Septoria nodorum blotch of wheat

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DISEASE: Septoria nodorum blotch a.k.a. Septoria glume blotch

PATHOGEN: *Parastagonospora nodorum*; most common synonyms encountered in literature are *Stagonospora nodorum*, *Septoria nodorum*, *Phaeosphaeria nodorum*, and *Leptosphaeria nodorum.*

HOSTS: Bread wheat (*Triticum aestivum*), durum wheat (*Triticum durum*), and Triticale are the major hosts.

Key words: *Parastagonospora nodorum*, *Stagonospora nodorum*, *Septoria nodorum*, *Phaeosphaeria nodorum*, *Leptosphaeria nodorum*, Stagonospora nodorum blotch, wheat foliar disease, glume blotch

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Symptoms of Septoria nodorum blotch on wheat leaves. (Courtesy U. Adhikari)

Symptoms and Signs

Symptoms

Parastagonospora nodorum, the causal agent of Septoria nodorum blotch (SNB), produces symptoms on all aboveground parts of the plant; i.e. leaves, leaf sheaths, stems, glumes, and awns (Figures 1, 2, and 3). On leaves, initial symptoms of SNB appear as small dark-brown to chocolate-colored lesions, usually on the mid- rib of older leaves that are close to the soil surface. These lesions typically have a yellow halo (Figure 1) as a result of diffusible toxins produced by the pathogen. The lesions expand and become oval (lens-shaped) or elliptical with dark-brown centers. A mature SNB lesion has a grayish-white center with a dark-brown periphery. In severe epidemics, lesions can coalesce to cover the whole leaf, resulting in the death of the leaf tissue. On the glumes and awns, symptoms appear as tan to brown-colored lesions (Figure 2). The lesion on a glume typically starts at the tip of the glume and progresses downward. The pathogen can also result in

dark-brown lesions on stems and nodes (hence the species name nodorum) of wheat plant. Infected glumes lead to shriveled kernels that reduce the quality and quantity of the produce (Figure 4).

Signs

As the diagnostic oval-shaped lesions expand and become necrotic, the center of the lesion turns light-brown in color and, at the center, small pin-head sized black dots can be seen, arranged in an irregular pattern. Those dots are flask-shaped asexual fruiting structures of the fungus (also known as pycnidia, singular pycnidium) (Figure 5, 6, and 7). Pycnidia contain diagnostic asexual spores known as conidia (singular conidium) or pycnidiospores (hereafter conidia) in a mucilaginous mass (Figure 8, 9, and 10). White to pinkish masses of conidia (cirrhi, singular cirrhus) exude from pycnidia after placing SNB- infected leaf tissue on moist paper for three to seven days. These asexual fruiting structures are produced on glumes as well as stems of the wheat plant. At the end of the season, another type of fruiting body is formed on plant debris. These structures, known as pseudothecia (singular pseudothecium), are also flask-shaped, but in contrast to pycnidia are sexual structures of the fungus that contain ascospores (sexual spores) in asci (sac-like structures holding ascospores, usually eight per ascus). A hand lens of 20× magnification may be needed to clearly see these structures in the field, while more details can be seen under dissecting and compound microscopes.

Pathogen biology

Asexual reproduction

Parastagonospora nodorum is a necrotrophic fungus (a fungus that feeds on dead plant tissue) that belongs to phylum Ascomycota of kingdom Fungi. Conidia of the fungus are hyaline and slender,

measuring 15–24 µm in length and 2.5–4.0 µm in width and have three (and occasionally one to two) conspicuous septa (Figure 11). A leaf wetness duration of 8 to 12 hours is required for pycnidia to release conidia. The majority of conidia are released during the first wetting of pycnidia. It is estimated that on average there are 3 million conidia available per 10 cm^2 of leaf to cause infection. This high conidial production ensures that at least some conidia can land on upper leaves with the help of rain-splash and cause disease. The pathogen is able to penetrate the leaf cuticle directly. Pycnidia form on the plant within 7 to 14 days after inoculation under optimum conditions of temperature and moisture. Such a short incubation period can result in multiple infection cycles per season, giving rise to a significant amount of secondary inoculum. The conidium germinates to produce a penetration peg, which releases enzymes to help in direct penetration of the wheat leaf cuticle. Colonization and necrosis (brown discoloration) of the host tissue follows direct penetration.

Sexual reproduction

The ascospores produced in sac-like asci in pseudothecia are 4 celled and slightly curved. Ascospores are hyaline to yellow in color, and measure 4 to 6 µm in width and 19 to 32 µm in length (Figure 12). The formation of pseudothecia requires the presence of two opposite mating types (heterothallic fungus). Sexual reproduction is important in introducing genetic variation into the pathogen population. Pseudothecia require a substantially longer period to develop than pycnidia, which is why they are more commonly found on wheat stubble than leaves.

Necrotrophic effectors or host-selective toxins

In addition to being an important pathogen of wheat, *P. nodorum* also serves as a model organism for necrotrophic fungal pathogens; its genome was published in 2007. It secretes various host-selective toxins (more recently termed "necrotrophic effectors") to kill host tissue during colonization. Necrotrophic effectors (NE) of *P. nodorum* interact with corresponding sensitivity genes in wheat in an "inverse gene-for-gene" manner. Recognition of a specific NE by the corresponding dominant sensitivity gene in wheat results in the disease. Oliver et al. (2012) suggested that, in interactions involving NE and sensitivity genes in wheat, the severity of disease is determined by the number and identity of such matches. Thus far, nine such NE-host sensitivity gene interactions have been identified: SnToxA-*Tsn1*, SnTox1-*Snn1*, SnTox2-*Snn2*, SnTox3-*Snn-B1*, SnTox3-*Snn3-D1*, SnTox4-*Snn4*, SnTox5-*Snn5*, SnTox6-*Snn6*, and SnTox7-*Snn7*. These interactions occur under field conditions as well.

Breeding efforts using mapping populations to find quantitative trait loci (QTL) associated with SNB resistance in wheat and facilitate marker-assisted selection. Advanced experimental lines from central and eastern U.S. breeding programs are screened each year for SNB resistance in irrigated, inoculated nurseries (USDA-ARS Eastern Stagonospora Nursery). Planting resistant cultivars is one of the most effective ways to manage SNB, and introgression of resistance into commercial wheat lines using these new discoveries is a critical step toward better disease management.

Disease cycle and epidemiology

Disease cycle

The pathogen overwinters on wheat residue in the form of pseudothecia and pycnidia. Ascospores released from pseudothecia are usually the source of primary inoculum; however, conidia splashed

from wheat debris to the young seedlings can also initiate the disease. The fungus is also known to survive on seed as dormant mycelium and colonized seed can be a source of primary infection. Mature lesions on plant leaves contain pinhead sized pycnidia that are the source of secondary inoculum. The secondary spread of the pathogen within the season occurs when rain-splashed conidia are spread from lower leaves to upper leaves and to glumes. This pathogen also produces multiple host selective toxins that aid in infection by killing the cells before hyphal colonization. Susceptibility in the host is influenced by the interaction between necrotrophic effectors produced by the pathogen and sensitivity genes present in the host, and likely by other interacting gene products as well. Infected wheat residue left in the field and infected grain (if used for seed) serve as the source of inoculum in the following year, and the disease cycle continues (Figure 13).

Epidemiology

Primary inoculum in the field can be infected seed (harboring mycelia of the fungus), rain-splashed conidia or windborne ascospores from infected wheat debris. Release of ascospores from pseudothecia is highly dependent on weather variables [rainfall >1 mm, temperature above 0°C, and high (75–95%) relative humidity]. Studies in New York have shown that initial infection is not solely dependent on immigrant ascospores but can also be caused by seedborne inoculum. Transmission of the pathogen from seed to coleoptile and the first leaf decreases with increase in temperature. It is likely that seed infection plays a relatively bigger role in regions where mean temperature is lower (around 9°C). Severity of Septoria nodorum blotch is known to increase with increasing amounts of wheat residue on the ground.

Disease symptoms appear first on the oldest leaves in early spring. Lesions can expand and coalesce, leading to necrosis of the entire leaf. Small (160–210 µm in diameter) fruiting bodies

(pycnidia) are formed at the center of mature lesions one to two weeks after infection under high relative humidity (Figure 5 and 6). Both conidia and ascospores can germinate and cause infection between 5 and 35°C (optimum 15 to 25°C), and penetration can happen directly through the cuticle or opportunistically through stomata. The optimum temperature for the development of disease symptoms and pycnidia formation is 20°C.

The rate at which an epidemic spreads is dependent upon the latent period, which is defined as the period between inoculation of host tissue and sporulation. The latent period of *P. nodorum* varies greatly -- from 6 to 49 days across various studies -- and is dependent on temperature, moisture, and cultivar. Septoria nodorum blotch development is also favored by rainstorms, which can cause sudden outbreaks and fast vertical spread from lower leaves to upper leaves.

Prediction models for SNB

Several prediction models have been developed to predict epidemics of SNB. Tyldesley and Thompson (1980) developed a model that had 71% accuracy in predicting SNB epidemics based on the frequency of rainfall in England and Wales. Similar qualitative thresholds were provided in Denmark for both Septoria nodorum blotch and Septoria tritici blotch, where eight days with rainfall ≥1 mm in a 30-day period starting at stem elongation correlated with disease severity and yield response. An expert system called EPINFORM was developed in Montana to provide estimates of damage caused by SNB and stripe rust. Their system relied upon the number of infection cycles necessary to cause yield penalty; however, it was assumed that inoculum is present in the field at all times and weather is the only deciding factor in initiating the infection cycle. In general, these modelling efforts have found rainfall to be a significant predictor of end-of-season SNB intensity.

More recently, a risk assessment model was developed to select cultivars at the beginning of the season based on the location of the field and residue management practices adopted by the grower. It has also been confirmed that early onset of disease results in increased yield losses, and disease onset can be predicted based on weather variables and pre-planting factors such as amount of wheat residue and location of the field. More research is needed to validate these models and deploy them for public use.

Disease management

Cultural management

Septoria nodorum blotch can be managed by using a variety of cultural practices that include crop rotation and tillage that ensures complete burial of residue. While crop rotation and tillage have been shown to reduce end-of-season severity of SNB, their effectiveness depends on their widespread adoption, because aerial ascospores from adjacent fields may lead to disease development in fields without wheat residue on the soil surface. Removal of wild grasses and wide row space planting may aid in reducing disease spread since conidia can only move few meters away from the source of infection.

Chemical management

Since one of the sources of inoculum for this pathogen is infected seed, proper seed treatment with a fungicide is recommended to reduce this source of primary inoculum. Infected seed has the potential to start epidemics at multiple foci in a disease-free field. Seed can be tested for the presence of the pathogen by plating them on a selective media SNAW (*S. nodorum* agar for wheat). If the seed contains mycelium of *P. nodorum*, it fluoresces under near ultraviolet light and also

sporulates within 7 days (Figure 14). Foliar fungicide sprays are effective in controlling SNB, and the recommended ones are triazoles (e.g. metaconazole and prothioconazole); site-specific ones such as strobilurins (e.g. pyraclostrobin, azoxystrobin, and picoxystrobin); and combinations of strobilurin and triazoles (e.g. trifloxystrobin plus prothioconazole). The goal of fungicide application should be to protect the flag leaf and F-1 (the leaf below flag leaf) leaf because these leaves provide majority of photosynthates to the developing spike.

Host resistance

Some winter wheat cultivars with partial resistance to SNB are available, and more breeding efforts at several universities are underway to develop resistant varieties for SNB. If available, the use of resistant cultivars in managing SNB is recommended (Figure 15). The disease resistance in wheat against *P. nodorum* is quantitative or partial in nature.

Significance

Septoria nodorum blotch occurs in wheat-growing areas worldwide, but the disease is more prevalent in areas with warm and moist weather, such as the southeastern United States, parts of Europe, southern Brazil, and Australia. The disease affects both the quantity and quality of yield, and the pathogen is capable of affecting wheat at both seedling and adult stages. Historically, losses up to 50% have been reported, in addition to lower grain quality, although in the U.S., lower levels of loss are typical. The yield losses are highest when flag leaf, F-1 (leaf below flag leaf), and F-2 (leaf below F-1) are infected. The disease is known to reduce thousand-kernel-weight, a yield parameter.

The fungus undergoes regular cycles of sexual recombination due to the availability of both mating types, and creates genetic variation in its population, thus enhancing its potential to overcome control measures. The pathosystem is also a model system for necrotrophic plant pathogens. So far, nine necrotrophic effectors and host susceptibility gene interaction have been identified, which have the potential to be used in marker assisted selection for breeding resistant wheat varieties.

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Figures

Figure 1. A typical elliptical lesion of Septoria nodorum blotch. Notice the prominent chlorotic (yellow) halo. (Courtesy Urmila Adhikari).

Figure 2. Brown to tan colored lesions of Septoria nodorum blotch on wheat glumes. (Courtesy Jimmy Clements).

Figure 3. Wheat experimental plots showing heavy glume infection (brown to tan colored discoloration of heads). (Courtesy Lucky Mehra).

Figure 4. Shriveled wheat kernels (right) in comparison to healthy kernels (left). Shriveled kernels were harvested from a wheat plot with head infection of *Parastagonospora nodorum*. (Courtesy Urmila Adhikari)

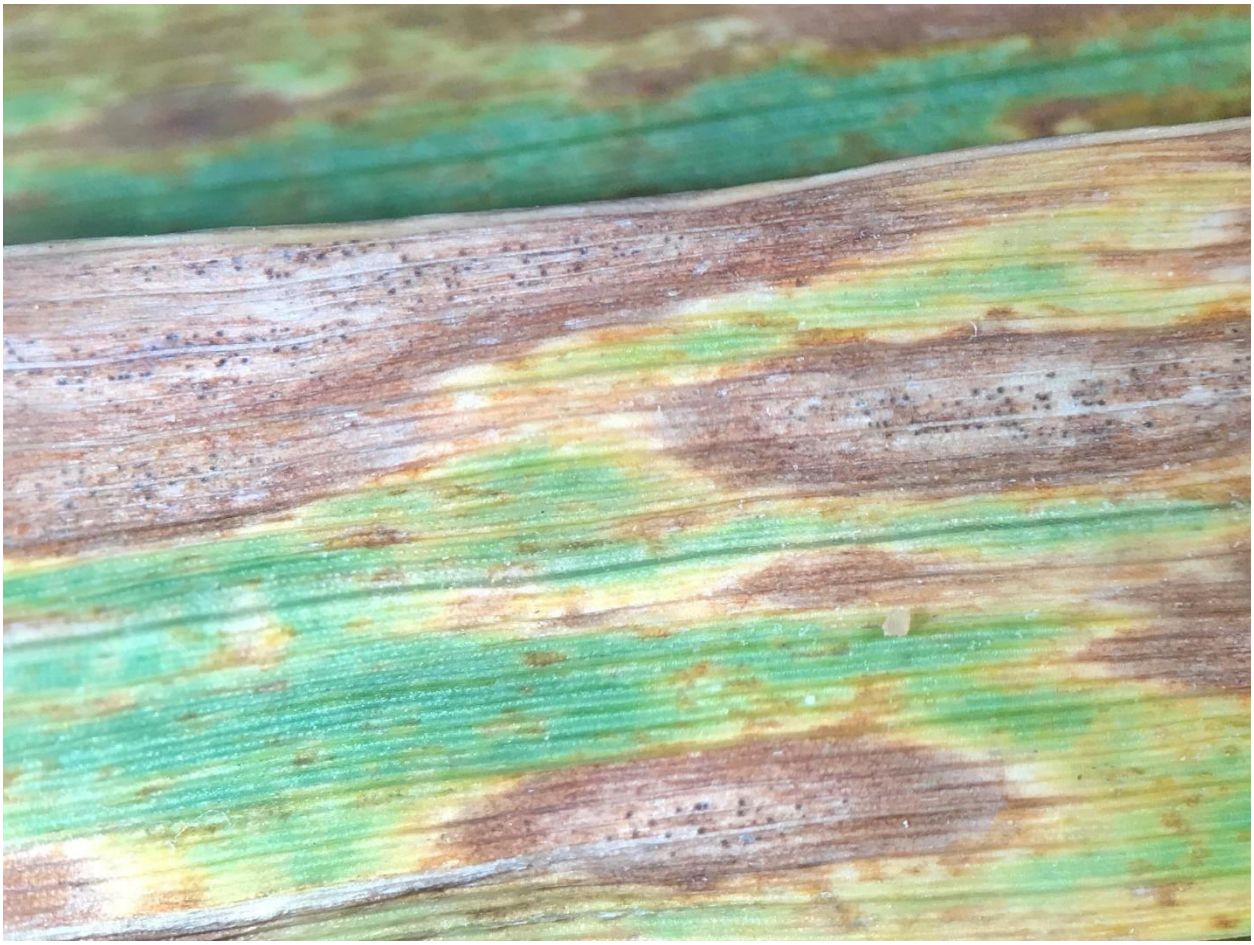

Figure 5. Pycnidia (pin-head sized black dots) of *Parastagonospora nodorum* are visible in the center of each individual lesion. (Courtesy Urmila Adhikari).

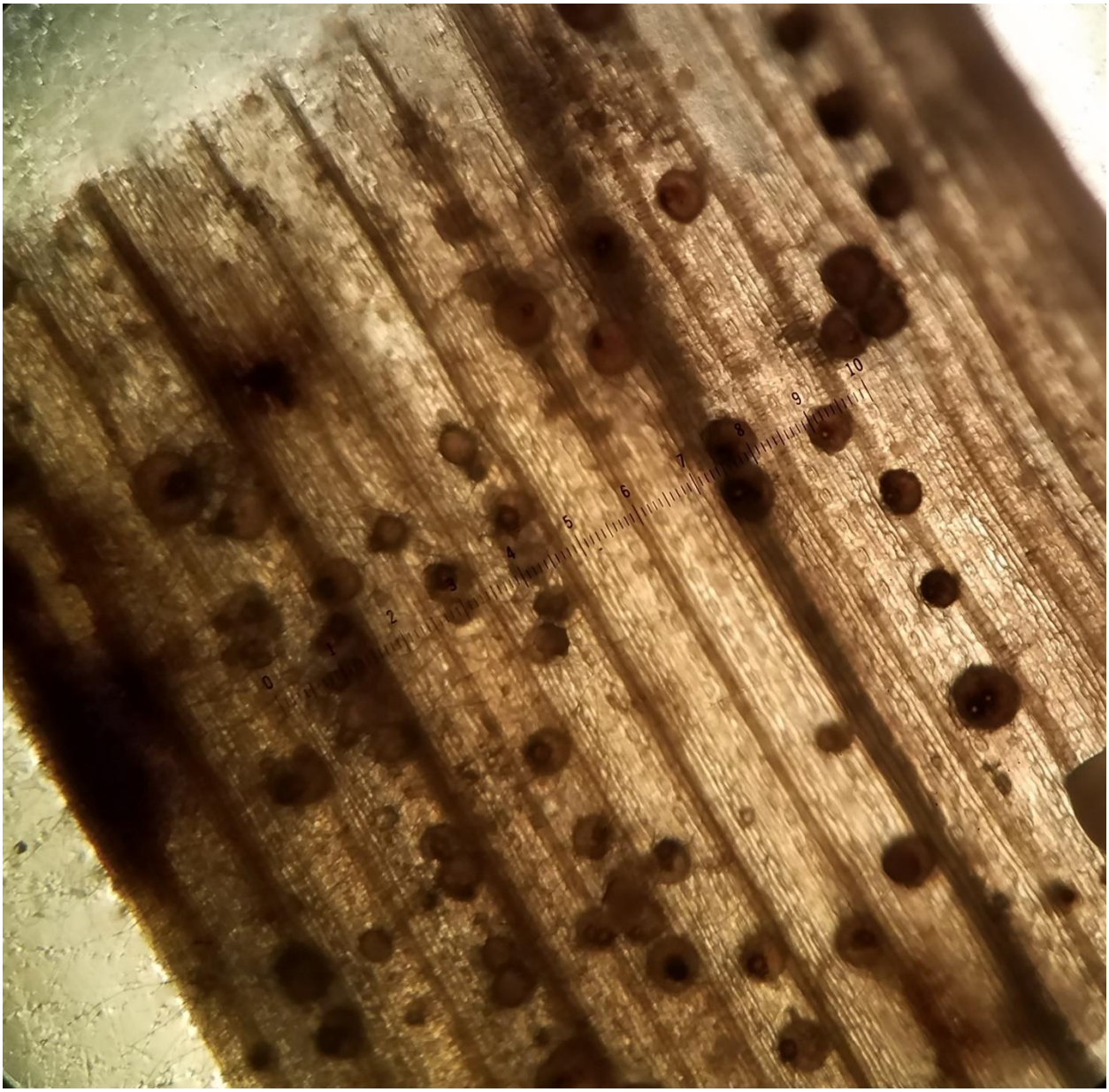

Figure 6. Pycnidia of *Parastagonospora nodorum* on wheat leaf tissue, five days after incubation on moist filter paper. In some pycnidia, circular opening (ostiolum) is also visible. (Courtesy Urmila Adhikari).

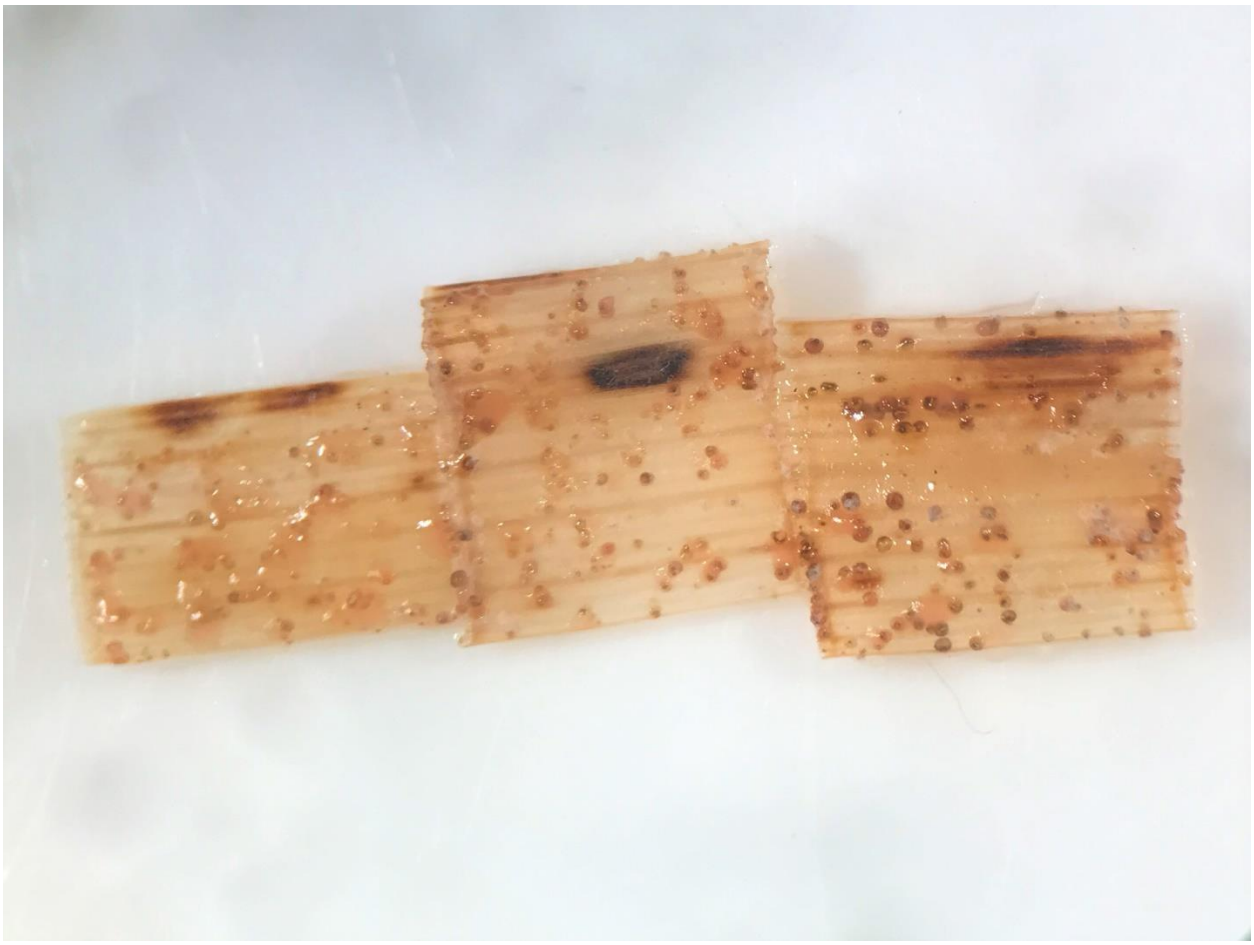

Figure 7. Pycnidia of Parastagonospora nodorum on wheat straw, seven days after incubation on water agar. (Courtesy Urmila Adhikari).

Figure 8. Seven-day-old colony of *Parastagonospora nodorum* on V-8 agar medium in 9-cm diameter Petri dish. (Courtesy Urmila Adhikari).

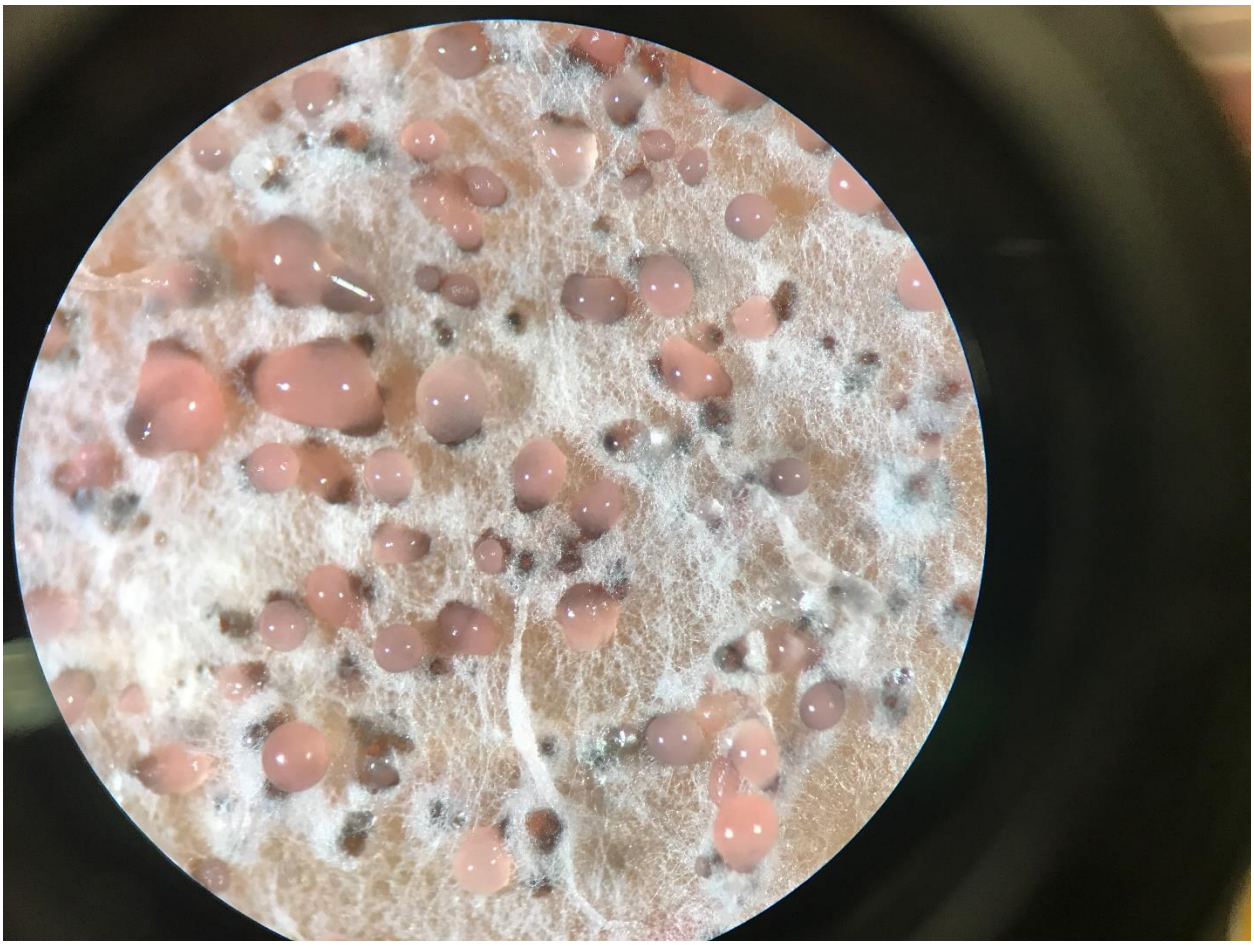

Figure 9. Mucilaginous masses (cirrhi) oozing out of pycnidia (asexual fruiting bodies) of *Parastagonospora nodorum* that are formed on artificial growth medium (V8-agar medium); magnified at 54×. (Courtesy Urmila Adhikari).

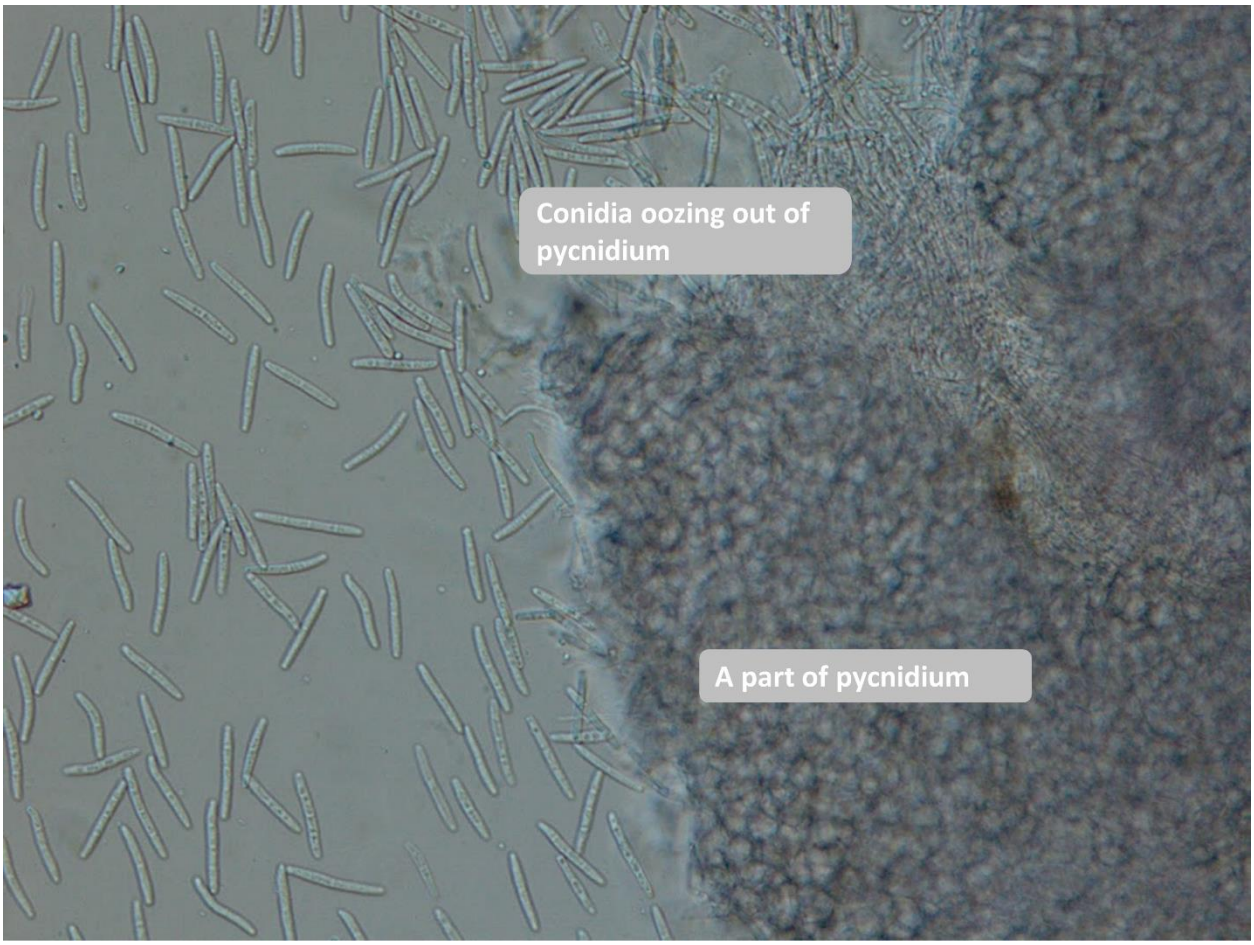

Figure 10. Conidia released from pycnidium of *Parastagonospora nodorum*; magnified at 400×. (Courtesy Urmila Adhikari).

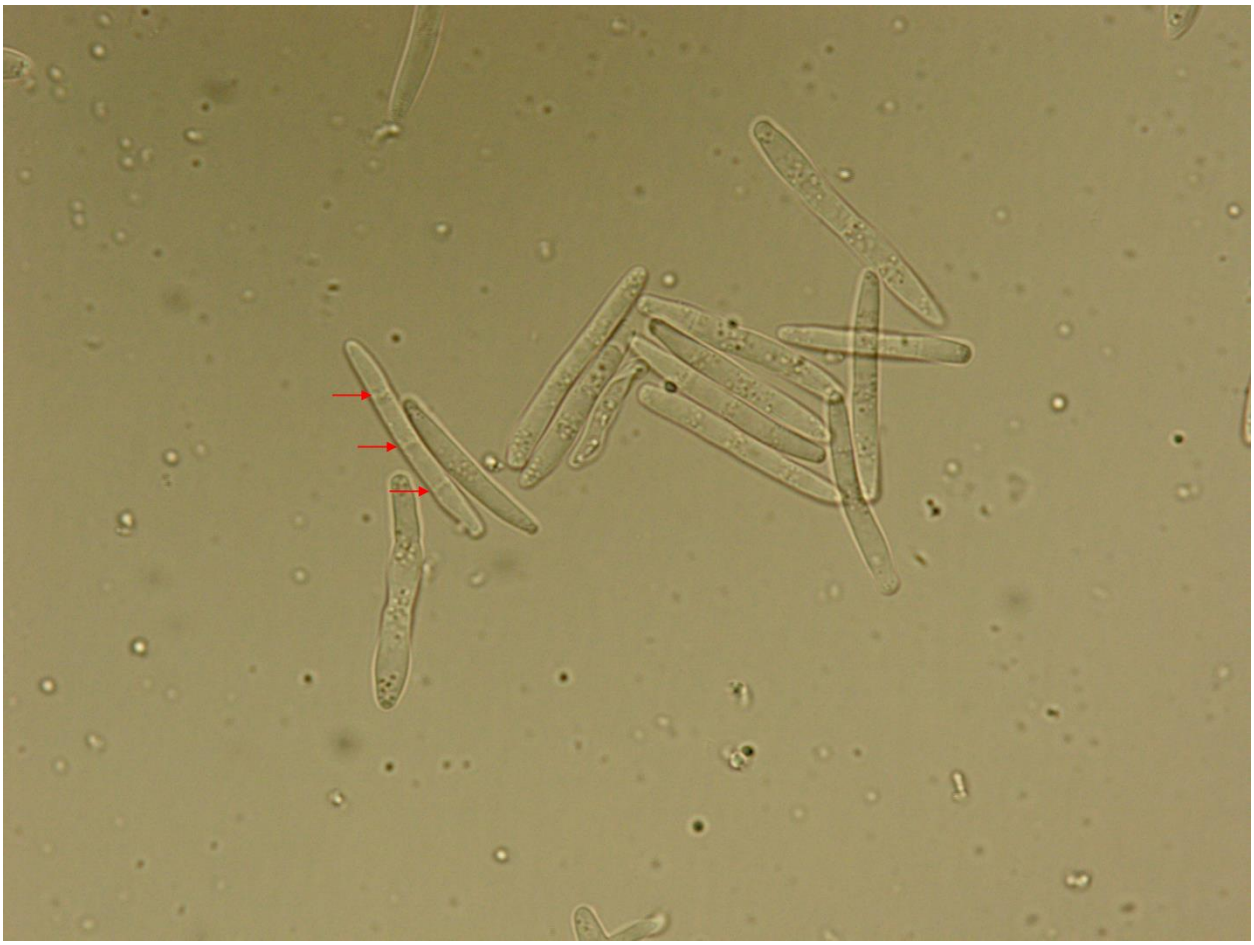

Figure 11. Conidia of *Parastagonospora nodorum*. Red arrows indicate three septa of a conidium; magnified at 1000× (Courtesy Urmila Adhikari).

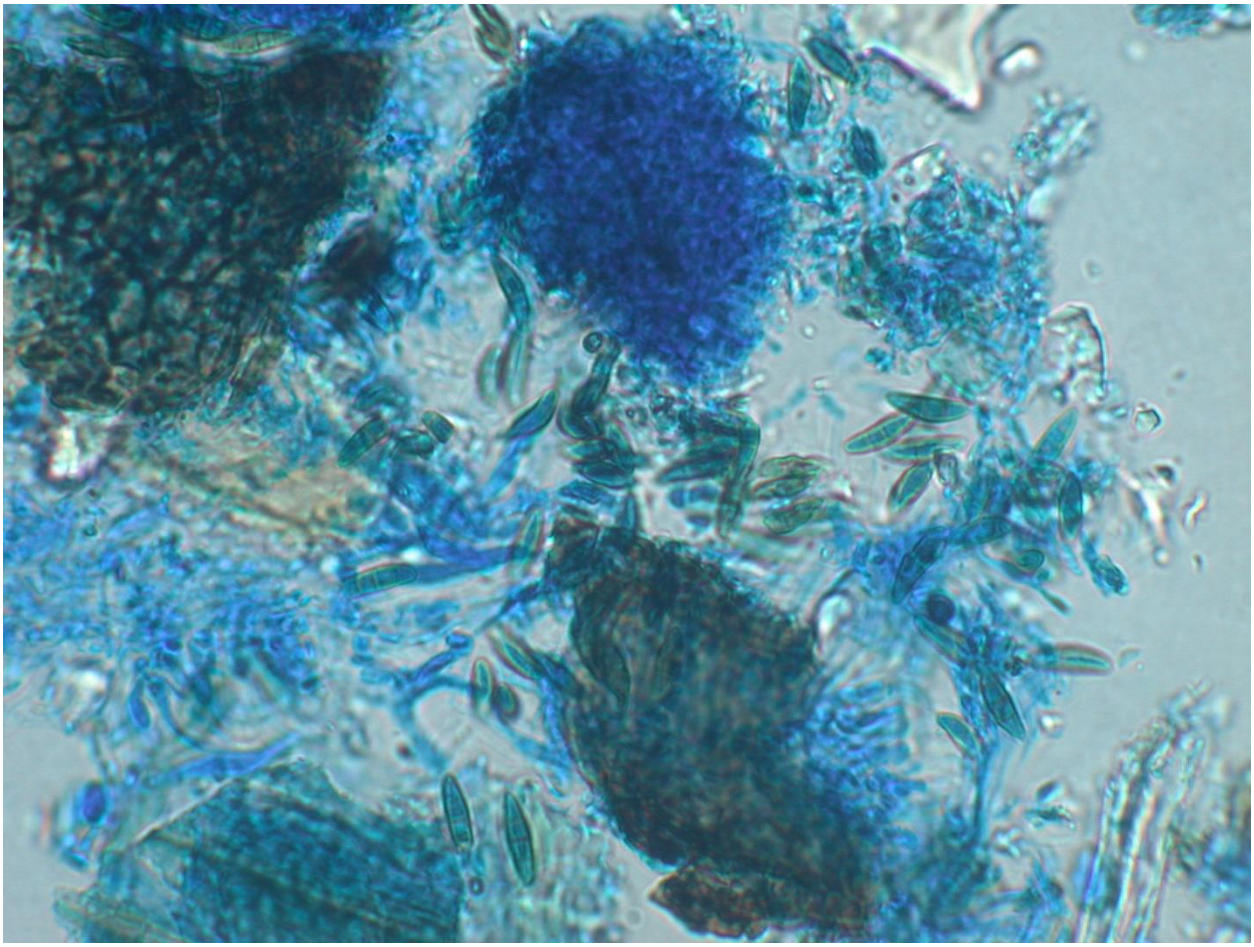

Figure 12. Ascospores of *Parastagonospora nodorum* stained in lactophenol blue; magnified at 400× (Courtesy Christina Cowger).

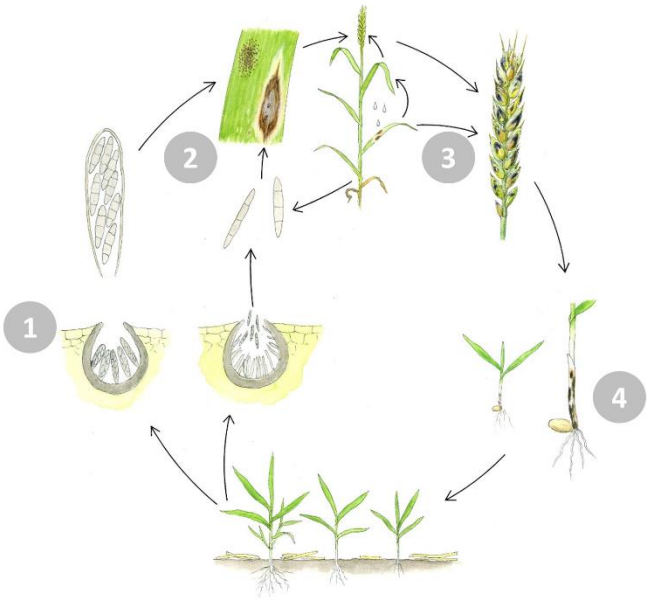

Figure 13. Disease cycle of Septoria nodorum blotch, caused by *Parastagonospora nodorum* (modified from Brodal et al. 2009, original drawing by Hermod Karlsen). Legend is as follows: (1) The pathogen overwinters in wheat debris in the form of pseudothecia and/or pycnidia. (2) The primary inoculum can be in the form of windborne ascospores released from pseudothecia or from splash-dispersed conidia released from pycnidia. (3) Vertical spread of the pathogen occurs with splash dispersal of conidia from lower canopy to upper, and to glumes eventually. (4) If infected seed is used for planting the following season, seedling infection can occur from the dormant mycelium in the seed.

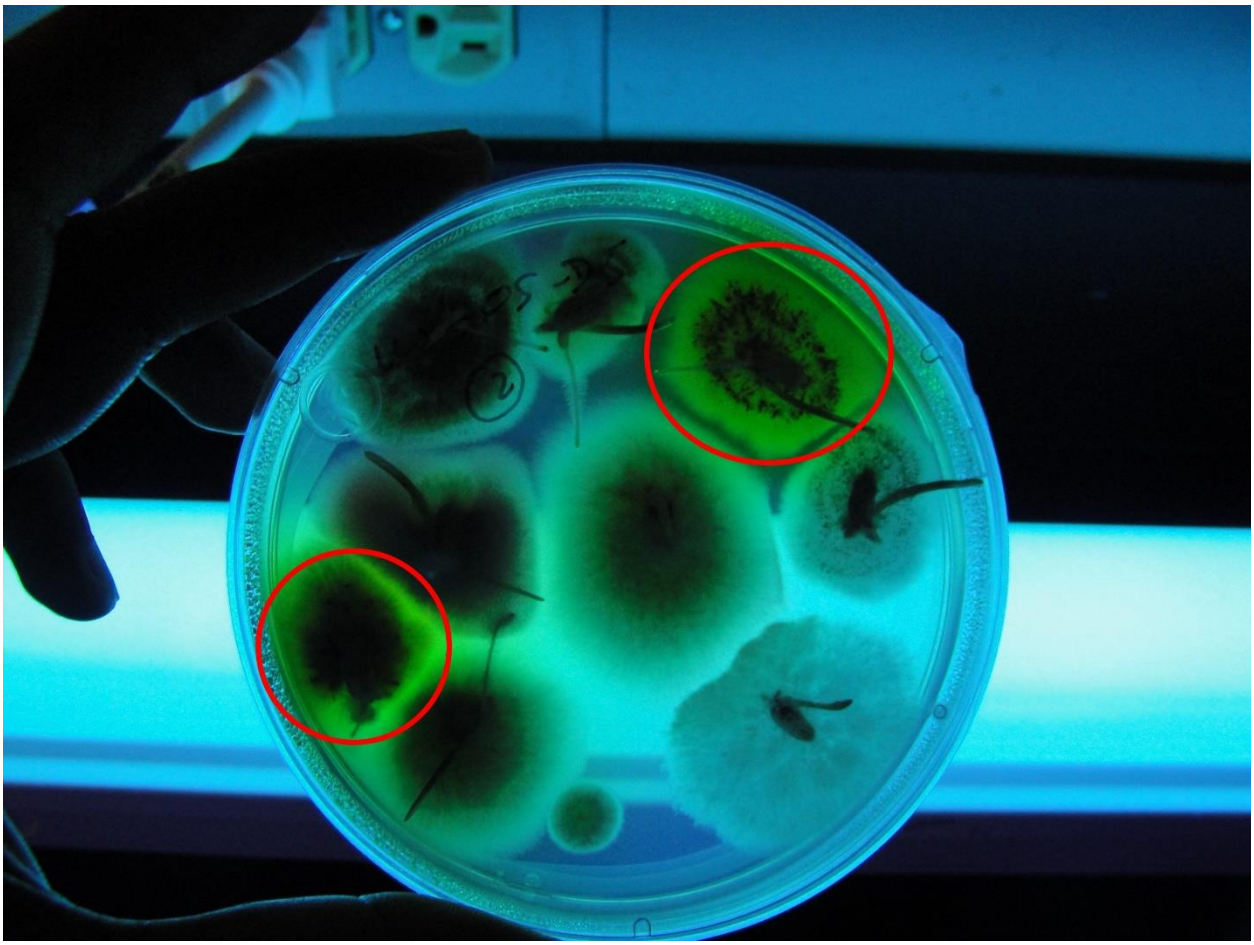

Figure 14. Seed infected with *Parastagonospora nodorum* fluorescing under near-ultraviolet light when grown on a selective medium for the pathogen (Courtesy Lucky Mehra).

Figure 15. SNB susceptibility (left) compared to moderate resistance (right) in advanced experimental winter wheat lines screened in an inoculated, irrigated USDA-ARS SNB nursery. Piedmont Research Station, North Carolina, 2009.