PATHOGENICITY OF *Drechslera avenae* FOR LEAVES OF SELECTED OAT GENOTYPES AND IST ABILITY TO PRODUCE ANTHRAQUINONE COMPOUNDS

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Abstract. *Drechslera* leaf spot in the common oat (*Avena sativa* L.) is caused by *D. avenae* (syn. *Pyrenophora chaetomioides*, *Helminthosporium avenae*). Susceptibility of various oat cultivars and breeding lines to leaf infection by this pathogen was investigated in the field in 2001–2002 and in a growth chamber. After natural infection in the field, brown or brown-red spots caused by *D. avenae* were found in all oat genotypes and both years, but were less common in the drier and colder growing season (2001). Mean values of leaf infection index in the studied oat genotypes for the 2 years varied from 5.65 (CHD 2100) to 10.33 (Akt). After artificial inoculation in the growth chamber, symptoms were similar but leaf infection index was much higher – from 15.0 (Bajka) to 41.3 (STH 4699). High-performance liquid chromatography showed that anthraquinone derivatives produced by strain 1 of *D. avenae* include anthraquinone compaunds – cynodontin and helminthosporin. These compounds are known to limit the growth of some other pathogens and saprophytes.

Key words: *Poaceae*, leaf spot, cynodontin, helminthosporin

INTRODUCTION

Drechslera avenae (Eidam) Scharif – also known as *Pyrenophora chaetomioides* Speg. or *Helminthosporium avenae* Eidam – is a specialized pathogen infecting various species of oats (*Avena* spp.) and some grasses [Frank and Christ 1988, Obst 1995, Paul 1995, Mehta 1999, Prończuk 2000, Carmona et al. 2004]. To a large extent, it damages oat leaves and seedlings in the USA [Frank and Christ 1988, Hagan 2007]. In the late

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1990s, *D. avenae* proved to be the most frequent pathogen of oat leaves in Canada [Fernandes et al. 1999 after Clear et al. 2000]. *Drechslera* leaf spot – also known as *Helminthosporium* leaf spot – in the areas where oat is grown in Brazil, is considered to be the most quickly spreading disease of this crop plant [Mehta 2001]. In European oat fields it has been reported from former Yugoslavia [Arsenijević et al. 1996] and Germany [Müller 1963 after Obst 1995].

Drechslera avenae causes spots on leaves of oat seedlings and older plants [Frank and Christ 1988, Obst 1995, Mehta 2001]. If the infection is severe, oat seedlings may die [Müller 1963 after Obst 1995].

This pathogen is able to produce secondary metabolites, e.g. pyrenophorol, pyrenophorin, and dihydropyrenophorin [Sugawara and Strobel 1986, Kastanias and Chrysayi-Tokousbalides 1999, 2000]. *D. avenae* also produces KM-01, which is similar to bipolaroxin, produced by the fungus *Bipolaris cynodontis,* which is the first described selective inhibitor of brassinolide – a steroid controlling plant growth – coming from a natural source [Kim et al. 1995, 1998].

Other secondary metabolites produced by *D. avenae* include anthraquinone derivatives [Engström et al. 1993]. Their biosynthesis involves condensation of acetyl-coenzyme A and malonyl-coenzyme A [Kohlmüzner 1998]. These compounds are produced not only by fungi but also by lichens, bryophytes, pteridophytes, gymnosperms, and angiosperms [Muzychkina 1998]. Anthraquinone derivatives are effective inhibitors of protein kinases [Fredenhagen 1995], so they may indirectly affect the processes of proliferation and differentiation, controlled by these enzymes [Chen 1987, Nishizuka 1988], and – at early stages of pathogenesis – the transduction of immunological signals [Ligterink et al. 1997]. These metabolites inhibit bacterial and fungal growth but may be also autotoxic, controlling the growth of the plant itself [Tsurushima et al. 1984 after Engström et al. 1993, Engström et al. 1993].

In the literature there is little information on the pathogenicity of *D. avenae* towards oats and on its ability to produce anthraquinone derivatives. This inspired us to investigate the susceptibility of oat genotypes to leaf infection by this pathogen and to identify anthraquinone compounds produced by *D. avenae* strain 1, which proved to be the most pathogenic in an earlier laboratory test*.

MATERIAL AND METHODS

Field observations of *Drechslera* leaf spot on the common oat (*Avena sativa* L.) in natural conditions were made in 2001–2002 at the Experimental Station for Cultivar Evaluation at Uhnin in the north-eastern part of the Lublin region (East Poland 51.639ºN, 22.900ºE). The plots were established on lessivé soil developed from loess overlying light clay. Each year, root crops were cultivated before oats on the study plots. The experiment involved 9 oat genotypes (tab. 1). Seeds of each studied cultivars and breeding lines in both study years were pretreated with carboxin and thiram, and sown on 4 plots of 10 m^2 each, in densities recommended for agricultural practice. We applied an N-P-K fertilizer (85: 60: 90 kg · ha^{-1}) and harrowing before seedling emergence and when coleoptiles with hidden leaves appeared on the surface. Harrowing was

aimed to destroy weeds and the soil crust. Besides, a herbicide was applied when the plants had 5–6 leaves (Granstar, 20 kg · ha⁻¹).

Table 1. Values of leaf infection index of *Drechslera avenae* in selected oat genotypes grown on fields of the Experimental Station for Cultivar Evaluation at Uhnin in 2001–2002

Tabela 1. Wartości indeksu porażenia liści przez *Drechslera avenae* różnych genotypów owsa wzrastającego na polach Stacji Doświadczalnej Oceny Odmian w Uhninie w latach 2001–2002

Oat genotypes Genotypy owsa	Leaf infection index – Indeks porażenia liści		
	2001	2002	mean – średnio
Akt	8.60 [a]	12.06 [c]	10.33 [b]
Bajka	6.50 [a]	8.19 [ab]	7.35 [ab]
Borowiak	7.50 [a]	11.94 [c]	9.72 [b]
Jawor	5.10 [a]	9.00 [bc]	7.05 [ab]
Polar	7.10 [a]	7.14 [ab]	7.12 [ab]
CHD 2100	4.80 [a]	6.50 [a]	5.65 [a]
STH 4699	6.90 [a]	12.94 [c]	9.92 [b]
STH 5300	7.20 [a]	7.81 [ab]	7.50 [ab]
STH 5400	7.70 [a]	8.94 [b]	8.32 [ab]

Values marked with the same letters within columns do not differ significantly at $p \leq 0.05$
Średnie wartości w kolumnach oznaczone tą samą literą nie różnią się istotnie przy $p \leq 0,05$

The level of infection was assessed at the late milk stage, i.e. 77 on the Tottman scale [Tottman 1987]. In each year of the study, 50 flag leaves were collected for analysis from 4 experimental fields, i.e. in total 200 leaves for each oat cultivar and breeding line. In the laboratory, leaf infection rate was assessed for individual leaves, on a scale of 0–8 [Łacicowa et al. 1991], where 0 = no symptoms on leaves, while 8 = infection of over 80% of leaf area. Spotted leaf fragments were kept in moisture chambers to identify the cause of the infection, on the basis of microscopic analysis of spores of *D. avenae.* Values of leaf infection index for each replication were calculated according to McKinney's formula [Łacicowa 1969]:

$$\text{Infection index} = \frac{\sum (a \times b)}{n \times c} \times 100$$

where:
    a – infection rate,
    b – number of leaves with this infection rate,
    n – total number of leaves,
    c – highest infection rate.

The mean infection index for each oat genotype in individual years and for all 3 years were calculated. The data were subjected to statistical analysis, using Tukey confidence half-intervals [Żuk 1989].

Information on weather conditions in the study area is based on data from the weather station in Uhnin.

Susceptibility of oat genotypes to leaf infection was tested in a growth chamber. *Drechslera avenae* strain 1 was used in this study, as it proved to be the most pathogenic in an earlier laboratory test according to the method of Mishra and Behr [1976] (tab. 2).

Table 2. Effect of *Drechslera avenae* on oat seed germination (cultivar Sławko) in Petri dishes
Tabela 2. Wpływ *Drechslera avenae* na kiełkowanie ziarna owsa odmiany Sławko w doświadczeniu szalkowym

Drechslera avenae strain Szczep *Drechslera avenae*	Strain origin Pochodzenie szczepu	Germination rate Zdolność kiełkowania, %
1*	oat seeds – ziarniaki owsa	6
2	oat seedlings – siewki owsa	7
15	oat seeds– ziarniaki owsa	12
Control – Kontrola		61

*Strain selected for artificial inoculation of leaves
*Szczep wytypowany do sztucznego zakażania liści

The infection experiment was made in a growth chamber at 23–24°C and relative humidity of 85%. The oat seedlings of selected genotypes were kept under fluorescent light (illuminance 18000 lx). The plants were grown in plastic pots (10 cm in diameter) filled with garden soil (with composted manure) and sand mixed at a ratio of 2:1 (pH 6.5), after double sterilization in an autoclave for 2 h at 121°C.

For this study seeds of 12 oat genotypes (tab. 3), with no spots and mechanical damage on the seed coat were selected. Next they were surface-sterilized for 1 min in 10% sodium hypochlorite (NaClO), and then washed 3 times in sterile distilled water and germinated for 5 days in a growth chamber on sterile Petri dishes lined with blotting paper. In the experiment, only the oat seeds whose sprouts reached 10 mm in length and were well-developed were used. In each pot, germinated seeds of 10 oat genotypes were sown. For each genotype, 4 replications were used in each experimental combination.

Pots with germinated seeds were kept in a growth chamber until the plants developed 5 leaves each. The inoculum of *D. avenae* strain 1 was prepared from single-spore cultures growth on potato dextrose agar (PDA, from Difco, $39 \text{ g} \times l^{-1}$) in a termostate at 24°C for 10 days. After this time, the cultures were washed with 10 ml of distilled water, and the mycelium with conidia was scraped off with a glass spatula from the colony surface. Next the suspension was filtered through a dense cloth, to separate fragments of fungal mycelium from the filtrate and conidia, into glass flasks containing 0.1% aqueous agar with $0.05 \text{ ml} \times l^{-1}$ of surfactant (added with the use of automatic pipette VE-100). Density of the spore suspension was adjusted to 25×10^4 spores $\times \text{ml}^{-1}$. Leaves of each plant were sprayed with 5 ml of the suspension (1 ml per leaf), containing spores of *D. avenae*, by using a spray bottle. To prevent evaporation of the suspen-

sion, the pots were kept in polyethylene moisture chambers for 48 h. Symptoms of the disease, i.e. necrotic spots on leaves, were recorded after 7 days of incubation. The infection rate of the third leaves of each of 10 plants were assessed on the basis of a graphic scale of 0–8 [Łacicowa et al. 1991]. Next, the leaf infection index was calculated for individual populations, and then the mean leaf infection index. The data were subjected to statistical analysis, using Tukey confidence half-intervals [Żuk 1989].

Table 3. Mean values of leaf infection index of selected oat genotypes inoculated with *Drechslera avenae* strain 1, as compared to the control, in a growth chamber

Tabela 3. Średnie wartości indeksu porażenia liści wybranych genotypów owsa w kombinacji z *Drechslera avenae* nr 1 w porównaniu z kontrolą w warunkach doświadczenia fitotronowego

Oat genotypem Genotypy owsa	Experimental wariant Kombinacja doświadczenia	
	Drechslera avenae	control – kontrola
Akt	35.0 [bc*]	3.5
Bachmat	40.0 [c*]	5.6
Bajka	15.0 [a*]	0
Bohun	22.5 [b*]	0.1
Borowiak	27.5 [b*]	0.3
Cwał	23.8 [b*]	0
Jawor	27.5 [b*]	0
Polar	32.5 [bc*]	0.4
CHD 2099	30.0 [bc*]	1.0
STH 4298	35.0 [bc*]	0.5
STH 4599	33.8 [bc*]	0.2
STH 4699	41.3 [c*]	0.9

Values marked with the same letters within columns do not differ significantly at $p \leq 0.05$
Średnie wartości w kolumnach oznaczone tą samą literą nie różnią się istotnie przy $p \leq 0,05$
*Means differ significantly from the control at $p \leq 0.05$
*Średnie różnią się istotnie w porównaniu z kontrolą przy $p \leq 0,05$

The ability of *Drechslera avenae* strain 1 to produce anthraquinone pigments was assessed as previously described for *D. tritici-repentis* [Kachlicki and Wakuliński 2002] with some modifications. Briefly, the pigment biosynthesis was conducted on the rice medium for 14 days and thereafter the medium was subjected to extraction with chloroform. The metabolite solution was filtered, evaporated in a vacuum evaporator at 35ºC and the residue was dissolved in methanol. The methanol solution was diluted with water (1:4, v/v) and metabolites were purified using solid phase extraction (SPE) on C_{18} columns. The analysis was performed using Beckman Gold HPLC System with a diode array detector and Alltima C_{18} columns. Individual compounds were identified by comparison of the retention times and UV spectra with those obtained for previously purified compounds [Kachlicki and Wakuliński 2002].

RESULTS

Field observations made at the late milk stage in 2001–2002, revealed *Drechslera* leaf spot caused by *D. avenae* in all the studied oat genotypes and in both growing seasons. However, differences were noticed in susceptibility of the oat cultivars and breeding lines to leaf infection by this pathogen (tab. 1).

On infected leaf blades, both in the central part and at the edges, round or slightly elongated spots were formed (up to 7 mm long). Most of the spots were 2 mm wide and 3–5 mm long, brown-red with yellow-orange halo. In laboratory conditions, on the surface of such spots, we observed sporulation of *D. avenae*.

In 2001, values of leaf infection index of the analysed genotypes varied from 4.80 (CHD 2100) to 8.60 (Akt) and did not differ significantly, while in 2002, from 6.50 (CHD 2100) to 12.94 (STH 4699) and differed significantly (tab. 1). Mean values of leaf infection index in the studied oat cultivars and breeding lines for the 2 years varied from 5.65 to 10.33. Significantly higher values of leaf infection index after 2 years of observations were noted for 3 genotypes: Akt, Borowiak, and STH 4699 (tab. 1).

After artificial inoculation of leaves with *D. avenae*, we observed on those organs elliptic, elongated, brown or brown-red spots, with a lighter centre and yellow-orange halo, 1–2 mm wide and 3–5 mm long. We noticed also narrow necroses, several centimetres long, sometimes fused, accompanied by chlorosis of surrounding tissues. In some cases, leaf blades were narrowed in the places with spots. Moreover, leaf tips sometimes were dry.

Among the 12 oat genotypes, Bachmat appeared to be most sensitive, as its infection index reached 40.0. In the laboratory experiment, the lowest leaf infection index (15.0) was recorded for cultivar Bajka, which differed significantly from the other genotypes (tab. 3). In all the studied oat genotypes, values of leaf infection index of inoculated plants differed significantly from the control (tab. 3).

Table 4. Temperature and precipitation at Uhnin in growing seasons 2001–2002
Tabela 4. Temperatura i opady w Uhninie w okresach wegetacji owsa 2001–2002

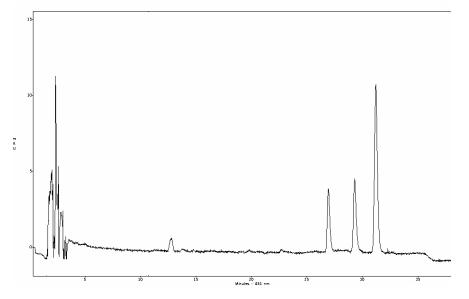| Month Miesiąc	Long-term mean Średnie wieloletnie (1881–1980)		Deviation of air temperature Odchylenia temperatury powietrza		% of long-term mean precipitation Procent normy opadów	
	temperature temperature, °C	precipitation opady, mm	2001	2002	2001	2002
April Kwiecień	7.4	36	+1.2	+1.0	140.6	57.2
May Maj	13.4	50	+0.4	+3.4	52.4	53.8
June Czerwiec	16.4	66	-1.4	+1.0	55.5	141.5
July Lipiec	18.1	68	+3.2	+3.1	19.7	100.7
August Sierpień	17.3	68	+1.2	+2.0	92.5	25.6

Fig. 1. HPLC chromatogram of anthraquinone pigments of *Drechslera avenae* strain 1
Rys. 1. Analiza HPLC pigmentów antrachinonowych szczepu *Drechslera avenae* nr 1

At the Experimental Station, air temperature in June 2001 was 1.4°C lower than the long-term mean, while mean temperature in April, May, July and August was 0.4–3.2°C higher than the long-term means. May, June and July were dry, as precipitation reached then 52.4, 55.5 and 19.7% of the long-term means, respectively. In August, precipitation was close to the long-term mean. By contrast, in April, precipitation was 40.6% higher than the long-term mean (tab. 4). The growing season of 2002 was characterized by higher temperature than the long-term means: from +1°C in April and June to +3.4°C in May. In June and July, precipitation was 41.5% and 0.7% higher than the long-term means, respectively. By contrast, in April, May, and August, precipitation reached 57.2%, 53.8%, and 25.6% of long-term means, respectively (tab. 4).

The performed HPLC analysis and the UV/VIS spectra over the range 210–600 nm indicate that low-molecular metabolites of *D. avenae* strain 1 include anthraquinone derivatives. In the anthraquinone mixture of the studied strain, three compounds were found. Their chromatogram is presented in figure 1.

　　– compaund 1
　　　　$RT_{HPLC} = 27.8$
　　　　$UV/VIS_{HPLC}\lambda_{max} = 230, 254, 288, 480$
　　– compaund 2
　　　　$RT_{HPLC} = 29.8$
　　　　$UV/VIS_{HPLC}\lambda_{max} = 232, 250, 288, 485$
　　– compaund 3
　　　　$RT_{HPLC} = 31.7$
　　　　$UV/VIS_{HPLC}\lambda_{max} = 244, 288, 512, 550$

a)

b)

cynodontin – cynodontyna
1, 4, 5, 8-tetrahydroxy-3-methyl-9,10-anthraquinon

helminthosporin – helmintosporyna
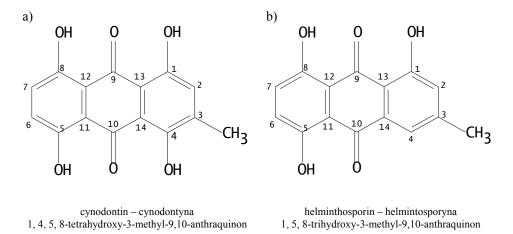1, 5, 8-trihydroxy-3-methyl-9,10-anthraquinon

Fig. 2. Chemical formulae of anthraquinone pigments of *Drechslera avenae* strain 1
Rys. 2. Wzory chemiczne zidentyfikowanych pigmentów antrachinonowych *Drechslera avenae* nr 1

Spectra of the second and third compound correspond with characteristics of helminthosporin (1, 5, 8-trihydroxy-3-methyl-9, 10-anthraquinon) and cynodontin (1, 4, 5, 8-tetrahydroxy-3-methyl-9,10-anthraquinon) (fig. 2a, b).

DISCUSSION

Results of the study show that *Drechslera* leaf spot on cultivated oats in the Lublin region (South-East Poland) appeared in both study years (2001–2002). Values of leaf infection index indicate that symptoms of the disease were not severe.

In various environmental conditions *D. avenae* is considered to be the cause of *Drechslera* leaf spot on oats and some grass species in Europe [Obst 1995, Paul 1995, Prończuk 2000], in both Americas [Earhart and Shands 1952, Frank and Christ 1988, Clear et al. 2000, Mehta 2001] and in Africa [Scott 1995]. Severity of the symptoms is determined by weather conditions. Temperature from 20 to 24ºC and high humidity facilitate leaf infection by *D. avenae* [Sprague 1950 after Scott 1995, Shaner 1981]. In this study, similar conditions were observed in the growing season of 2002. This seems to be the reason of the higher values of leaf infection index in that year than in 2001.

The results confirm earlier reports about brown-red spots with orange-red edges on leaves infected by *D. avenae* and about sporulation of this fungus at the infection sites [Müller 1963, 1964b after Obst 1995]. This type of symptoms of the disease was classified by Müller [1963, 1964b after Obst 1995] as type II of *Drechslera* leaf spot.

To assess the susceptibility of plant genotypes to pathogens, various methods are applied. The choice depends on the amount of tested plant material and on experimental conditions [Earhart and Shands 1952, Frank and Christ 1988, Almgren et al. 1999, Kiecana and Cegiełko 2007, Cegiełko 2008].

Considering the variable virulence of strains within the genus *Drechslera* [Buchannon and McDonald 1965, Makela 1977 after Gacek 1979, Arabi et al. 1992], we used in the laboratory experiment a strain of *D. avenae* No. 1 whose pathogenicity was earlier checked by the method of Mishra and Behr [1976].

The major criterion of leaf infection assessment in oat cultivars and breeding lines was the leaf infection index. Spots on leaves of oat seedlings in the laboratory experiment, due to artificial inoculation, were smaller but similar to the symptoms observed on those organs in field conditions at the late milk stage. However, the spots covered in some genotypes a larger proportion of the leaf, reflected by higher values of leaf infection index.

Differences in reactions of oat genotypes to leaf infection both in the field and in the laboratory, were observed also by Earhart and Shands [1952]. In their research, the analysed oat cultivars differed in spot size and rate of dieback of leaf tissues. According to those authors, the cultivars that mature later are less sensitive to infection in field conditions than in a greenhouse experiment, while early cultivars were more sensitive in field conditions than those grown in a greenhouse.

As a result of this study, cultivar Bajka appeared to be the least sensitive to leaf infection by *D. avenae,* while cultivar Akt and breeding line STH 4699 were the most sensitive.

The infection of leaves by *D. avenae* is facilitated by the hydrolysis of monodesmoside forms of avenacosides A and B, present in leaves. Enzymes of this pathogen hydrolyse those compounds to the aglycone nuatigenin, which precipitates and does not inhibit fungal growth [Lüning and Schlösser 1976, Oleszek 2001]. The pathogenicity of *D. avenae* towards oat leaves depends on the plant's ability to synthesise dihydropyrenophorin [Sugawara and Strobel 1986].

The anthraquinones helminthosporin and cynodontin, produced by *D. avenae* strain 1, are compounds of similar structure, commonly produced by various fungal species [Engström et al. 1993, Kachlicki and Wakuliński 2002]. In the cultures of *D. avenae* the co-occurrence of helminthosporin and cynodontin reflects the frequently reported rule that anthraquinones occur as a mixture of compounds. For example, in the cultures of *D. tritici-repentis* (teleomorph of *Pyrenophora tritici-repentis*) nine anthraquinone compounds were found [Kachlicki and Wakuliński 2002]. Among pathogens of the genus *Drechslera*, the ability to synthesize anthraquinones was observed, e.g., in *D. teres* (Sacc.) Shoem., *D. graminea* (Rabenh. ex Schlecht.) Shoem., *D. tritici-repentis* (Died.) Shoem., and *D. dictyoides* (Drechsler) Shoem. [Engström et al. 1993, Wakuliński 2004].

The presence of chromophore in anthraquinone molecules determines their ability to bind with the purine and pyrimidine bases of the DNA chain by way of intercalation. This stabilizes the structure of the acid, as it does not allow its decoiling, and thus replication. This characteristic explains the antibiotic activity of this group of compounds in many Gram-positive bacteria [Bucklew et al. 1972, Anke et al. 1980a, b, Engström et al. 1993, Wakuliński 2004]. In natural conditions, anthraquinones can help to limit population growth of some pathogens and saprophytes that colonize various plant organs and compete with the species producing these compounds.

CONCLUSIONS

1. Among the tested oat genotypes, none was completely resistant to leaf infection by *Drechslera avenae.*

2. On the basis of germination rate of oat seed in Petri dishes the diversity among *D. avenae* isolates was proved.

3. Strain *Drechslera avenae* no 1 with the highest pathogenicity to oats showed the ability to produce anthraquinone derivatives – helminthosporin and cynodontin.

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PATOGENICZNOŚĆ *Drechslera avenae* DLA LIŚCI WYBRANYCH GENOTYPÓW OWSA I JEGO ZDOLNOŚĆ DO PRODUKCJI ZWIĄZKÓW ANTRACHINONOWYCH

Streszczenie. Plamistość liści owsa (*Avena sativa* L.) powodowana jest przez gatunek *Drechslera avenae* (*Drechslera chaetomioides, Helmintosporium avenae*). Podatność wybranych odmian i rodów hodowlanych owsa na infekcję przez ten patogen badano w warunkach polowych w latach 2001–2002 oraz w fitotronie. W wyniku naturalnej infekcji w warunkach polowych, brązowe lub brązowo-czerwone plamy, powodowane przez *D. avenae* stwierdzono w przypadku wszystkich genotypów owsa w obu latach badań, ale występowały one w mniejszym nasileniu w sezonie bardziej suchym i chłodniejszym (2001). Średnie wartości indeksu porażenia liści w przypadku badanych genotypów owsa wynosiły od 5.65 (CHD 2100) do 10.33 (Akt). W wyniku sztucznej inokulacji liści owsa w warunkach fitotronowych objawy na liściach były podobne, ale indeks porażenia liści znacznie wyższy – od 15.0 (Bajka) do 41.3 (STH 4699). Badania przy zastosowaniu wysokorozdzielczej chromatografii cieczowej wykazały zdolność do produkcji przez szczep *D. avenae* nr 1 pochodnych antrachinonowych – cynodontyny i helmintosporyny. Związki te mogą ograniczać wzrost niektórych patogenów i saprotrofów.

Słowa kluczowe: *Poaceae*, plamistość liści, cynodontyny, helmintosporyny