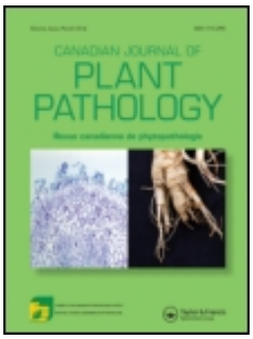

Canadian Journal of Plant Pathology

ISSN: 0706-0661 (Print) 1715-2992 (Online) Journal homepage: www.tandfonline.com/journals/tcjp20

An overview of the ergot (*Claviceps purpurea*) issue in western Canada: challenges and solutions

J. G. Menzies & T. K. Turkington

To cite this article: J. G. Menzies & T. K. Turkington (2015) An overview of the ergot (*Claviceps purpurea*) issue in western Canada: challenges and solutions, Canadian Journal of Plant Pathology, 37:1, 40-51, DOI: 10.1080/07060661.2014.986527

To link to this article: https://doi.org/10.1080/07060661.2014.986527

Can. J. Plant Pathol., 2015
Vol. 37, No. 1, 40–51, http://dx.doi.org/10.1080/07060661.2014.986527

Taylor & Francis
Taylor & Francis Group

Symposium contribution / Contribution à un symposium

An overview of the ergot (*Claviceps purpurea*) issue in western Canada: challenges and solutions

J. G. MENZIES[1] AND T. K. TURKINGTON[2]

[1]*Agriculture and Agri-Food Canada, Cereal Research Centre, 101 Route 100, Morden MB, R6M 1Y5, Canada*
[2]*Agriculture and Agri-Food Canada, Lacombe Research Centre, 6000 C & E Trail, Lacombe AB, T4L 1W1, Canada*

(Accepted 6 November 2014)

Abstract: Ergot, caused by the ascomycete fungus *Claviceps purpurea* (Fr.) Tul., has been described to occur since the Middle Ages, but more recently has become a significant issue on the Canadian prairies. In 1999, there was an outbreak of ergot where 12% of all Canadian Western Red Spring (CWRS) and 4% of Canadian Western Amber Durum (CWAD) wheat samples delivered to elevators were infested with ergot sclerotia. A similar outbreak occurred in Manitoba in 2005, followed by outbreaks in 2008 and 2011 in all three Prairie provinces. In 2008, 12–15% of all CWRS samples were infested with ergot, while in 2011, these levels rose to 15–29% of all CWRS samples. In CWAD wheat, 15% of samples were infested in Saskatchewan in 2008 and 14% in 2011. Management practices that may influence ergot severity include crop rotation, management and nutrition, seeding practices, pesticide applications, nature of the crop (i.e. autumn vs. spring crop and self-pollinated vs. cross-pollinated host species), and harvest and postharvest management. Recently, research has been conducted on host resistance and avoidance of infection, although problems in assessing host resistance and variability among pathogen strains have been encountered. Unfortunately, none of these control measures has been found to be entirely successful in controlling ergot on their own. As a consequence, producers should always approach ergot management as an integrated process with strategies that target all components of the disease triangle. Although ergot outbreaks can be sporadic in nature, sustained research funding is necessary to support research that leads to the development of more effective strategies for ergot management.

Keywords: *Claviceps purpurea*, disease avoidance, disease management, ergot, pathogen variability, resistance, *Triticum aestivum*, *Triticum turgidum* var. *durum*, wheat

Résumé: C'est depuis le Moyen-Âge que nous connaissons l'ergot causé par l'ascomycète *Claviceps purpurea* (Fr.) Tul., mais, récemment, c'est devenu un problème de taille sur les Prairies canadiennes. En 1999, il y a eu une éclosion d'ergot. En conséquence, 12 % des échantillons de blé roux de printemps de l'Ouest canadien (CWRS) et 4 % de ceux de blé dur ambré de l'Ouest canadien (CWAD) livrés aux silos-élévateurs étaient infestés par des sclérotes d'ergot. Une éclosion semblable s'est produite au Manitoba en 2005, suivie d'autres éclosions en 2008 et 2011 dans les trois provinces des Prairies. En 2008, de 12 à 15 % de tous les échantillons de CWRS étaient infestés par l'ergot, tandis qu'en 2011, ces taux ont atteint de 15 à 29 % de tous les échantillons. Chez le CWAD, en 2008 en Saskatchewan, 15 % des échantillons étaient infestés et, en 2011, 14 %. Les pratiques de gestion qui peuvent influencer la gravité de l'infestation incluent la rotation des cultures, la gestion des sols et la fertilisation, les méthodes d'ensemencement, les applications de pesticides, le type de culture (c.-à-d. culture d'automne *vs* culture de printemps et espèces hôtes à pollinisation directe *vs* celles à pollinisation croisée) et la gestion durant et après la récolte. Bien que des problèmes aient surgi quant à l'évaluation de la résistance de l'hôte et à la variabilité chez les souches d'agents pathogènes, dernièrement, des recherches ont été menées sur la résistance de l'hôte et sur l'évitement de l'infection. Malheureusement, aucune de ces mesures de lutte ne s'est avérée pleinement satisfaisante quant à leur gestion individuelle de l'ergot. Les producteurs devraient alors toujours aborder la gestion de

Correspondence to: J. Menzies. E-mail: jim.menzies@agr.gc.ca
This manuscript was a contribution to the symposium entitled 'Ergot and strip rust: continuing challenges for Canadian cereal producers', held during the Canadian Phytopathological Society Annual Meeting in Edmonton, Alberta, June 2013.

Published online 15 Dec 2014

l'ergot en tant que processus intégré comportant des stratégies qui ciblent toutes les composantes du triangle de la maladie. Bien que les éclosions d'ergot soient sporadiques, le financement soutenu est essentiel pour encourager la recherche qui mène à l'élaboration de stratégies plus efficaces en ce qui a trait à sa gestion.

Mots clés: blé, *Claviceps purpurea*, ergot, évitement des maladies, gestion des maladies, résistance, *Triticum aestivum*, *Triticum turgidum* var. *durum*, variabilité de l'agent pathogène

Introduction

Ergot is one of those diseases that can capture people's imaginations. Often associated with the Middle Ages, the ability of the fungus to produce alkaloids which can, at times, reap terrible devastation on people and livestock, or at times, be used as lifesaving drugs, instils in us a measure of curiosity, awe and fear. This interesting mix of 'good' and 'evil' entices most of us to be somewhat knowledgeable of the pathogen and its disease cycle. The reality is, however, that there are very few active plant pathologists that have extensively worked with this pathogen and can be considered experts on the agricultural aspects (non-alkaloid chemistry) of this disease.

The sporadic incidence of ergot from year to year is likely one reason why few plant pathologists have worked extensively on this disease. Interest in this disease is stimulated in years when there is a large outbreak on cereal crops, but this interest wanes in subsequent years if the disease is not present. Incidence and severity depends greatly on weather. Cool damp weather favours ergot by promoting germination of sclerotia and ascospore release, while inhibiting pollination and extending the period of host susceptibility when florets are open (Bailey et al. 2003).

Incidence of ergot in Canada

Ergot was not reported at significant levels throughout most of the 1980s and 1990s on the Canadian Prairies. In 1999, surveys conducted by the Grain Research Laboratory as part of the Harvest Survey Program of the Canadian Grain Commission (Anonymous 2013) revealed that 12% of all samples of Canadian Western Red Spring (CWRS) wheat and 4% of Canada Western Amber Durum (CWAD) contained ergot sclerotia. In Saskatchewan, 17% of the CWRS wheat delivered to the elevators was infested with ergot at an average 0.02% ergot sclerotia by weight. Manitoba had a problem with ergot in 2005, when 10% of CWRS wheat samples were infested with ergot sclerotia (Fig. 1). This was followed by ergot infestation levels of CWRS wheat samples in Alberta, Saskatchewan and Manitoba of 12%, 15% and 13%, respectively in 2008. These levels declined in 2009, but in 2011, ergot infestation levels rose to 29%, 19% and 15% in Alberta, Saskatchewan and Manitoba, respectively. The 2012 levels of ergot infestation of CWRS wheat declined by 50% or more from 2011, but in Alberta and Saskatchewan, they were still three to five times greater than what was found in 2002 to 2007.

Ergot infestation of durum wheat samples has also increased over this time period. Ergot infestation of durum wheat samples started to increase in 2008 in Alberta, when 5% of samples were infested, and has steadily increased to a 10% infestation level in 2012 (Fig. 2). Saskatchewan did not experience a steady increase in ergot infestation of durum wheat, but had two severe years in 2008 and 2011, when 15% and 14% of durum wheat samples contained ergot sclerotia, respectively. (Data for durum wheat samples are not reported for Manitoba because there were too few samples for a meaningful comparison with Alberta and Saskatchewan.)

Causal agent and impact

The ergot diseases of different monocots are caused by species of the fungal genus *Claviceps*. The species of greatest interest on cereals in western Canada is

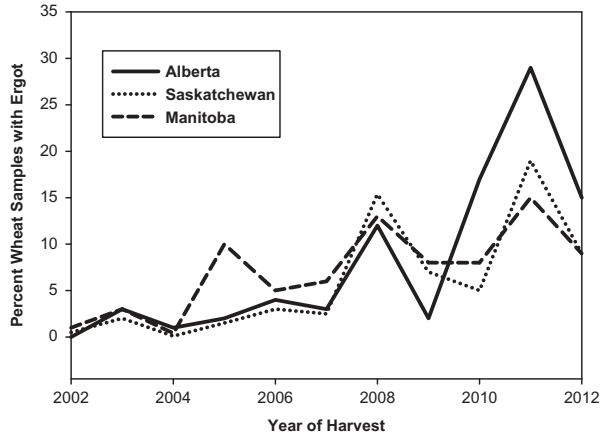

Fig. 1 The percent of Canadian Western Red Spring samples infested with ergot delivered to elevators on the Canadian Prairies. Data provided by the Canadian Grain Commission from their Harvest Sample Program. www.grainscanada.gc.ca.

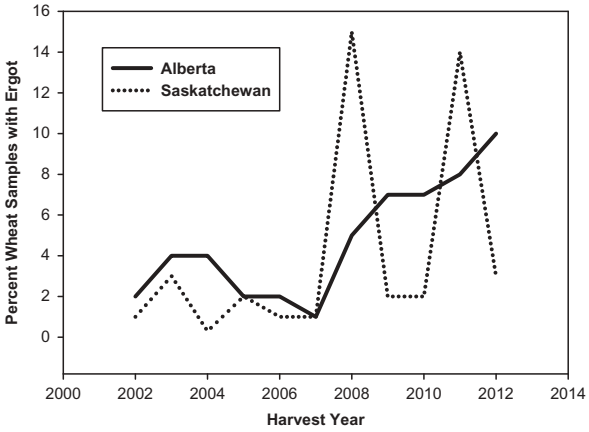

Fig. 2 The percent of Canadian Western Amber Durum samples infested with ergot delivered to elevators on the Canadian Prairies. Data provided by the Canadian Grain Commission from their Harvest Sample Program. www.grainscanada.gc.ca.

Claviceps purpurea (Fr.) Tul. (Domain-Eukarya, Kingdom – Fungi, Phylum – Ascomycota, Class – Sordariomycetes, Order – Hypocreales, Family – Clavicipitaceae). *Claviceps* is Latin for 'club headed' and *purpurea* means 'purple'. 'Club headed' and 'purple' refer to the conspicuous dark purple to black sclerotia or ergot bodies which are quite visible on infected heads of susceptible crops such as rye (*Secale cereal* L.) (Fig. 3), triticale (*X triticosecale* Wittmack) and wheat [*Triticum aestivum* L., and *T. turgidum* L. var. *durum* (Desf.) Mk.]. Sclerotia can be so common on rye that early botanical drawings of the rye plant included sclerotia without realizing that they were not part of the rye plant (Gaudet et al. 2000; Schumann 2000). The term 'ergot' is of French origin, referring to the 'argot' or cockspur (large pointed nail) on the heel of a cock. The sclerotia or ergot bodies produced by the fungus on grass heads resemble 'argots' or cockspurs (Fig. 3).

The effect of the ergot bodies on the host crop is a replacement of the seed, resulting in a reduction in yield. Yield loss is usually of minor and secondary importance. A greater impact of this pathogen comes from the presence of various toxic alkaloids in the ergot bodies, which can infest the grain and cause severe health problems to humans and other animals that ingest the grain (Gaudet et al. 2000; Schumann 2000; Bailey et al. 2003). Ingestion of the sclerotia by humans, often through products made from flour from grain infested with sclerotia, results in ergotism, a disease that can cause devastating physiological and psychological effects. The ergot bodies contain psychoactive compounds similar to lysergic acid diethylamine (LSD) (Moir 1955). Severe chronic ergot poisoning can turn

Fig. 3 (Colour online) Protruding sclerotia of *Claviceps purpurea* on rye. (Reproduced with permission of the Canadian Phytopathological Society; Bailey et al. 2003).

sufferers into screaming, gibbering individuals whose fingers, toes, arms and legs blacken and corrode away from gangrene (Gaudet et al. 2000; Schumann 2000; Bailey et al. 2003). Women can suffer from miscarriages and reduced fertility. Livestock, including horses, cattle, sheep, pigs and chickens, which are fed ergot-infested grain, can suffer severely as well. These animals can have a reduced growth rate, and an increased food-to-gain ratio. Severe cases can include gangrene of extremities, diarrhoea, internal bleeding, reduced pregnancy and abortion. Death can result in both humans and livestock if proper treatment is not provided. The negative effects of ergot sclerotia has resulted in severe downgrading penalties for ergot-infested grain. The standard of quality of different grades of cereals or cereal classes regarding ergot infestation in Canada is listed in Table 1 (Canadian Grain Commission 2012). There is a grade reduction as the amount of ergot in the grain increases,

Table 1. The percent weight of ergot which can be tolerated in different grades of grain from different grain classes in Canada (Canadian Grain Commission 2012).

Wheat, rye, oats, triticale and barley	Threshold ergot levels (% net weight)				
	Grade				
	1	2	3	4	Feed
CWRS, CWHWS, CWAD[1]	0.01	0.02	0.04	0.04	0.1
CWRW, CWSES, SER, CERS, CEHRW, CEAD, CEHWW, CEWW, CESWS, CEHWS[2]	0.01	0.02	0.04		0.1
CWES, CPSR, CPSW[3]	0.03	0.06			0.1
CWGP[4]	0.1	0.1			
Canada Western Rye	0.05	0.02	0.33		
Canada Western Oats	Nil	0.025	0.025	0.05	
Canada Eastern Oats	Nil	0.05	0.05	0.1	
Triticale	0.025	0.05	0.1		
General Purpose Barley	0.05	0.1			
Select Malting Barley	0.025				
Select Food Barley	0.02				

[1]Wheat Classes: Canada Western Red Spring, Canada Western Hard White Spring and Canada Western Amber Durum.
[2]Wheat Classes: Canada Western Red Winter, Canada Western Soft White Spring, Canada Eastern Red, Canada Eastern Red Spring, Canada Eastern Hard Red Winter, Canada Eastern Amber Durum, Canada Eastern Hard White Winter, Canada Eastern White Winter, Canada Eastern Soft White Spring and Canada Eastern Hard White Spring.
[3]Wheat Classes: Canada Western Extra Strong, Canada Prairie Spring Red and Canada Prairie Spring White.
[4]Wheat Class: Canada Western General Purpose.

which results in a lower price for the grain, or, in the worst case, a rejection of the grain for sale.

Ergot and St Anthony's fire

Historically, *C. purpurea* has likely been associated with its grass hosts for thousands of years. It is reported to attack more than 400 species of the Gramineae (Bove 1970; Gaudet et al. 2000). There are numerous references to the conspicuous sclerotia in historical records. The greatest impacts of this fungus to humans have been in association with its most susceptible cereal host, rye. Rye was the popular grain corn of the Teutons or Germans, and was grown extensively during the Middle Ages in an area extending from France, through Holland, Germany, Poland and Central Russia. Ergot was probably always associated with these crops, but the first recorded large-scale epidemic from ergot was recorded in the Rhine Valley in 857 A.D. Thousands of peasants died, and affected individuals often experienced an intense burning sensation and were thought to be cursed by God. The disease became known as *sacer ignis* or 'holy fire'. Ergot epidemics during the Middle Ages were sporadic, occurring every 25–50 years. Ergot poisoning was often most frequent shortly after harvest, as the previous year's food reserves were exhausted and the grain falling from the ear during harvest was immediately ground into flour. This grain was often heavily infested with ergot sclerotia, as the sclerotia are easily detached from the spikes at harvest. It was also seen as a disease of the poor, as the wealthy could afford to buy clean grain, or other high-quality grains such as wheat, which is less susceptible to *C. purpurea*. Gaston de la Valloire built a hospital to care for those afflicted by ergotism in 1039 in France and dedicated the hospital to the memory of St Anthony. The disease then became known as 'St Anthony's fire'. An order of monks, the Order of St Anthony, was established to care for those suffering from this disease, and sufferers often improved under their care. This 'cure' was likely the result of the monks feeding the peasants a diet without ergoty bread, which would allow recovery from the ergotism. The peasants could relapse, however, when they returned home and resumed their diet of ergoty bread.

The cause of St Anthony's fire became a little better understood in the 1670s, when a French physician named Thuillier observed that city dwellers and the wealthy did not succumb to ergotism, but the country peasants were quite susceptible (Gaudet et al. 2000; Schumann 2000). He reasoned that the disease must be linked to the peasants' diet. He observed rye fields heavily infected with ergot, and being a physician, knew the medicinal uses and toxic effects of ergot bodies. He was, however, unable to prove that the ergot bodies were responsible for ergotism, and convince the peasants to stop eating ergoty grain. Gradually during the 1700s, more and more evidence began to point to the ergot sclerotia as the cause of St Anthony's fire, and people started to avoid eating ergoty rye, if possible. Rye was also becoming less important as a staple in the diet of peasants, being replaced by a newly introduced crop, the potato. It was approximately 200 years after Thuillier that Louis Tuslane in 1853 (Tuslane 1853) and Kühn in 1863 (Walker 1950) unravelled the life cycle of *C. purpurea* and demonstrated that the ergot bodies were fungal sclerotia.

Ergot is still commonly found on grasses in temperate regions of the world, but our understanding of the cause of ergotism has allowed us to eliminate ergot poisoning of humans in most developed countries. Problems can still arise, however, if proper care is not taken. An outbreak of ergotism occurred as late as 1951 in Pont-Saint-Esprit, France (Gaudet et al. 2000) despite our knowledge of the cause of this malady. The infliction, which became locally known as 'Le Pain Maudit' affected more than 250 people, resulting in 4–7 deaths, and approximately

30–50 people being interned in an asylum. It was traced back to an unscrupulous farmer, miller and baker who took advantage of the low price of the ergoty grain to make a profit. Ergotism can still be a problem for animals grazing in ryegrass pastures if the ryegrass has gone to seed or where farmers unknowingly feed ergoty grain to various livestock species.

Life cycle of *C. purpurea*

Ergot bodies are the overwintering sclerotia of the fungus, *C. purpurea*, an obligate parasite in nature (Fig. 4). The sclerotia are easily dislodged from the florets when the host crop is mature and during harvest, and may fall to the ground or contaminate the grain (Fig. 5) (Gaudet et al. 2000; Schumann 2000; Bailey et al. 2003). The sclerotia may be introduced into a field if contaminated seed is sown. Sclerotia may also be present from volunteer or wild grasses in the ditches or headlands of fields. Sclerotia can survive in or on the soil or in contaminated grain for about 1 year (Mitchell & Cooke 1968), although it has been suggested that they can survive for up to 3 years in the field (Rapilly 1968). The sclerotia require 4–8 weeks of cold temperature treatment near 0°C, followed by several weeks of temperatures near 18°C, for optimal germination. Optimal germination occurs during cool, wet weather in the spring or summer. Sclerotial germination appears to be only loosely correlated with the development of potential host plants (Wood & Coley-Smith 1982). There is no evidence that sclerotial germination is influenced by host-produced 'factors' which ensure a synchrony of development of host and pathogen. Sclerotia on or near the soil surface germinate and produce 1–60 drumstick-like ascomata (Sprague 1950) (Fig. 6). These ascomata have knob-shaped heads which contain numerous perithecia. These flask-shaped fruiting bodies are filled with numerous club-shaped asci. Each ascus contains eight filliform ascospores. There must be adequate soil moisture or rainfall for ascospores to be forcibly ejected into the air (Schwartingl & Hiners 1945; Hadley 1968; Colotelo & Cook 1977; Alderman 2003). The ascospores are disseminated by wind currents or rain splash (Bove 1970) and must land on a host stigma or ovary for infection to occur. Ascospores germinate and infect the ovary within 24 hours. A mass of mycelium called a sphacelia develops on the infected ovary surface, upon which millions of conidia are produced (Bove 1970). These conidia become suspended in a sugary host sap and are often exuded from the floret in droplets known as honeydew (Fig. 7). Campbell (1957) found that the average time between infection and honeydew appearance was 6.9 days for barley, 8.5 days for rye and 10.3 days for wheat. The conidia in the honeydew can cause secondary spread of the pathogen to other host

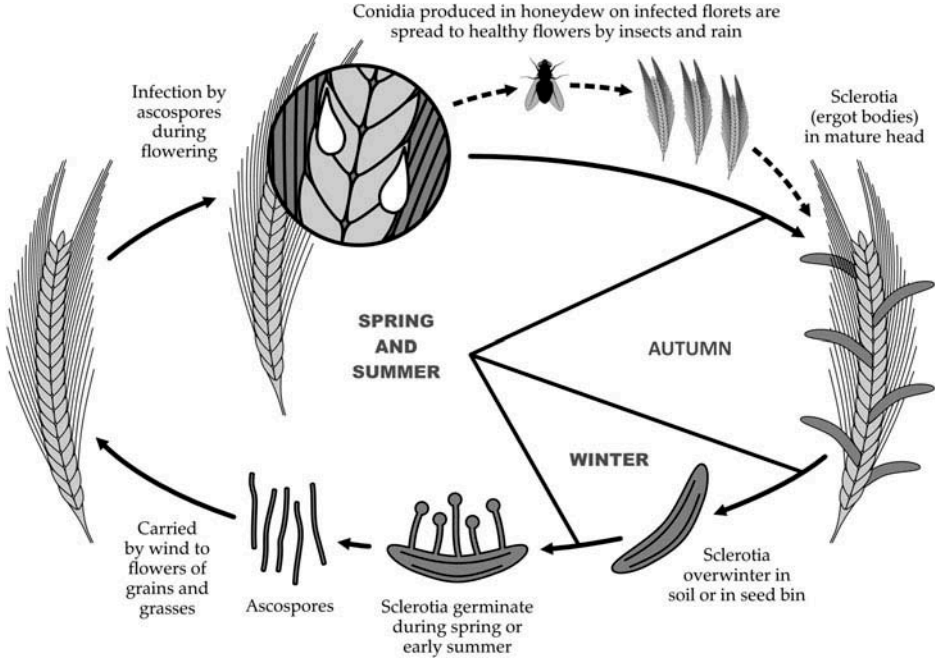

Fig. 4 Disease cycle of *Claviceps purpurea* on cereals and other grasses. (Reproduced with permission of the Canadian Phytopathological Society; Bailey et al. 2003).

Fig. 5 (Colour online) Rye grain infested with sclerotia of *Claviceps purpurea*. (Reproduced with permission of the Canadian Phytopathological Society; Bailey et al. 2003).

Fig. 6 (Colour online) Germinated sclerotium of *Claviceps purpurea* with ascomata. (Reproduced with permission of the Canadian Phytopathological Society; Bailey et al. 2003).

Fig. 7 (Colour online) Honeydew exuding from florets of durum wheat infected with *Claviceps purpurea*. (Photograph by Michael Shillinglaw, AAFC).

flowers either by rain splash, or by insects which are attracted to the honeydew. The honeydew must be diluted by dew or rain in order for the conidia to germinate (Schwartingl & Hiners 1945). The mycelia in the infected ovary develop into sclerotial hyphae, which elongate from the base and differentiate into a hard, purple-black surface layer, an inner fertile hyphal mass and a central layer of storage cells (Bove 1970). The maturation of these sclerotia completes the fungal disease cycle.

Management of ergot

There are a number of different strategies for management of ergot. None of the control measures have been found to be entirely successful in controlling ergot on

their own, so one should always approach ergot management as an integrated process.

Reduction of primary inoculum

The sclerotia of *C. purpurea* are the overwintering stage, so reducing the number of sclerotia in the area will reduce the amount of initial inoculum for primary infection in the spring and early summer. The use of ergot-free or certified seed (maximum two ergot bodies per kg seed for wheat, barley and oats, and maximum four ergot bodies per kg seed for rye and triticale) would help prevent the introduction of sclerotia into the field (Bailey et al. 2003). The sclerotia are believed to survive normally for 1 year, but some can survive up to 3 years (Mitchell & Cooke 1968; Rapilly 1968), so crop rotation with crops that are not susceptible to ergot following susceptible crops would result in reductions of viable sclerotia. Cultivation following a crop which was infected with ergot or deep seeding when using ergot-infested seed would help to bury the sclerotia. Sclerotia which are buried to a depth of 5 cm will germinate but the stipes are generally of insufficient length to reach the soil surface to release ascospores (Bretag & Merriman 1981). Burning of stubble will reduce the number of viable sclerotia, but reaching the critical temperatures needed to destroy sclerotia over an entire field may be difficult to achieve (Bretag 1985; Johnston et al. 1996).

Role of alternative hosts

The wide host range of *C. purpurea* means that it is important to consider other grass species that grow in the areas such as ditches, headlands or adjacent fields when trying to control ergot (Campbell 1957). The importance of these reservoirs of inoculum is indicated by the most severely infected areas of grain fields being usually near the edges of the fields (Campbell & Freisen 1959). The presence of susceptible hosts in adjacent areas would result in a source of sclerotia produced on these hosts for overwintering of the pathogen and providing initial inoculum the next season. These grass hosts have a role in epidemic development beyond one of producing sclerotia, however. The different flowering periods of the different susceptible grass species allows a wider time range for *C. purpurea* to infect a susceptible host. A study in the UK suggests that ascospores may be important in infecting early flowering host plants, such as early flowering grasses in field margins or autumn-seeded crops (Bayles et al. 2009). Infection of wheat florets by ascospores was rare, but infection of wheat by secondary conidia occurred frequently in tillers around the edges of wheat plots. The secondary conidia were transported from infected honeydew-producing flowers of grassy hosts in field margins to the wheat flowers by direct contact or local rain splash over short distances or by insect vectors over longer distances. Wood & Coley-Smith (1982) concluded that the potential for disease spread from a primary focus was large, with the majority of infections occurring in tillers of late flushes. Secondary spread of infection or sclerotial production in field margins can be controlled by mowing to prevent flowering of susceptible grass hosts in the margins. Herbicides can also be used to selectively control susceptible grass weeds in fields and field margins.

Fungicides

Research on the use of fungicides to control ergot has been conducted in the UK and the USA. Fungicide seed treatments have been found to reduce germination and/or production of ascomata by sclerotia treated with fungicide (Shaw 1988; Puhl et al. 2007). However, fungicide seed treatments may delay decomposition of sclerotia, allowing them to persist for periods of more than 1 year (Shaw 1988). Foliar fungicide treatments have had mixed success in preventing infection of host florets by *C. purpurea*. The multiple applications of fungicides have reduced levels of ergot bodies in harvested grain. However, most trials determined that fungicide applications to control ergot are not very effective or economically practical (Schumann 2000; Evans et al. 2000; Gladders et al. 2001; Bailey et al. 2003). It is also important to note that most of the fungicides used in these trials are not registered for use in Canada or not registered for use to control ergot in Canada.

Cultural management

Proper management of the host crop can also influence the incidence of ergot infection. The use of ergot-free seed with high germination, sown at an appropriate density at an even depth in a well-prepared soil bed will promote a uniformly developing crop (Bailey et al. 2003). This can reduce the number of late-developing tillers in the crop, which tend to be the tillers most severely infected by *C. purpurea* (Bayles et al. 2009). Autumn sown crops are usually less infected than spring sown crops. The flowering of autumn sown crops occurs earlier in the spring when there is less secondary spread of the pathogen from other nearby grasses (Sosulski & Bernier 1975; Bailey et al. 2003). Proper timing and rates of herbicide applications will help reduce ergot infection by reducing rates of floret sterility (Bailey et al. 2003).

Proper soil fertility can be important in reducing severe effects of ergot infection. Ergot is more severe on copper- and boron-deficient plants (Graham 1983; Evans et al. 2007). Copper deficiency leads to the development of small anthers and pollen sterility (Graham 1975) so that the floret must open to become fertilized instead of staying closed for self-pollination in crops such as wheat and barley. Similar responses to boron deficiency have been reported (Graham 1983). Ensuring adequate levels of copper and boron are present in soils will help to improve pollen viability. This will ensure cereal flowers remain closed and less accessible to any ergot ascospores or foraging insects contaminated with conidia that may be present during the heading period (Evans et al. 1990, 2007; Simojoki 1991; Solberg et al. 1994). However, application of copper and boron does not eliminate the risk of ergot, as other factors such as cool or hot temperatures at heading, or improperly timed herbicide applications may lead to pollen sterility and thus increase ergot risk. For example, Hamm et al. (2006a, 2006b) found that soil incorporation of copper or boron or foliar application of boron did not reduce ergot in harvested seed lots of tall fescue.

Host genotype

The incidence and severity of ergot has been reported to be influenced by host genotype. Rye and triticale are considered the most susceptible of hosts among the cereal crops, followed by wheat, barley and oat (Dillon-Weston & Taylor 1942; Platford & Bernier 1976). The greater susceptibility or incidence of ergot in rye and triticale is thought to be the result of their flowers remaining open for a longer period of time as compared with wheat, barley and oat, giving the pathogen greater opportunity to infect (Gaudet et al. 2000). Additionally, wheat and barley are less affected by *C. purpurea* because these crops are self-pollinated with fewer sterile florets on the spike (Puranik 1971). The use of male sterile lines of wheat and barley to produce F_1 hybrid lines can result in severe levels of infection by *C. purpurea* because the florets are open for a longer period of time than male fertile lines, and pollination will not occur until after flower opening. Pollination prior to inoculum introduction to the host floret favours the host. Fertilized ovaries are susceptible immediately after fertilization, but by 4 days after fertilization, susceptibility decreases until at 7–9 days after fertilization, ovary infection does not occur in wheat and barley (Puranik 1971; Darlington & Mathre 1976; Bayles et al. 2009).

The variation among lines within species for susceptibility or proneness to infection by *C. purpurea* has not been extensively studied. Nevertheless, some studies have been conducted examining the differences in ergot severity among lines of rye (Sosulski & Bernier 1975), wheat (Watkins & Littlefield 1976; Pageau et al. 1994; Menzies 2004; Bayles et al. 2009; Malo & Hucl personal communication) and barley (Pageau & Lajeunesse 2006; Oxley et al. 2009), with differences reported. While these studies have identified 'resistant' germplasm, they have also served to demonstrate some of the issues with identifying germplasm resistant to ergot, and potential pitfalls when trying to breed for ergot-resistant lines.

A cereal genotype may be more or less prone to *C. purpurea* infection as a result of avoidance of infection, or physiological resistance. Avoidance of infection can be through mechanical factors which prevent access to the susceptible infection site by the pathogen. This may include flowering early to avoid high levels of ergot inoculum, self-fertilization before flower opening, a very short period of flowering to limit exposure to pathogen inoculum or high floret fertility so that few sterile florets are available for infection. Experiments to identify resistant germplasm have relied on natural infection in field experiments, or active inoculation. The use of natural infection can help to identify germplasm that is less prone to infection, but it does not demonstrate if the lower levels of ergot infection are the result of disease avoidance or physiological resistance. Experiments utilizing natural infection experience a high degree of variability in ergot infection among replicates, sites and years, and inconsistency in variety ratings (Sosulski & Bernier 1975; Pageau & Lajeunesse 2006; Bayles et al. 2009). This is a reflection of the large influence environmental conditions have on the life cycle of the pathogen and the flowering of the host. Despite this, researchers have been able to identify some lines which have consistently low infection levels, and others which have consistently high infection levels.

Avoidance of infection

Two interesting studies examining ergot avoidance by assessing flower 'openness' of different wheat and barley lines in relation to their relative susceptibility to *C. purpurea* were conducted in the UK by Bayles et al. (2009) and Oxley et al. (2009). Oxley et al. (2009) examined three flowering characteristics of barley. The three characteristics were flowering time (early or late), flowering duration (ranged from 2 to 5 days) and flower habit. The flower habit was considered closed if the anthers only partly emerged or did not emerge, such that very few anthers are apparent, partly open if only a few anthers are observed per spike, or open if there are one or two anthers

observed in many florets of the spike. Bayles et al. (2009) used four flowering characteristics of wheat to attempt to quantify the degree of flower openness of wheat lines and determine if this could infer any degree of escape from infection by ergot. The four characteristics studied were anther extrusion (no anther appearing to anther fully extruded), anther size (small to large), ear density (number of spikelets/length of rachis) and proportion of blind florets (number of empty grain sites/ear as a percentage of the total number of florets). Oxley et al. (2009) conducted their experiments over 2 years, but only experienced ergot infection in 1 year. Oxley et al. (2009) separated their lines based on ergot severity into not detected, low, moderate and high. Some of the lines had ergot severities which could be predicted from their flowering habits, i.e. open flowers with long flowering duration on a line with high ergot severity, or closed flowers with short flowering duration on lines with no ergot detected. Lines that flowered early or late, with closed or partly opened flowers with a flowering duration of 2–5 days could be found in categories ranging from not detected to moderate ergot severity (there was only one line in the high ergot severity category). Bayles et al. (2009) conducted their study over 3 years. They observed a high degree of variability in ergot infection over sites and years when wheat lines were exposed to natural infection, and little consistency in terms of variety effects. There was an overriding effect of environment on flower openness. They also inoculated the florets of some of their lines to determine physiological resistance and found that lines with good physiological resistance always had good field performance. They concluded that varietal differences in openness of flowering are unlikely to be a major determinant of the relative susceptibility of different varieties in the field, and tissue resistance is of more importance.

Physiological resistance

Active inoculation of florets of different cereal lines is useful in determining the physiological resistance of these lines to infection by *C. purpurea*. Wheat has been the small grain cereal studied the most in this regard, although not very many studies have been conducted. One of the earliest studies was conducted by Campbell & Tyner (1959) who examined varietal differences among 12 lines of barley to ergot infection at different times of heading. They inoculated the barley lines at 2-day intervals, starting at heading and finishing 10 days after heading, and assessed the percent infection of florets. They found that if the plants were inoculated at heading, most lines were susceptible, but one barley line, 'Peatland', was less infected than the others. As the time from heading for inoculation increased, the susceptibility to ergot infection decreased. Some lines became resistant to infection quicker than others, so that at different times after heading, the relative resistance rating of the lines varied. This suggests that the timing of inoculation may affect the resistance ranking of different genotypes. The most susceptible lines did not vary in their relative ratings to the rest of the lines; they were always the most susceptible. Campbell & Tyner (1959) compared their results from inoculation tests to field tests where the barley lines were exposed to natural inoculum. They observed that lines that showed susceptibility under natural infection, also showed susceptibility when inoculated, and lines with good resistance in the field had shown good resistance when inoculated.

A number of studies examining ergot resistance in lines of wheat have been conducted by Platford & Bernier (1970, 1976), Puranik (1971), Darlington & Mathre (1976), Watkins & Littlefield (1976), Coley-Smith & Watkinson (1987), Menzies (2004) and Bayles et al. (2009). Puranik (1971) studied male sterile lines of barley and wheat, and observed no differences among the lines in terms of the percentage of florets infected. All the other studies observed differences among lines of wheat for resistance to the pathogen. These findings are significant because it indicates that differences in susceptibility among lines of wheat (and in barley in the case of Campbell & Tyner 1959) to *C. purpurea* is not a rare trait, even though some of the differences were not great. Unfortunately for producers, the lines with the best resistance to ergot were often breeding lines which would not be suitable for commercial production.

Resistance to *C. purpurea* infection was observed in the durum line 'Carleton' by Platford & Bernier (1970). They found that 'Carleton' was more resistant than the durum line 'Stewart 63′ in terms of percentage of florets with sclerotia, size of individual sclerotia and amount of honeydew produced. They concluded that the resistance in 'Carleton' would be adequate for commercial lines because the lower number of small sclerotia being produced would reduce the initial inoculum for subsequent years, and the reduced honeydew production would reduce secondary spread. Menzies (2004) also tested 'Carleton', but did not find it to be any more resistant than any of the other lines tested. Menzies reported that the durum line 'Pelissier' had significantly lower honeydew production than most lines tested, and the breeding line 9260B-173A to be significantly more resistant than all other lines tested in terms of percentage of florets with sclerotia, size of sclerotia and honeydew production. In terms of hexaploid spring wheats, the line 'Kenya Farmer' has been reported to be resistant to *C. purpurea*

in a few studies. Platford & Bernier (1970) observed 'Kenya Farmer' to have significantly lower values for percentage of florets with sclerotia, size of sclerotia and honeydew production. Similarly, Darlington & Mathre (1976) reported 'Kenya Farmer' to be the most resistant line in their tests. Menzies (2004) found 'Kenya Farmer' to be more resistant to ergot infection than the other lines tested, but only in terms of honeydew production. Bayles et al. (2009) studied lines of winter wheat in the UK and found 'Robigus' to be significantly more resistant to ergot than 'Solstice' and 'Riaito' in terms of weight of sclerotia per spike and the mean weight of individual sclerotia. The identification of lines with better resistance is encouraging, as improvements to commercial lines are therefore possible. However, assessment of ergot resistance can be a time consuming and labour intensive process. Studies are underway with some of these lines to characterize the resistance and its nature of inheritance. If molecular markers for these different types of resistance could be developed, it would greatly enhance any breeding efforts to develop ergot resistance lines.

Pathogen variability

The identification of lines of different species with resistance to *C. purpurea* is a good step in the development of commercial resistant lines. The numbers of studies in this area are few, however, and the effectiveness of the identified sources of resistance in different geographic locations has not been examined. Campbell (1957) studied the host specificity of 421 isolates of *C. purpurea* on 38 different host species. He found that each grass species became infected and concluded that indigenous and forage grasses constitute a reservoir of ergot inoculum for rye, wheat and barley. There was no host specificity among ergot strains. Darlington et al. (1977) took this a step further and examined the existence of specific biotypes or races within *C. purpurea*. They inoculated 48 strains of the pathogen to eight lines of barley and four lines of wheat. They observed a large range in the aggressiveness of these isolates, but did not find good evidence of specific races. Cagaš & Macháč (2002) inoculated seven cultivars of Kentucky bluegrass (*Poa pratensis* L.) with four strains of *C. purpurea* (one from the Czech Republic, two from Germany and one from the USA) to determine if the amount of ergot bodies formed varied according to strain. They found significant differences in the amount of ergot bodies formed between the European strains as compared with the American strain, and concluded that their American strain was more aggressive. Menzies (unpublished data) has also studied strain variability among 42 strains of *C. purpurea* from western Canada, and five strains from the UK kindly supplied by Dr D. O'Sullivan and Dr A. Gordon (National Institute of Agricultural Botany, Cambridge, UK). Strain variability was studied by needle inoculation of a differential host series made up of eight spring wheat lines found to have different levels of resistance by Menzies (2004), supplemented by three winter wheat lines with different levels of resistance as determined by Bayles et al. (2009). The results are being analysed, but some preliminary data of honeydew production on three durum wheat lines after inoculation with four western Canadian strains and four UK strains of *C. purpurea* along with the honeydew rating scale are presented in Table 2. The wheat lines chosen for this work were 9260B-173A because it was found to be highly resistant to the pathogen, 'Kyle' because it had some weak resistance and 'Melita' because it was susceptible (Menzies 2004). Line 9260B-173A was resistant to the Canadian strains, but this line was not resistant to the UK strains of the pathogen. The UK strains were able to produce honeydew quite well on 9260B-173A, perhaps with the exception of strain UK-2. 'Kyle' was found to have good resistance to the UK-2 strain, and to a lesser extent to Canadian strain C-1, and UK strain UK-1. 'Kyle' was susceptible to strains C-2 and UK-4. 'Melita', the susceptible line, was susceptible to all strains, except for strain UK-2. *Claviceps purpurea* strain UK-2 may be a less aggressive strain than all the others, which would agree with the finding of Darlington et al. (1977) and Cagaš & Macháč (2002) that some strains are less aggressive than others. The reactions of 'Kyle' to the different strains ranged from 1 for UK-2 and 1.6 for C-1, suggesting good resistance to 3.7 for strain C-2 and 3.8 for strain UK-4. This indicates that different strains of *C. purpurea* have differential reactions, possibly different virulence, on different wheat genotypes. For breeding programmes, this suggests that

Table 2. The reaction of three lines of durum wheat (*Triticum turgidum* L. var. *durum*) to needle inoculation with four western Canadian strains and four UK strains of *Claviceps purpurea* in terms of honeydew production (Menzies, unpublished data).

	Strain of *C. purpurea*							
Wheat line	C-1	C-2	C-3	C-4	UK-1	UK-2	UK-3	UK-4
9260B-173A	1.1[1]	1	1	1	4	2.3	3.4	3.8
'Kyle'	1.6	3.7	2.8	2.8	1.5	1	2.8	3.8
'Melita'	3.8	4	4	3.8	3.8	1.8	3.9	3.9

[1]The spikes were rated from 1 to 4 with 1 = none, 2 = honeydew confined within the glumes, 3 = honeydew exuding from the florets in small drops, and 4 = large drops of honeydew running down the spike.

the strains used in assessing germplasm for resistance need to be carefully considered to achieve effective resistance in the field.

Harvest and post-harvest management

Harvest and post-harvest practices can reduce the amount of ergot in the harvested grain, if ergot infection is evident. The delaying of swathing or harvest can reduce the amount of ergot in harvested grain because the ergot bodies can be shaken off the spikes by the wind. Selective harvesting of the field can also be used to separate out heavily infested ergot grain from clean grain. As noted above, the areas of the field around grassy ditches or headlands are often the most severely affected because of the epidemiology of this disease. Harvesting the areas of the field close to these wild grass areas separately from the rest of the field will separate out the grain which is more heavily infected from the cleaner grain (Bailey et al. 2003). Commercial cleaning of grain using gravity sorters or colour sorters can remove sclerotia from infested grain (Bailey et al. 2003). The cleaning of the grain does have limitations, however, and heavily infested grain may not be cleaned sufficiently enough for the market. Heavily infested grain which cannot be adequately cleaned should be disposed of, preferably by burying. Heavily infested grain should not be used as feed for livestock.

Summary

Traditionally, ergot has been an issue for cereals that are typically cross-pollinated, particularly triticale and rye. The disease can also affect wheat and barley, but in the prairie region of Canada, it tended to be an infrequent problem on these latter two crops. More frequent reports of significant levels of ergot and downgrading of grain lots have occurred in wheat across the prairie region since 1999, with additional reports of ergot in barley. One potential reason for this increase in prevalence and severity may be the shortening of crop rotations, as the typical rotation across many areas of the prairies has become canola-cereal-canola-cereal. A 1-year break away from cereals such as wheat or barley may not be sufficient to allow for the natural destruction of the ergot sclerotia in or on the soil. Furthermore, tight rotations may have increased the levels of ergot bodies in grassy areas adjacent to cereal fields, thus resulting in the build-up and spread of *C. purpurea* inoculum. Although there are a number of different strategies for management of ergot, it is difficult for cereal producers to completely eliminate the risk of ergot. None of the recommended control measures have been found to be entirely successful in controlling ergot on their own. As a consequence, producers should always approach ergot management as an integrated process with strategies that target all components of the disease triangle. Crop rotation directly targets the persistence of ergot sclerotia. Preventing flower development and thus the potential for disease development in grassy hosts in field border areas can reduce sclerotial production and thus a future inoculum source. Limiting plant stress via adequate soil fertility and avoidance of herbicide applications close to crop flowering, while reducing late tiller development in cereals through increased seeding rates, can reduce ergot risk. Ultimately, host resistance, especially physiological resistance will play a key role in providing improved ergot management. Effective long-term management of ergot requires an investment in research on this disease. However, the sporadic nature of ergot from year to year is likely one reason why few plant pathologists have worked extensively on this disease. Interest in this disease is stimulated in years when there is a large outbreak on cereal crops, but this interest wanes in subsequent years if the disease is not present.

References

Alderman SC. 2003. Diversity and speciation in *Claviceps*. In: White JF, Bacon CW, Hywel-Jones NL, Spatafora, JW editors. Clavicipitalean fungi: evolutionary biology, chemistry, biocontrol, and cultural impacts. New York, NY: Marcel Dekker, Inc.; p. 221–225.

Anonymous. 2013. Harvest survey of Canadian grain quality [online]. Canadian Grain Commission. 28 May 2013. Available from http://www.grainscanada.gc.ca/quality-qualite/hsp-per/hs-er-eng.htm [accessed 30 October 2013].

Bailey KL, Gossen BD, Gugel RK, Morrall RAA. 2003. Diseases of Field Crops in Canada. 3rd ed. Saskatoon, SK: The Canadian Phytopathological Society and University Extension Press, University of Saskatchewan; p. 89–93.

Bayles R, Fletcher M, Gladders P, Hall R, Hollins W, Kenyon D, Thomas J. 2009. Towards a sustainable whole-farm approach to the control of ergot. Project Report No. 456 to the Home Grown Cereals Authority, Stoneleigh Park, UK; p 170.

Bove FJ. 1970. The Story of Ergot. S. Karger AG, Basel, SUI; p. 298.

Bretag TW. 1985. Control of Ergot by a selective herbicide and stubble burning. Trans Br Mycological Soc. 85:341–343. doi:10.1016/S0007-1536(85)80199-6

Bretag TW, Merriman PR. 1981. Effect of burial on survival of sclerotia and production of stromata by Claviceps purpurea. Trans Brit Mycol Soc. 77: 658–660.

Cagaš B, Macháč R. 2002. Different pathogenicity of ergot isolates [*Claviceps purpurea* (Fr.) Tul.] on Kentucky bluegrass (*Poa pratensis* L.). Plant Prot Sci. 38:18–22.

Campbell WP. 1957. Studies on ergot infection in gramineous hosts. Can J Bot. 35:315–320. doi:10.1139/b57-028

Campbell WP, Freisen HA. 1959. The control of ergot in cereal crops. Plant Dis Rep. 43:1266–1267.

Campbell WP, Tyner LE. 1959. Comparison of degree and duration of susceptibility of barley to ergot and true loose smut. Phytopathology. 49:348–349.

Canadian Grain Commission. 2012. The Official Grain Grading Guide. http://grainscanada.gc.ca/oggg-gocg/04/oggg-gocg-4f-eng.htm [Accessed 08/05/2013]

Coley-Smith JR, Watkinson L. 1987. Susceptibility of durum wheat cultivars to ergot disease. Ann Appl Biol. 110:162–163.

Colotelo NC, Cook W. 1977. Perithecia and spore liberation of *Claviceps purpurea*: scanning electron microscopy. Can J Bot. 55:1257–1259. doi:10.1139/b77-146

Darlington LC, Mathre DE. 1976. Resistance of male sterile wheat to ergot as related to pollination and host genotype. Crop Sci. 16: 728–730. doi:10.2135/cropsci1976.0011183X001600050034x

Darlington LC, Mathre DE, Johnston RH. 1977. Variation in pathogenicity between isolates of *Claviceps purpurea*. Can J Plant Sci. 57:729–733. doi:10.4141/cjps77-106

Evans I, Solberg E, Huber DM. 2007. Copper and plant disease. In: Datnoff LE, Elmer WH, Huber DM, editors. Mineral nutrition and plant disease. St. Paul. MN: The American Phytopathological Society; p. 177–188.

Evans VJ, Jenkyn JF, Gladders P, Mantle PG 2000. Fungicides for control of ergot in cereal crops. Proceedings of the BCPC Conference: Pests and Diseases 2000, 1–3:511–514.

Gaudet D, Menzies J, Burnett P. 2000. Smuts, Bunts and Ergot. Encyclopedia of Microbiology. 4. 2nd ed. San Diego, CA, USA: Academic Press; p. 297–315.

Gladders P, Evans EJ, Jenkyn JF. 2001. Project report no. 254. Laboratory and field testing of fungicides for control of ergot in wheat and rye. London: Home Grown Cereals Authority.

Graham RD. 1975. Male sterility in wheat plants deficient in copper. Nature. 254:514–515. doi:10.1038/254514a0

Graham RD. 1983. Effects of nutrient stress on susceptibility of plants to disease with particular reference to the trace elements. Adv Bot Res. 10: 221–276. doi:10.1016/S0065-2296(08)60261-X

Hadley G. 1968. Development of stromata in *Claviceps purpurea*. Trans Br Mycological Soc. 51:763–769. doi:10.1016/S0007-1536(68)80096-8

Hamm PB, David NL, Horneck DA. 2006b [online]. Controlling ergot with soil and foliar applied fungicides in seedling tall fescue var. Labarinth grown for seed in the southern Columbia Basin. In: Young WC, editor. Seed Production Research at Oregon State University. USDA-ARS Cooperating. EXT/CrS 126, 4/07. Corvallis, OR, USA: Oregon State University; P. 84–85. http://seed-ext.cropandsoil.oregonstate.edu/Pub?2006/30.pdf. [[Accessed 30 October 2013]].

Hamm PB, David NL, Horneck DA. 2006a [online]. Controlling ergot with soil applied copper, foliar applied boron, and foliar applied Quilt in established tall fescue in the southern Columbia Basin. In: Young WC, editor. Seed Production Research at Oregon State University. USDA-ARS Cooperating. EXT/CrS 126, 4/07. Corvallis, OR, USA: Oregon State University; p. 82–83. http://seed-ext.cropandsoil.oregonstate.edu/Pub?2006/29.pdf. [[Accessed 30 October 2013]].

Johnston WJ, Golob CT, Sitton JW, Schultz TR. 1996. Effect of temperature and postharvest field burning of Kentucky bluegrass on germination of sclerotia of *Claviceps purpurea*. Plant Dis. 80:766–768. doi:10.1094/PD-80-0766

Menzies JG. 2004. The reactions of Canadian spring wheat genotypes to inoculation with *Claviceps purpurea*, the causal agent of ergot. Can J Plant Sci. 84:625–629. doi:10.4141/P03-086

Mitchell DT, Cooke RC. 1968. Some effects of temperature on germination and longevity of sclerotia in *Claviceps purpurea*. Trans Br Mycological Soc. 51:721–729. doi:10.1016/S0007-1536(68)80092-0

Moir JC. 1955. The history and present-day use of ergot. Can Med Journ. 72:727–734.

Oxley SJP, Havis ND, Hoad SP 2009. Understanding ergot risk in spring barley. Project Report No. 457 to the Home Grown Cereals Authority, Stoneleigh Park, UK.; p. 37.

Pageau D, Collin J, Wauthy J-M. 1994. Une note sur la résistance à l'ergot chez le blé tendre, le blé dur et le triticale. Phytoprotection. 75: 45–49. doi:10.7202/706051ar

Pageau D, Lajeunesse J. 2006. Évaluation de la résistance à l'ergot (*Claviceps purpurea*) chez l'orge et le blé. Phytoprotection. 87: 63–68. doi:10.7202/013974ar

Platford RG, Bernier CC. 1970. Resistance to *Claviceps purpurea* in spring and durum wheats. Nature. 226:770. doi:10.1038/226770a0

Platford RG, Bernier CC. 1976. Reaction of cultivated cereals to *Claviceps purpurea*. Can J Plant Sci. 56:51–58. doi:10.4141/cjps76-009

Puhl T, Suty-Heinze A, Adam N. 2007. Mutterkornbekämpfung – bei der Saatguthygiene fängt es an. Gesunde Pflanzen. 59: 147–150. doi:10.1007/s10343-007-0166-y

Puranik SB. 1971. Biology and control of ergot on male sterile wheat and barley. Phytopathology. 61:1075–1080. doi:10.1094/Phyto-61-1075

Rapilly F. 1968. Ètudes sur l'ergot du blé: *Claviceps purpurea* (Fr.) Tul. Ann Epiphyt. 19:305–329.

Schumann GL. 2000. Ergot. The Plant Health Instructor. doi:10.1094/PHI-I-2000-1016-01.

Schwartingl AE, Hiners LD. 1945. A study of domestic ergot of wheat and rye. J Am Pharmaceut Assoc. 34:11–16.

Shaw S 1988. Further evaluation of triadimenol seed treatments for the control of ergot in cereal seed. BCPC Mono. No. 39: Application to seeds and soil. Proceedings of a symposium organized by the British Crop Protection Council, Univ. Surrey, Guildford, UK 4–6 Jan. 1988; p. 105–112.

Sosulski F, Bernier CC. 1975. Ergot tolerance in spring rye. Can Plant Dis Surv. 55:155–157.

Sprague R. 1950. Diseases of Cereal Grasses in North America. New York, NY: The Ronald Press Company.

Tulasne L-R. 1853. Mémoire sur l'ergot des glumacéses. Ann Sci Nat (Parie Botanique). 20:5–56.

Walker JC. 1950. Plant Pathology. New York, NY: McGraw-Hill Book Company, Inc.; p. 699.

War D-W, Taylor RE. 1942. Observations on ergot in cereal crops. J Agric Sci. 32:457–466. doi:10.1017/S0021859600010364

Watkins JE, Littlefield LJ. 1976. Relationship of anthesis in Waldron wheat to infection by *Claviceps purpurea*. Trans Br Mycological Soc. 66:362–363. doi:10.1016/S0007-1536(76)80076-9

Wood G, Coley-Smith JR. 1982. Epidemiology of ergot disease (*Claviceps purpurea*) in open-flowering male-sterile cereals. Ann Appl Biol. 100:73–82. doi:10.1111/j.1744-7348.1982.tb07193.x