inoculum build-up and overseasoning potentially involve various fungal structures (ascospores, pycnidiospores, mycelium) and various plant material (wheat seeds, wheat stubble and debris, wheat volunteers, grass species).

This paper puts together the available information on STB cryptic and initial stages, and evaluates the reliability of this information. First, the literature dealing with ascospores is reviewed, the quantitatively most significant and most studied form of primary inoculum. Secondly, the significance of the other, less-studied, sources of primary inoculum (wheat seeds, grass species; wheat volunteers and pycnidiospore-bearing wheat debris) is addressed. Finally, the available information is summarized in a schematic, spatio-temporal representation of a STB epidemic, highlighting the nature and origin of initial inoculum, and the importance of the primary inoculum sources in STB epidemiology is discussed.

### Ascospores on wheat debris are quantitatively the most significant form of primary inoculum

#### Ascospore occurrence and biology

Ascospores of *M. graminicola*, discovered in New Zealand in 1972 (Sanderson, 1972), have been subsequently considered as the main source of primary inoculum in STB epidemics. The sexual stage has been reported in Australia (Brown, 1975), USA (Garcia & Marshall, 1992), Canada (Horne *et al.*, 2002), Chile (Madariaga, 1986), Argentina (Cordo *et al.*, 1999), Brazil (Mehta, 1989), UK (Scott *et al.*, 1988), the Netherlands (Kema *et al.*, 1996), France (Halama, 1996), Germany (Verreet *et al.*, 1990), Denmark (Eriksen & Munk, 2003), Poland (Glazek & Sikora, 1998) and Slovakia (Pastircak, 2005). It is probable that the sexual stage would be found in other wheat growing areas as well if effectively searched for.

Ascospores of *M. graminicola* are held in asci produced in fruiting bodies of the sexual stage, pseudothecia, also called ascocarps or perithecia. Ascospores result from an encounter between strains with opposite mating types (MAT1-1 and MAT1-2) that are required for sexual reproduction (Kema *et al.*, 1996). The mating system is believed to be bipolar and heterothallic. In STB, most lesions are apparently initiated by a single genotype (McDonald & Martinez, 1990), making it necessary for lesions to coalesce for the two mating types to meet (Eriksen & Munk, 2003). Thus, sexual reproduction of *M. graminicola* is likely to be conditioned by infection density, and the formation of pseudothecia is suspected to be more frequent when epidemics are intense (Cowger *et al.*, 2002). Experimental production of ascospores *in planta* requires a complex combination of laboratory and natural environment conditions (Kema *et al.*, 1996; Dumont *et al.*, 2006). Each ascus contains eight two-celled ascospores (Halama, 1996). Eriksen & Munk (2003) established that a pseudothecium contains 19–45 asci (average 26). Assuming that all asci reach maturity and hold eight ascospores each, this gives a potential number of 200 ascospores per pseudothecium. When shaded or stored in darkness, ascospores remained viable for 1–2 weeks after discharge, but they survived only 2 days when exposed to sunlight (Brown *et al.*, 1978). Under favourable conditions, ascospores can therefore survive long-distance, aerial dispersal.

#### Dynamics of pseudothecia production

Pseudothecia can be observed regularly during the season but always appear a long time after the appearance of pycnidia on an infected leaf layer, in artificial as well as natural conditions (Hunter *et al.*, 1999; Eriksen & Munk, 2003). The pseudothecia/pycnidia ratio then increases over the lifetime of each leaf layer. This could logically be seen as a survival strategy in response to the exhaustion of host resources.

A delay of 29–53 days was measured between the end of the latent period (defined as the time from infection to appearance of the first pycnidia) and the appearance of pseudothecia on a particular leaf layer (Eriksen & Munk, 2003). The time from infection to appearance of pseudothecia was estimated to be 46–76 days by adding this delay to the latent period in the field (17–23 days). This estimate is in good agreement with other field observations in the United Kingdom (Hunter *et al.*, 1999), in which the interval between onset of pycnidia appearance and pseudothecia appearance was maximal (95 days) after inoculation in early January and minimal (62 days) after inoculation in early February. In artificial infection experiments, the period from inoculation to appearance of pseudothecia was estimated as 35 days by Kema *et al.* (1996) and 84–132 days by Hunter *et al.* (1999). These last figures are surprisingly high compared to field estimates, maybe because of environmental factors (Eriksen & Munk, 2003). Pseudothecia produced on green leaves during the epidemics remain active (see below) on

senescent leaves and plant debris, and constitute ascospore sources for later infections.

#### Release of ascospores produced on wheat debris

Sanderson & Hampton (1978) first suggested that *M. graminicola* ascospores contribute to the primary inoculum. Later, Scott *et al.* (1988) observed *M. graminicola* pseudothecia, asci and ascospores on field-sampled wheat bundles exposed to natural weather. The role of ascospores as the main source of primary inoculum was demonstrated using wheat seedlings as a biological trap (Shaw & Royle, 1989). When protected from airborne inoculum (enclosed in tents), seedlings placed in a field previously sown with wheat (thus exposed to local inoculum) exhibited a much lower STB severity than seedlings left unprotected. The same result was observed with seedlings kept away from local inoculum.

Ascospores of *M. graminicola* have been trapped nearly all the year round using a Burkard volumetric spore trap placed adjacent to or in wheat fields infected with STB (Brown *et al.*, 1978; Kema *et al.*, 1996; Hunter *et al.*, 1999; Bathgate & Loughman, 2001). However, ascospore release exhibits a marked seasonal pattern. In the Southern Hemisphere, ascospore counts peak in winter (May–July) with concentrations exceeding 1000 ascospores per cubic metre of air frequently recorded in Australia (Brown *et al.*, 1978) and in New Zealand (Sanderson & Hampton, 1978). In the Northern Hemisphere, ascospore counts usually peak first in late autumn (early October–December), while a second peak occurs at the end of the growing season (June or July) (Hunter *et al.*, 1999; Eriksen & Munk, 2003). The first peak was interpreted as the result of ascospore discharge from pseudothecia present on debris of the previous wheat crop while the second peak was interpreted as the result of ascospore discharge from pseudothecia produced on infected leaves of the current wheat crop (Hunter *et al.*, 1999).

#### The significance of ascospores as primary inoculum is confirmed by population genetic studies

The genetic structure of *M. graminicola* populations provides indirect evidence that sexual reproduction occurs at high frequency. Several “signatures of sex” (McDonald, 2008) have been found: a high level of genetic variability and a high degree of recombination within and among populations of *M. graminicola* on a very small spatial scale (McDonald & Martinez, 1990; Linde *et al.*, 2002), a global population at migration-drift equilibrium resulting from gene flow on a world-wide scale and a very high effective population size (Zhan *et al.*, 2003). More specifically, Zhan *et al.* (2001) showed in Oregon (USA) that a large number of immigrant ascospores (at least 70 per square metre) initiated STB epidemics in wheat fields. In another experiment, the same authors found that epidemics in control (non-inoculated) plots originated from ascospores. Moreover, they established that, within an

inoculated plot, a significant fraction (up to 70%) of the novel isolates sampled during the epidemic originated from sexual recombination among the inoculated isolates (Zhan *et al.*, 1998).

Direct (spore trapping and pseudothecia observation) as well as indirect evidence provided by population structure analysis show that ascospores, resulting from sexual reproduction, are the main component of primary inoculum in STB.

#### Wheat seeds and grass species are a suspected, but probably epidemiologically anecdotal, source of primary inoculum

##### Seed-borne mycelial inoculum

In contrast to the seed-transmitted fungus *Phaeosphaeria nodorum*, anamorph *Staganospora nodorum*, (the causal agent of glume blotch disease), *M. graminicola* has rarely been isolated from seeds. Accordingly there is currently a consensus among plant pathologists to consider seed-borne mycelium as a negligible form of STB primary inoculum.

In natural epidemics, however, infection of wheat heads has been observed. While glumes of wheat plants sometimes exhibit STB symptoms (King *et al.*, 1983), attempts to isolate *M. graminicola* from seeds produced either on naturally infected plants (Luthra *et al.*, 1938; Jones & Cooke, 1969) or after artificial head infections (Williams & Jones, 1973a) have failed. Nevertheless, the review by King *et al.* (1983) and the reference sheet by Sutton & Waterston (1966) state that *M. graminicola* is seed-transmitted. However, these statements are only based on circumstantial evidence provided by Noble *et al.* (1958) and Hampton (1980). After inoculation of adult wheat heads with *M. graminicola*, Brokenshire (1975a) was able to detect the fungus by microscopic observation on only 5% of the grains. Brokenshire’s findings were recently corroborated by the first detection of naturally contaminated wheat seeds (Consolo *et al.*, 2009). In this experiment, seeds collected in the field were surface sterilized and incubated in a moist chamber to increase the fungal biomass. Detection of *M. graminicola* was then realized by PCR. The authors proposed two hypotheses for the transmission of *M. graminicola* by seeds: first, the presence of vegetative mycelium inside internal tissues of the seed and, secondly, as latent mycelial tissue in the embryo. However, the accurate localization of the pathogen in the seed cannot be inferred from their results. Detection of the fungus within seed tissue does not necessarily mean that *M. graminicola* is actually seed-transmitted.

Given the available information, seed transmission of *M. graminicola* in the field seems unlikely.

##### Grass species as alternative hosts

A comprehensive survey of the literature returns 26 weed and cultivated grass species reported as hosts of *M. graminicola* (Table 1). However, only six studies

Table 1 Grass species reported as infected by *Mycosphaerella graminicola*

Host <sup>a</sup>	Common name	Pathogen <sup>b</sup>	Type of investigation <sup>c</sup>	Reliability <sup>d</sup>	Reference
<i>Agrostis capillaris</i> ( <i>Agrostis tenuis</i> )	Common Bent	<i>Septoria tritici</i>	i	++	Williams & Jones (1973b)
		<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Alopecurus pratensis</i>	Meadow Foxtail	<i>S. tritici</i>	i	–	Derevyankin (1969) (in Williams & Jones, 1973b)
<i>Anisantha sterilis</i> ( <i>Bromus sterilis</i> )	Barren Brome	<i>S. tritici</i>	i	+	Williams & Jones (1973b)
		<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Arrhenatherum elatius</i>	False Oat-grass	<i>S. tritici</i>	i	++	Brokenshire (1975b)
<i>Avena sativa</i>	Oat	<i>S. graminum</i>	o	–	Miège (1922)
		<i>S. graminum</i>	o	–	Garbowski (1926)
		<i>S. graminum</i>	o	–	Gram & Thomsen (1927)
		<i>S. graminum</i>	o	–	Cunningham (1928)
		<i>S. graminum</i>	o	–	Marchionatto (1929)
<i>Brachypodium sylvaticum</i>	False Brome	<i>S. tritici</i>	o	+	Frandsen (1946)
<i>Briza maxima</i>	Greater	<i>S. graminum</i>	o	–	Guyot (1945)
	Quaking-grass				
<i>Bromus hordeaceus</i> subsp. <i>hordeaceus</i> ( <i>Bromus mollis</i> )	Soft Brome	<i>S. tritici</i>	i	++	Brokenshire (1975b)
		<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Dactylis glomerata</i>	Cock's Foot	<i>S. tritici</i>	o	–	Zaprometoff (1929)
<i>Elytrigia repens</i> ( <i>Agropyrum repens</i> )	Couch	<i>S. tritici</i>	i	–	Derevyankin (1969)
		<i>S. tritici</i>	o	–	Teterevnikova-Babajan & Bokhjan (1970)
		<i>S. tritici</i>	i	+	Williams & Jones (1973b)
<i>Festuca arundinacea</i>	Tall Fescue	<i>S. graminum</i>	o	–	Zaprometoff (1929)
		<i>S. tritici</i>	i	++	Brokenshire (1975b)
		<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Glyceria fluitans</i>	Floating Sweet-grass	<i>S. tritici</i>	o	–	Grove (1935)
<i>Holcus lanatus</i>	Yorkshire Fog	<i>S. tritici</i>	i	++	Williams & Jones (1973b)
<i>Hordeum murinum</i>	Wall Barley	<i>S. tritici</i>	i	++	Brokenshire (1975b)
<i>Lolium multiflorum</i>	Italian Ryegrass	<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Lolium perenne</i>	Ryegrass	<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Lolium rigidum</i>	Wimmera	<i>S. tritici</i>	i	++	Haghdel & Banihashemi (2005)
	Ryegrass				
<i>Pennisetum glaucum</i>	Pearl Millet	<i>S. graminum</i>	o	–	Gram & Thomsen (1927)
<i>Phleum pratense</i>	Timothy	<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
<i>Poa annua</i>	Annual	<i>S. tritici</i>	i	+	Sprague (1944)
		<i>S. tritici</i>	i	++	Brokenshire (1975b)
	Meadow-grass	<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
		<i>S. tritici</i>	i	+	Prestes & Hendrix (1978)
		<i>S. tritici</i>	i	++	Weber (1922)
<i>Poa pratensis</i>	Swamp	<i>S. tritici</i>	i	++	Weber (1922)
		<i>S. tritici</i>	o	–	Atanasoff <i>et al.</i> (1932)
	Meadow-grass	<i>S. tritici</i>	i	+	Sprague (1944)
		<i>S. tritici</i>	i	++	Williams & Jones (1973b)
		<i>S. tritici</i>	i	++	Brokenshire (1975b)
		<i>S. tritici</i>	i	++	Ao & Griffiths (1976)
		<i>S. tritici</i>	i	+	Prestes & Hendrix (1978)
<i>Poa scabrella</i> ( <i>Poa secunda</i> )	Pine Bluegrass	<i>S. tritici</i>	i	+	Sprague (1944)
<i>Poa trivialis</i>	Rough	<i>S. tritici</i>	i	+	Williams & Jones (1973b)
	Meadow-grass				
<i>Polypogon viridis</i> ( <i>Agrostis verticillata</i> )	Water Bent	<i>S. graminum</i>	o	–	Zaprometoff (1929)

Table 1 Continued

Host <sup>a</sup>	Common name	Pathogen <sup>b</sup>	Type of investigation <sup>c</sup>	Reliability <sup>d</sup>	Reference
<i>Secale cereale</i>	Rye	<i>S. graminum</i>	o	–	Gram & Thomsen (1927)
		<i>S. graminum</i>	o	–	Mourashkinsky (1940)
		<i>S. tritici</i>	o	–	Atanasoff <i>et al.</i> (1932)
		<i>S. tritici</i>	o	+	Sprague (1950)
		<i>S. tritici</i>	i	++	Weber (1922)
		<i>S. tritici</i>	i	–	Derevyankin (1969)
		<i>S. tritici</i>	i	++	Haghdel & Banihashemi (2005)
<i>Vulpia bromoides</i>	Squirreltail Fescue	<i>S. tritici</i>	i	++	Brokenshire (1975b)

<sup>a</sup>Binomial name used in the original source shown in brackets.

<sup>b</sup>First described by Desmazières (1842), *Septoria tritici* was subsequently considered by the same author as a 'variety' of *S. graminum* (Desmazières, 1847). Until Sprague (1938) showed *S. graminum* to be distinct from *S. tritici*, the binomial *S. graminum* was assigned to a pathogen of wheat, presumably as a synonym of *S. tritici*.

<sup>c</sup>Type of investigation: o = records of disease occurrence without experimental confirmation of the pathogen status; i = inoculation test performed.

<sup>d</sup>Reliability: – = low reliability due to either records of disease occurrence without experimental confirmation or experimental method not described accurately enough; + = medium reliability due to accurate description of the experimental method without fulfillment of Koch's postulates, but with an effort of taxonomic identification; ++ = high reliability due to accurate description of the experimental method with an acceptable fulfillment of Koch's postulates.

(Weber, 1922; Sprague, 1944; Williams & Jones, 1973b; Brokenshire, 1975b; Ao & Griffiths, 1976; Haghdel & Banihashemi, 2005), were actually aimed at investigating the role of grasses in the epidemiology of STB, and can be considered as a reliable source of information. Among all the species found susceptible to *M. graminicola* in these studies, six (*Agrostis capillaris*, *Bromus hordeaceus* subsp. *hordeaceus*, *Anisantha sterilis*, *Festuca arundinacea*, *Poa annua* and *P. pratensis*) were reported in at least two independent studies and can therefore be highlighted as most probable alternative hosts of STB. The reliability of all the other reports was deemed too low to ascertain the epidemiological role of other grass species. Some of these reports included only circumstantial evidence (isolated records). Moreover, most older reports are spurious because the exact taxonomical status of the fungus was clarified only in the middle of the 20th century (Sprague, 1938, 1950); previous reports of '*Septoria graminum*' on cultivated grass species (oats, millet, rye; Table 1) were subsequently assigned to different *formae specialis* of *S. tritici* (Sprague, 1950). The only reference to a non-graminaceous alternative host for *M. graminicola*, provided by Prestes & Hendrix (1978), who reported 'pathogenicity of *S. tritici*' on *Stellaria media* (the common chickweed, Family Caryophyllaceae), was not confirmed by any other study.

*Mycosphaerella graminicola* is considered as a specialized pathogen of wheat but its capacity to infect other grasses has been demonstrated. Suggesting that 'grass weeds at the perimeter of wheat fields may act as disease foci, which possibly explains severe early winter wheat infections' and that 'the perennial growth habit of most grass species could enable the generation of

inoculum of *M. graminicola* from year to year in the grass canopy for wheat infection', Brokenshire (1975b) called for a field evaluation of the role of grass species in the epidemiology of STB. Such an evaluation does not seem to have been performed yet. However, it is unlikely that wild grasses play a crucial role in STB epidemiology. Grasses able to harbour STB cannot be involved in a hypothetical 'green bridge' (Zadoks & Bouwman, 1985) between harvest and volunteer emergence, since they wither, like wheat, at the end of summer. Moreover, there are strong sources of primary inoculum in the form of wheat debris at the time when the grasses would act as an additional source. Nevertheless, the grass hosts could play a role in the long term conservation of inoculum or allow particular genotypes to be maintained. In addition, if sexual reproduction occurs on these grasses, they could constitute potential sources for long-distance dispersal of the disease through ascospores.

While the anamorph *S. tritici* was found on *Triticum turgidum* var. *dicoccoides* in natural stands in Israel (Eyal, 1999) and was able to infect several species of *Triticum-Aegilops* complex under controlled conditions (McKendry & Henke, 1994; Jlibene *et al.*, 1995; Seifbarghi *et al.*, 2009), the role of indigenous wild emmer wheat as a source of primary inoculum for commercial durum (*T. durum*) or soft wheat (*T. aestivum*) is not known. The reconstruction of the evolutionary history of *M. graminicola*, by coalescence analysis, revealed a relatively recent origin of the pathogen, coinciding with the known domestication of wheat in the Fertile Crescent (Stukenbrock *et al.*, 2007). This reinforces the idea that wheat is the main host for *M. graminicola*.



## Infected volunteer plants and pycnidiospores on wheat debris are underinvestigated, potential forms of primary inoculum

### Volunteer wheat plants

The contribution of wheat volunteers to the survival and spread of STB has not been explicitly considered yet, in spite of reports of the presence of *M. graminicola* on volunteer leaves. Weber (1922) reported in Wisconsin (USA) that wheat volunteers infected with *M. graminicola* can survive the intercrop period and eventually act as a source of inoculum for the next wheat crop, a report which was confirmed by Wenham (1959) in New Zealand. Wheat volunteers bearing pseudothecia of *M. graminicola* were collected in a field previously sown with wheat in the Netherlands (Kema *et al.*, 1996). Surveying volunteers in British set-aside fields following a previous unsprayed wheat crop, Hunter *et al.* (1999) found pseudothecia from November to January. Ascospore discharge was demonstrated in both studies. Pycnidia were detected on volunteers in a set-aside field in South West France in October 2007 and in a freshly ploughed wheat field near Paris in October 2008 and 2009 (unpublished data), and by Hunter *et al.* (1999) in the United Kingdom. This limited set of observations suggests that volunteers can be considered as inoculum sources for both pycnidiospores and ascospores and could contribute to the pathogen overseasoning.

### Pycnidiospores produced on wheat debris

Pycnidiospores are usually considered as the main source of inoculum of *M. graminicola* during the epidemic period. However, pycnidia can also be found on decaying leaves and on stubble. Produced in a mucilaginous matrix (cirrhous), pycnidiospores can remain viable during extended periods of dry weather and could be a potential form of primary inoculum.

#### Primary contamination by pycnidiospores

Pycnidia containing viable pycnidiospores of *M. graminicola* have been observed on wheat stubble and debris in several instances (Weber, 1922; Luthra *et al.*, 1938; Hilu & Bever, 1957; Wenham, 1959; Brokenshire, 1975c; Djerbi, 1977). The infectious potential of wheat stubble was demonstrated by successfully contaminating wheat seedlings in the field with pieces of wheat straw artificially inoculated with *M. graminicola* (Holmes & Colhoun, 1975).

Eyal *et al.* (1987) and Djerbi (1977) reported that *M. graminicola*, in contrast to *P. nodorum*, is not able to produce new pycnidia on dead tissue. Within pycnidia, a pool of pycnidiospores are produced and then released sequentially, according to rain events. Pycnidiospore production peaks after the first wetting, fewer and fewer pycnidiospores being released after each subsequent wetting; pycnidia are not capable of regenerating new

pycnidiospores after a release event (Eyal, 1971). In Tunisia, Djerbi (1977) found pycnidia on wheat stubble collected in the field during the five summer months. The pycnidia collected in September, after autumn rains, produced bacillus-shaped microspores instead of the expected pycnidiospore-containing cirrhi when placed in moist conditions. Microspores, first described by Sprague (1950), result from pycnidiospore germination inside the pycnidium (Djerbi *et al.*, 1974). Similar microspores are commonly observed *in vitro* and used for the vegetative multiplication of *M. graminicola*. The role of these microspores produced inside field-collected pycnidia, especially their germinative ability, remains to be investigated.

Pycnidiospore production on wheat debris, as obtained in laboratory conditions by Brokenshire (1975c), requires moist conditions (Weber, 1922). On debris exposed to a high relative humidity (RH), sporulation was maximal after 6 days and then declined; at low RH the spore production was lower but did not decline with time (Brokenshire, 1975c).

Pycnidiospores are dispersed by rain-splash over short distances (Holmes & Colhoun, 1975; Shaw, 1987). They could act as primary inoculum in areas where wheat is stubble-sown or for sowing dates later than the main period of ascospore release (Brown *et al.*, 1978). Leaf debris bearing pycnidia could be wind-dispersed and constitute an additional source of exogenous inoculum. This means of dispersal, however, was dismissed by Sanderson & Hampton (1978) who argued that fertile pycnidia were rarely found on debris small enough to be transportable by wind.

#### Pycnidiospore viability and survival

On wheat debris, pycnidiospore viability was reported to decrease with time at a rate depending on environmental conditions and cultural practices.

In Tunisia, Djerbi (1977) showed that during the post-harvest, dry period in summer, the germination rate of pycnidiospores obtained on wheat debris decreased from 100% (June) to 0% (August). This decrease in viability during summertime could be related to the RH and temperature conditions. In New Zealand, Wenham (1959) reported, without providing data, that pycnidiospores had remained viable up to 5 months after harvest (February–June). Gough & Lee (1985) showed that pycnidiospores, when maintained in cirrhi, remain viable for a long period of time at low RH (95% germination after 50 days at 35–55% RH) but not at high RH (0% germination after 50 days at 65–85% RH). Temperature seems to have a strong effect on pycnidiospore viability. When collected from pycnidia that were produced on naturally infested leaves, pycnidiospores lost their *in vitro* germination capacity after 2 months at 15–30°C, while they remained viable for more than 9 months at 5–15°C (Hilu & Bever, 1957).

Pycnidiospore viability is dramatically decreased in pycnidia on buried debris. Pycnidia disappeared and

pycnidiospores did not survive on infected debris buried at 7.6 cm depth in soil for 1 month (Hilu & Bever, 1957). Brokenshire (1975c) showed that pycnidiospores are better preserved on surface debris (65% survived after 50 days) than on buried debris (< 10% survived after 50 days), irrespective of the burial depth (2.5, 5 or 7.5 cm); this fact was confirmed by Haghdel & Banihashemi (2005) under arid field conditions. Baker (1969) suggested that *M. graminicola* is a poor competitor in soil, where the pycnidial content can be decomposed by soil microorganisms. Indeed, pycnidiospores of *M. graminicola* maintained their viability up to 24 months in sterilized soil (Shearer *et al.*, 1974).

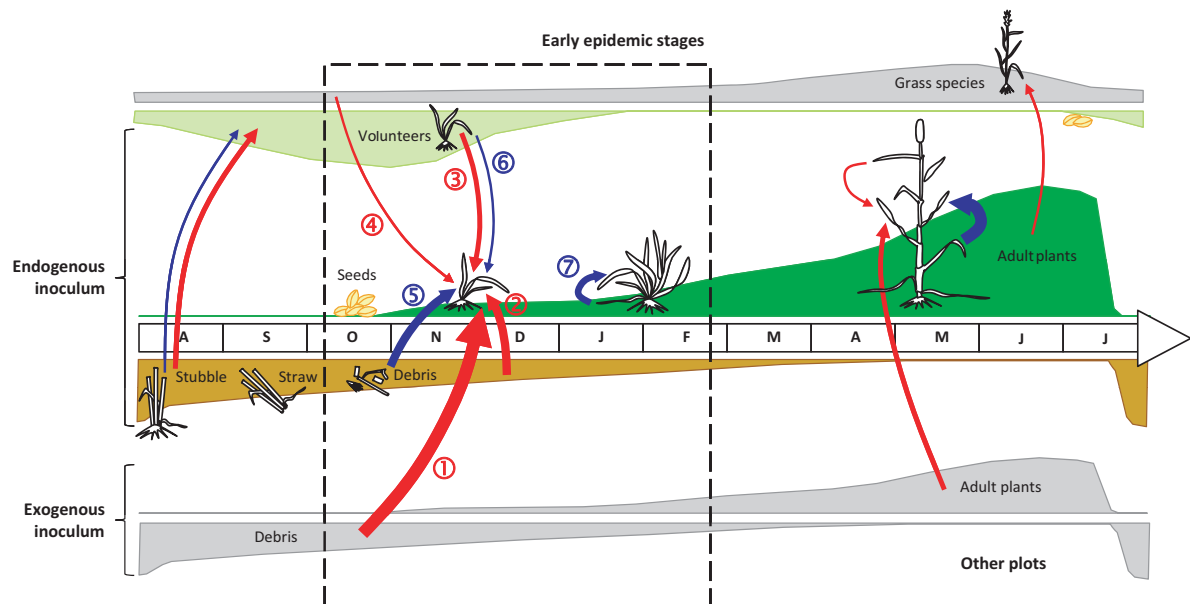
### Primary inoculum in STB epidemiology

A review of the literature, summarized in Fig. 1, suggests that a STB epidemic is more complex than usually admitted. Eyal *et al.* (1987) defined it as two-staged, the first stage being early seedling infection by wind-blown ascospores, and the second stage being later infection by pycnidiospores on upper plant parts. In contrast, Fig. 1 highlights the importance of the different potential sources of STB primary inoculum bearing ascospores or pycnidiospores, of their origin, and of their potential contribution to the early epidemic stages.

In Western Europe, the management of STB during the epidemic stage relies on the use of resistant varieties and

fungicide sprays. Nevertheless, it has proved difficult to produce varieties which combine effective disease resistance with high yield (Brown *et al.*, 2008) and the field performance of fungicides is currently declining due to the development of fungal resistance (Leroux *et al.*, 2006). This makes necessary the development of alternative methods, such as promoting disease escape (Lovell *et al.*, 1997) and reducing inoculum availability before the epidemic phase, through cropping practices like stubble management and crop rotation. Accordingly, modulation of sowing date, sowing density, and host resistance should be investigated at the early stages of epidemics.

Reduction of primary inoculum decreases the early severity of STB and, in some experimental situations, this effect is extended beyond the early stages of the epidemics. Parker & Lovell (2001) showed that primary infection of *M. graminicola* was delayed when wheat seeds were coated with a triazole fungicide; on basal leaves, production of pycnidiospores, the potential sources of secondary infections, was still reduced 5 months after sowing, while the fungicide no longer had biological activity. Similarly, Sutton (1985) reported that triazole seed treatments controlled STB for more than 200 days. Parker *et al.* (1999) suggested that suppression of the winter inoculum pool (mainly ascospores) by cold temperature can reduce the severity of the summer epidemic. Based on a large data set collected in the UK, the best predictive model of STB severity was based on the frequency of occurrence of air temperature below  $-2^{\circ}\text{C}$  during the



**Figure 1** A schematic representation of the yearly dynamic of a septoria tritici blotch (STB) epidemic, with emphasis on the sources and the nature of primary inoculum. Red arrows: ascospores; blue arrows: pycnidiospores. The width of the arrows is proportional to the expected significance of the represented mechanism. The early stages of the epidemic discussed in the review are enclosed in the dotted box; 1: ascospores wind-dispersed from distant infected wheat debris; 2: ascospores wind-dispersed from neighbouring wheat debris; 3: ascospores wind-dispersed from wheat volunteers; 4: ascospores wind-dispersed from grass species; 5: pycnidiospores splash-dispersed from neighbouring wheat debris; 6: pycnidiospores splash-dispersed from wheat volunteers; 7: pycnidiospores splash-dispersed from senescent basal leaves.

period from November to early December, which typically coincides with early crop emergence and initial ascospore infection (Parker *et al.*, 1999; Gladders *et al.*, 2001).

Infection by ascospores wind-dispersed from either local (endogenous) or distant (exogenous) infected wheat debris (1 and 2 on Fig. 1), demonstrated in field studies, is considered to be quantitatively the most significant mechanism involved in early epidemic stages. Sanderson & Hampton (1978) reported that ascospores were still being liberated from wheat stubble 8 months after harvest. Brown *et al.* (1978) could not retrieve fertile pseudothecia in stubble that had remained in the field for two seasons. Nevertheless, recent field experiments (Maumené *et al.*, 2009) suggest that ploughing modifies the earliness and severity of STB probably by bringing back the wheat debris to the surface, when the previous crop was a non-host crop preceded by wheat. However, this mechanism would probably have a minor effect in most field situations, since little stubble remains available for more than 1 year and new neighbouring wheat crops provide a pool of fresh inoculum prone to airborne dispersal every year.

Two other initial inoculum origins have been inferred from indirect evidence: the infection by pycnidiospores splash-dispersed either from endogenous wheat debris or from senescent basal leaves (detached from the tiller or not) during the tillering stage (5 and 7, respectively, in Fig. 1). Several experiments performed after the discovery of the *M. graminicola* teleomorph failed to consider that pseudothecia-bearing wheat debris also bear pycnidia, which could release a significant amount of pycnidiospores acting as a local primary inoculum (Brokenshire, 1975c; Djerbi, 1977). Mechanism 7 was probably underestimated since early STB severity assessments were performed only on green leaves and did not explicitly take into account symptoms on basal senescent leaves.

In conventional wheat cropping systems, wheat stubble is ground after harvest and debris is subsequently buried into the soil by ploughing. In alternative cropping systems, debris is either not incorporated into the soil (no grounding, standing stubble), or partially left on the soil surface (conservation tillage). Dinoor (1977) established that burning of infected straw after harvest significantly reduced STB intensity under semi-arid conditions. Under favourable weather conditions, a 2-year rotation between wheat crops provided adequate control of the septoria disease complex, including STB (Pedersen & Hughes, 1992). Similarly, STB incidence was decreased by crop rotations either with wheat cropping intervals of 3–5 years (Eyal *et al.*, 1987) or increased crop diversity (Bailey *et al.*, 2001). Unexpectedly, STB was less severe (Sutton & Vyn, 1990; Gilbert & Woods, 2001) or did not substantially increase (Bailey *et al.*, 2001) in conservation systems compared with conventional tillage systems. Schuh (1990) concluded that ‘tillage systems or the straw residue left in fields do not lead to different disease incidences at the beginning of or during the growing season’; in his study, disease was first assessed on leaves 3 and 4

(middle of April, while the crop had been sown in the second and third week of October), when, most probably, several infection cycles had already occurred. Such a delayed first disease assessment date and the lack of respective quantification of mechanisms 1 and 2 (Fig. 1) can explain the aforementioned unexpected results; to test the influence of stubble and debris management on early STB development, disease should be assessed as soon as wheat emerges.

While volunteers (3 and 6) and grass species (4) have been suspected to be other sources of STB inoculum, a systematic destruction of volunteers and alternative hosts would not necessarily delay the onset of STB epidemics, taking into account the main mechanisms of primary infection (1, 2, 5 and 7). The importance of these mechanisms and their occurrence in time (during the early growing season) and space (within fields and between fields) remains to be investigated.

Qualitative studies of STB epidemics should consider alternative survival forms of the fungus beyond ascospores. Population surveys are usually performed during epidemic peaks, when large numbers of isolates can be sampled (e.g. Goyeau *et al.*, 2006). In agro-ecosystems, selection by hosts is probably the most important evolutionary force (McDonald & Linde, 2002) constraining the pathogen; selective forces are studied during epidemics much more frequently than during inter-epidemic stages of the parasite life cycle. Selective pressures imposed on the pathogen during the non-epidemic phases and the epidemic peak can be very different, therefore the most aggressive isolates are not necessarily the fittest during intercrop. Such effects can lead to transmission – survival trade-offs in crop pathogens (e.g. Montarry *et al.*, 2007). Considering the different sources and forms of inoculum, as well as their importance in time over the year, would allow a better identification of the selective constraints the pathogen undergoes throughout the year, and explain the survival of low-frequency genotypes or the emergence of new strains.

Regional conditions can modify the respective importance of the different forms of inoculum. Pycnidia survival and pycnidiospore viability on wheat debris depend on environmental conditions and cropping practices, which may explain contradictory reports in the literature. On the one hand, pycnidiospores remained viable for 2–3 months on infected wheat debris during the intercrop period in oceanic and semi-oceanic climatic zones, where they were considered as an additional form of primary inoculum. On the other hand, survival of pycnidia, viability of pycnidiospores, and potential regeneration of microspores (Djerbi, 1977) was reported to be higher in dry climate areas, while ascospores were not systematically detected (Obaedo *et al.*, 1999). Abrinbana *et al.* (2010) recently suggested that pycnidiospores may be a significant source of primary inoculum because clonal haplotypes were identified in different sampling sites in two Iranian provinces, not only on the same leaf or in the same sampling site within a field (Linde *et al.*, 2002), and because of the aggregated structure of the epidemics in



some sites. In intermediate situations (continental and Mediterranean climatic zones) or during seasons with exceptional meteorological conditions, the respective importance of pycnidiospores and ascospores probably fluctuates and the effective role of wheat debris cannot be easily predicted.

Endogenous sources of inoculum for a given plot will also act as exogenous sources for neighbouring plots; at the plot scale, exogenous inoculum (ascospores) can become higher than endogenous inoculum (ascospores and pycnidiospores) after a certain date. Therefore, STB management by cropping practices (crop rotation, tillage, debris management) aimed at limiting primary infection should also be considered on a larger scale (production region).

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