

Different Pathogenicity of Ergot Isolates (*Claviceps purpurea* [Fr.] Tul.) on Kentucky Bluegrass (*Poa pratensis* L.)

BOHUMÍR CAGAŠ and RADEK MACHÁČ

OSEVA PRO Ltd., Grassland Research Station Rožnov-Zubří, Czech Republic

Abstract

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

Ergot, caused by the fungus *Claviceps purpurea* (Fr.) Tul., belongs to the main constraints in seed production of Kentucky bluegrass (*Poa pratensis* L.). The level of ergot occurrence depends on the weather conditions during the growing period, harvest year, storage conditions of seed, and post-harvest treatment. The degree of resistance of the grown cultivar plays an important role. Based on greenhouse trials with four different ergot populations (Zubří, Czech Republic; Leutewitz and Munich, Germany; Pullman, Washington, USA) during 3 years, we found pathogenicity to be one of the most important factors. There were significant differences in the amount of ergot bodies formed between the Central European populations (Zubří, Leutewitz) and the American one from Washington State. The degree of pathogenicity has a major impact on the occurrence of ergot sclerotia in seed of *Poa pratensis*.

Keywords: *Poa pratensis* L.; *Claviceps purpurea* (Fr.) Tul.; isolates; pathogenicity

Ergot, caused by *Claviceps purpurea* (Fr.) Tul., attacks many grasses, including species grown for seed. Over 20 years of observations in the Czech Republic have shown that varieties of Kentucky bluegrass (*Poa pratensis* L.) belong to the most important host plants (CAGAŠ 2001). Ergot is one of the basic diseases in grass seed growing in the northwestern part of the USA (Oregon, Washington). It causes not only a decrease in seed yield and quality (e.g. poorer storability) of Kentucky bluegrass, but also an increase in production costs (cleaning) and restricted export possibilities (CHASTAIN 1992). Under European conditions, ergot belongs to the important grass diseases, but not to the limiting factors of seed growing. Its occurrence differs between growing areas and years.

The amount of ergot bodies (sclerotia), which affects the intensity of seed cleaning, differs very strongly between the years. It could be assumed that this variation of occurrence is influenced by the following factors:

1. Age of the grass sward (number of harvest years) – with the age of the grassland increases the concentration of ergot sclerotia on the soil surface where they are able to produce ascospores.

2. Favourable climatic conditions in spring stimulate the germination of sclerotia, formation of asci and spreading of ascospores and conidia into grass inflorescences. MÜHLE *et al.* (1971) counted in humid years 6000 sclerotia in 100 g of seed of *Poa pratensis*, which is about 4.5 g of ergot bodies.

3. Storage conditions of contaminated seed – HORN (1989) found that ergot sclerotia in harvested grass seed that was stored in dry and cold conditions lost the ability to germinate. When sown together with grass seed such ergot sclerotia were of no epidemiologic importance.

4. Variety – CAGAŠ (1996), in greenhouse and field trials, detected large differences in the degree of resistance between various cultivars of *Poa pratensis*.

5. Method of cultivation – Kentucky bluegrass grown for seed under irrigation is more attacked by ergot than under standard conditions (JOHNSTON *et al.* 1995).

6. Type of post-harvest management – low cutting and burning of the grass sward after the seed harvest decrease the level of ergot infection in the next seeding year.

These factors can only partially explain the differences in ergot occurrence in various geographical zones, countries and growing areas. It is well known that within *Claviceps purpurea* there exist many specialised strains or populations with different properties and different host ranges (PAŽOUTOVÁ *et al.* 2000), but it is not known how large an impact the different pathogenicity of these populations (strains) have on the severity of ergot incidence. There is no reliable information about the impact of isolates of *C. purpurea* that differ in place of origin on the intensity of creation of ergot bodies. The main aim of this study was, therefore, to investigate the influence of different ergot isolates on the intensity of infection of *Poa pratensis*. It could help to explain the different levels of ergot incidence in various geographical regions.

MATERIAL AND METHODS

Four different isolates of *C. purpurea* were used for artificial inoculation in the greenhouse. Their characteristics are summarised in Table 1.

The sclerotia (six to eight from each origin) were cultured on malt agar in a growth chamber (room temperature); the medium had been modified as follows: brewery malt, casein acid hydrolysate 1%, $MgSO_4 \cdot 7H_2O$ 0.1%, agar 3%. Developed mycelium with conidia were used to prepare the inoculum. Before inoculation the number of conidia was checked and standardised (25 million conidia in 1 ml).

Based on the field observations of a large assortment of Kentucky bluegrass cultivars (CAGAŠ & HORN 1994), seven cultivars differing by degree of resistance to ergot in field conditions were chosen for the glasshouse trials.

Flowering plants of these cultivars (Haga, Krasa, Lovegreen, Moravanka, Rožnovská, Sheba, Slezanka) were inoculated in 1993 on May 27–31, in 1994 on May 26–June 1, in 1995 on June 2–9 in a greenhouse with a

Table 1. Characteristics of four ergot isolates used in the glasshouse trials

Ergot isolate	Origin	Host plant	Date of collection	Collect by
CZ	Zubří, Czech Republic (Grassland Research Station)	<i>Poa pratensis</i> cv. Slezanka	July 1992	B. Cagaš
D I	Leutewitz, Germany (DSV Breeding Station)	<i>Poa pratensis</i> (different cultivars)	July 1992	G. Horn
D II	Munich, Germany (seminatural meadows near the city)	<i>Poa pratensis</i> (natural populations)	September 1992	A. Obst
USA II	Pullman, USA (field trials of Washington State University)	<i>Poa pratensis</i> (different cultivars)	September 1992	W.J. Johnston

Table 2. Pathogenicity of four different ergot isolates, expressed by number and weight of sclerotia

Ergot isolate	Year	Sclerotia (per 100 spikelets)		Ergot isolate	Year	Sclerotia (per 100 spikelets)	
		number	weight (mg)			number	weight (mg)
CZ	1993	3.29	1.05	D II	1993	7.23	3.42
	1994	2.67	1.46		1994	6.02	5.73
	1995	4.97	3.43		1995	3.71	2.50
	Mean	3.64	1.98		Mean	5.65	3.88
D I	1993	1.78	0.37	USA II	1993	10.74	8.94
	1994	1.44	1.52		1994	5.54	6.47
	1995	3.67	3.80		1994	15.02	14.16
	Mean	2.29	1.89		Mean	10.34	9.85

conidial suspension in water between 7 and 10 h at 2-day intervals. The plants used in the trials were transferred from the plant nursery of the Grassland Research Station at Zubří one month before the inoculation (age of plants: sown in April 1992). The inoculated plants were covered with paper bags after honeydew formation. There were four replicates per cultivar, each replicate consisting of 100 spikelets from which the ripe sclerotia were harvested and the number and weight of sclerotia were determined. Differences between the isolates (in number and weight of sclerotia) were analyzed by ANOVA and the significance expressed.

RESULTS AND DISCUSSION

The greenhouse trials with seven cultivars of Kentucky bluegrass with different degrees of susceptibility to ergot and four different ergot isolates carried out under standardised conditions in three years brought the following results (summarised in Table 2):

- the American isolate (USA II) caused the highest production of sclerotia (with one exception in 1994); that production differs significantly or highly significantly from the Czech isolate, highly significantly from the isolate D I and significantly from the isolate D II (weight) – no significance was found in number of sclerotia between the American isolate and isolate from Bavaria (D II) – Table 3. The results of single years 1993–1995 were similar (Table 4);
- two Central European isolates (CZ and D I) produced very low amounts of sclerotia;

– the isolate from Bavaria (D II) caused a relatively high production of sclerotia, and is one of the proofs of ergot differentiation within the European continent.

Although the trials were carried out under standardised conditions in the greenhouse (the differences between the isolates could not be evaluated on the field because of the strong influence of natural conditions) and the level of pathogenicity (expressed by the number and weight of sclerotia) varied between the years, the ability of the USA isolate to cause a higher production of sclerotia was evident (Table 2). The glasshouse trial brought new knowledge about the direct relationship between the degree of pathogenicity and the intensity of formation of ergot sclerotia. The degree of pathogenicity influences very strongly the degree of ergot incidence in grasses grown for seed. That factor was not mentioned in the previous investigations explaining the different incidence of ergot bodies in grasses in various countries.

SITTON *et al.* (2001) investigated similarly the differences in pathogenicity between the USA ergot isolates and the Czech isolate from Zubří; the assortment of American and Czech Kentucky bluegrass cultivars showed a lower harvest of ergot sclerotia after inoculation with the Czech isolate than with USA isolates. The high resistance of cultivar Slezanka to isolates with high pathogenicity was confirmed there.

Based on these investigations it should be recommended to avoid growing Kentucky bluegrass in those areas where populations of the fungus with high pathogenicity occur. To other, non-chemical control possibilities belongs the growing of cultivars with a higher degree of resistance.

Table 3. Significances between four ergot isolates expressed by number of sclerotia in 100 spikelets and weight of sclerotia (mg per 100 spikelets)

	1993	1994	1995	Mean	CZ					
Number of sclerotia (in 100 spikelets)										
CZ	3.29	2.67	4.97	3.64	D I	0				
D I	1.78	1.44	3.67	2.30	D II	0	0			
D II	7.23	6.20	3.71	5.65	USA II	*	**	0		
USA II	10.74	5.54	15.02	10.43		CZ	D I	D II	USA II	
Weight of sclerotia (mg per 100 spikelets)										
CZ	1.05	1.46	3.43	1.98	D I	0				
D I	0.37	1.52	3.80	1.90	D II	0	0			
D II	3.42	5.73	2.50	3.88	USA II	**	**	*		
USA II	8.94	6.47	14.16	9.86		CZ	D I	D II	USA II	

Number of sclerotia
sd_{0.05} 5.15; sd_{0.01} 7.97

Weight of sclerotia
sd_{0.05} 4.52; sd_{0.01} 7.01

0 = not significant; significant at * $P < 0.05$, ** $P < 0.01$

Table 4. Number of sclerotia and weight of sclerotia after inoculation with four different ergot isolates in 1993, 1994 and 1995

Year	Isolate	Varieties							Mean	Significance			
		Lovegreen	Sheba	Haga	Moravanka	Rožnovská	Krasa	Slezanka		CS	D I	D II	USA II
Number of sclerotia (per 100 spikelets)													
1993	CS	3.34	0.85	8.45	2.57	3.52	1.50	2.81	3.29				
	D I	2.15	0.33	4.08	2.02	1.88	0.29	1.73	1.78	0			
	D II	8.11	5.96	12.56	7.14	6.65	6.10	4.07	7.23	*	**		
	USAII	8.85	9.80	17.62	20.64	6.47	8.51	3.30	10.74	**	**	*	
1994	CS	6.06	4.30	0.13	2.15	2.74	3.24	0.21	2.67				
	D I	1.40	1.62	0.02	3.15	2.50	1.17	0.22	1.44	0			
	D II	5.13	5.72	4.18	10.96	4.89	9.67	1.56	6.02	**	**		
	USAII	6.62	8.66	1.46	10.07	4.50	5.28	1.63	5.54	*	**	0	
1995	CS	4.32	5.26	6.34	5.95	5.86	5.50	1.58	4.97				
	D I	5.49	0.26	8.28	3.20	2.78	3.65	2.01	3.67	0			
	D II	4.35	0.54	6.90	7.08	1.76	3.86	1.58	3.71	0	0		
	USAII	10.74	7.46	31.60	19.36	12.57	17.62	5.79	15.02	**	**	**	
Weight of sclerotia (mg per 100 spikelets)													
1993	CS	1.42	0.37	2.56	0.63	1.16	0.48	0.75	1.05				
	D I	0.54	0.07	0.78	0.57	0.30	0.09	0.27	0.37	0			
	D II	5.28	3.61	5.89	2.36	2.90	2.21	1.68	3.42	0	*		
	USAII	8.16	8.39	15.93	15.49	5.91	6.57	2.11	8.94	**	**	**	
1994	CS	3.65	1.41	0.09	1.36	1.63	1.09	0.12	1.46				
	D I	2.05	1.57	0.06	3.22	2.27	1.28	0.21	1.52	0			
	D II	4.59	5.96	3.43	9.85	5.72	9.48	1.08	5.73	**	**		
	USAII	7.74	8.86	2.47	13.29	5.88	5.95	1.11	6.47	**	**	0	
1995	CS	3.58	4.59	4.42	4.34	4.00	2.54	0.56	3.43				
	D I	7.45	0.21	7.42	3.62	3.09	3.64	1.14	3.80	0			
	D II	3.87	0.31	4.78	4.52	1.05	2.21	0.74	2.50	0	0		
	USAII	14.72	8.55	20.05	21.80	11.58	19.52	2.88	14.16	**	**	**	
Number of sclerotia					Weight of sclerotia								
1993: $sd_{0.05}$ 3.15		1994: $sd_{0.05}$ 2.23		1995: $sd_{0.05}$ 4.57		1993: $sd_{0.05}$ 2.92		1994: $sd_{0.05}$ 2.37		1995: $sd_{0.05}$ 3.73			
$sd_{0.01}$ 4.31		$sd_{0.01}$ 3.06		$sd_{0.01}$ 6.25		$sd_{0.01}$ 4.00		$sd_{0.01}$ 3.24		$sd_{0.01}$ 5.11			

0 = not significant; significant at * $P < 0.05$, ** $P < 0.01$

References

- CAGAŠ B. (1996): Study of differential resistance of Kentucky bluegrass varieties to ergot (*Claviceps purpurea* [Fr.] Tul.). Genet. Šlecht., **32**: 161–168.
- CAGAŠ B. (2001): Harmful and beneficial microorganisms in grasses. In: VOLKER H.P., DAPPRICH P.D. (eds): Proc. 3rd Int. Conf. Harmful and Beneficial Microorganisms in Grassland, Pastures and Turf. Soest (Germany), 26 September 2000: 7–16.
- CAGAŠ B., HORN G. (1994): Possibilities of breeding *Poa pratensis* L. for resistance to ergot (*Claviceps purpurea* [Fr.] Tul.). In: KROHN K., VOLKER H. P., THOMAS J. (eds): Proc.

- Int. Conf. Harmful and Beneficial Microorganisms in Grassland, Pastures and Turf. 17: 251–258.
- HORN G. (1989): Untersuchungen zur Wärmebehandlung und zur epidemiologischen Bedeutung von Mutterkornsklerotien in Wiesenrispesaatgut zum Vermehrungsanbau. (Report – unpublished). VEB Saat- und Pflanzgut Landwirtschaftliche Kulturpflanzenarten, PSF 532, Halle (Saale), Germany.
- CHASTAIN T.G. (1992): Relationship of ergot to Kentucky bluegrass seed production and quality. J. Appl. Seed Product., **10**: 7–10.
- JOHNSTON W.J., GOLOB C.T., SITTON J.W. (1995): Control of ergot in Kentucky bluegrass seed fields using SDI fungicides and a surfactant. In: Proc. 3rd Int. Herbage Seed Conf. Yield and Quality in Herbage Seed Production. June 18–23, Halle (Saale), Germany: 309–311.
- MÜHLE E., FRAUENSTEIN K., SCHUMANN K., WETZEL T. (1971): Krankheiten und Schädlinge der Futtergräser. S. Hirzel Verlag, Leipzig.
- PAŽOUTOVÁ S., OLŠOVSKÁ J., LINKA M., KOLÍNSKÁ R., FLIEGER M. (2000): *Claviceps purpurea* populations: biotope, specificity and chemoraces. Appl. Envir. Microbiol., **66**: 5419–5425.
- SITTON J.W., CAGAŠ B., JOHNSTON W.J., GOLOB C.T., KLIMEŠOVA A., JOHNSON R.C. (2001): Identification of ergot pathogenicity and resistance in Kentucky Bluegrass accessions in the U.S.A. and Czech Republic. Plant Disease (in print).

Received for publication November 10, 2000

Accepted after corrections January 18, 2002

Souhrn

CAGAŠ B., MACHÁČ R. (2002): **Rozdílná patogenita izolátů námele (*Claviceps purpurea* [Fr.] Tul.) z lipnice luční (*Poa pratensis* L.)**. Plant Protect. Sci., **38**: 18–22.

Námel, vyvolaný houbou *Claviceps purpurea* (Fr.) Tul., patří k nejzávažnějším problémům v semenářství lipnice luční. Množství sklerocií námele v osivu závisí na mnoha faktorech, především na klimatických poměrech ve vegetačním období, stáří porostu, skladovacích podmínkách a posklizňovém ošetření osiva. Důležitou roli hraje i stupeň odolnosti pěstované odrůdy. Na základě tříletých skleníkových pokusů se čtyřmi různými izoláty námele (Zubří, ČR; Leutewitz a Mnichov, SNR a Pullman, Washington, USA) bylo zjištěno, že i patogenita jednotlivých populací může hrát významnou úlohu. Byly zjištěny významné rozdíly v množství sklerocií tvořených po inokulaci izoláty ze střední Evropy (Leutewitz, Zubří – nízká tvorba sklerocií) a americkým izolátem (vysoká tvorba sklerocií). Stupeň patogenity má tedy výrazný dopad na výskyt sklerocií námele u lipnice luční pěstované na semeno.

Klíčová slova: lipnice luční; *Claviceps purpurea* (Fr.) Tul.; izoláty; patogenita

Corresponding author:

Ing. BOHUMÍR CAGAŠ, CSc., OSEVA PRO, s.r.o., Výzkumná stanice travinářská Rožnov-Zubří, Hamerská 698,
756 54 Zubří, Česká republika
tel.: + 420 651 65 82 94, fax: + 420 651 65 81 97, e-mail: cagas@iol.cz
