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THE VIRULENCE OF *HETEROBASIDION PARVIPORUM* POPULATION FROM NORWAY SPRUCE STAND IN SUWAŁKI FOREST DISTRICT

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Abstract. The aim of this study was to determine the virulence of *H. parviporum* genets with known genetic similarity and representing pathogen's population, which infested a part of Norway spruce stand. Genets caused average 47% mortality of spruce seedlings in infection experiment *in vitro.* The mortality rate varied from 16% to 80%. In infection experiment *in vivo* the mycelium of *H. parviporum* caused the wood necrosis on distance from 0.8 to 7.78 mm. 56% of genets of investigating population characterized by high virulence, 22% by average and 22% by low virulence.

Key words: *Heterobasidion parviporum*, genets, virulence, Norway spruce

INTRODUCTION

Heterobasidion root and butt rot disease is one of the most important forest trees diseases in Poland and boreal zone. *Heterobasidion* spp. cause white pocket root and butt rot. The rot column could spread several meters in stem height [Korhonen et al. 1998, Sierota 2001, Mańka 2005]. In Poland occur three *Heterobasidion* species – *H. annosum* (Fr.) Bref. sensu stricto, *H. parviporum* Niemelä et Korhonen i *H. abietinum* Niemelä et Korhonen [Łakomy et al. 2000, Łakomy and Werner 2003]. The occurrence of these species cover the range of its main plant hosts – *Pinus sylvestris* L. for *H. annosum* s. str., *Picea abies* (L.) H. Karst. for *H. parviporum* and *Abies alba* Mill. for *H. abietinum* [Łakomy and Werner 2003]. The host list of *Heterobasidion* is very wide and includes more than 200 species of trees, shrubs and heathers [Webb and Alexander 1985]. *Heterobasidion annosum* s. str. attacks the largest number of host species. By contrast *H. parviporum* is a narrow specialized species, which occurs mainly on Norway spruce and *H. abietinum* on European fir, rarely attacks larch (*Larix decidua* Mill.) [Wagn 1980, Łakomy and Werner 2003, Łakomy and Cieślak 2008].

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Different inoculation method were used to characterize the virulence of pathogens and resistance of its hosts. Including the roots inoculations, in almost all experiments the mycelium was inserted into the wound. The ability of mycelium growth in alive wood and appearance of wood necrosis were the criteria for detection of pathogens' aggressiveness [Dimitri 1969 a, b, Delatour 1982, Stenlid and Swedjemark 1988, Werner and Łakomy 2002 b]. Werner and Łakomy [2002 a] found that seedlings could be used *in vitro* experiments for determination of *Heterobasidion* spp. virulence. Previously, there were used *Heterobasidion* spp. isolates that belonged to different populations in studies concerning its virulence [Werner 1987, 1991, Stenlid and Swedjemark 1988, Werner and Łakomy 2002 a, b]. Łakomy et al. [2007] displayed the results of population studies of *Heterobasidion* spp. virulence in Poland.

The aim of this study was to describe the virulence of *H. parviporum* isolates representing different genotypes of determined relationships and belonged to the population that existed in a part of Norway spruce stand.

MATERIAL AND METHODS

Nine isolates were used in this experiment, which represented different genotypes of *H. parviporum* population, and which colonized stumps in Norway spruces stand in Suwałki Forest District (110-year-old, 54°34'N; 23°01'E). The genetic relationships of these genotype were described in previous study (Table 1) [Łakomy et al. 2011].

Table 1. Genetic relationship (%) of *H. parviporum* population [Łakomy et al. 2011]
Tabela 1. Podobieństwo genetyczne (%) populacji *H. parviporum* [Łakomy i in. 2011]

Genotypes Genotyp	8/3	8/4	9/1	10/1	11/1	12/1	13/2	14/1
8/1	36	68	36	45	45	45	0	45
8/3		36	62	36	36	36	0	36
8/4			36	45	45	45	0	45
9/1				36	36	36	0	36
10/1					68	68	0	56
11/1						56	0	56
12/1							0	36
13/2								0

Infection experiment *in vitro*. Norway spruce seedlings were growing in pure cultures on the medium containing macro- and microelements [Ingestand 1979]. After a month the seedlings were infected by putting the mycelium close to their root collar. Observations of seedlings growth, appearance of disease symptoms, seedlings mortality were started after inoculation. Each combination was duplicated 20 times. After 9 months the genotypes virulence was determined on the base of seedlings mortality [Werner and Łakomy 2002 a].

Infection experiment *in vivo*. 3-year-old Norway spruce seedlings were used in this study. The seedlings were planted in pots three months before the inoculation. Pots were put in an open area and watered as desired. The inoculum consisted of a small piece of wood (5 mm long and 2 mm in diameter) colonized by pathogen mycelium. The inoculum was inserted in the hall made by drill just under the root collar. Next wounds were banded by laboratory film. Each combination was duplicated 10 times. For control Norway spruce seedlings were inoculated with sterile wood pieces. After four months the seedlings were examined on the presence of necrosis caused by pathogen's colonization and rot process [Werner and Łakomy 2002 b].

The virulence of genotypes was calculated on the base of the ability of alive wood colonization, causing the wood necrosis and seedlings mortality.

The analysis of variance (ANOVA/MANOVA) and HSD Tukey test were done (Statistica v. 6).

RESULTS

Nine months after inoculation, genotypes caused mortality of Norway spruce seedlings on average 47%. The isolate 13/2 was characterized by the highest mortality. This isolate caused 80% of seedlings mortality. For other genets the mortality varied from 16% to 65% (Fig. 1). The analysis of variance showed the significant differences in mortality caused by different genotypes of *H. parviporum* (Table 2). In 42% cases genotypes differ in virulence and caused seedlings mortality (Table 3). Three genets 8/1, 13/2 and 14/1 differed in comparison to other in most cases pairings, and genets 8/1 and 14/1 caused the lowest mortality (appropriately 20% and 16%). Average genetic similarity of genets, which caused the significant different mortality was 22%, but on the other hand in 45% these differences were connected with unrelated genotypes.

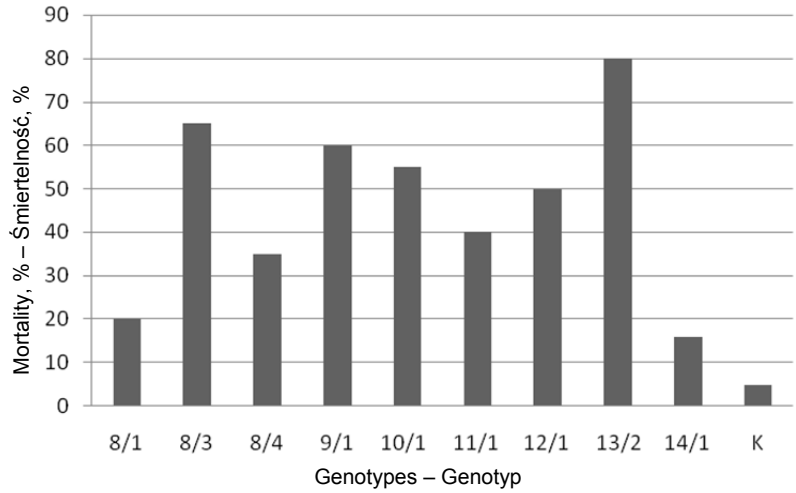

Fig. 1. Mortality of Norway spruce seedlings caused by *H. parviporum* genotypes

Rys. 1. Śmiertelność siewek świerka pospolitego powodowana przez genotypy *H. parviporum*

Table 2. Analysis of variance of Norway spruce seedlings mortality caused by *H. parviporum* genotypes
Tabela 2. Analiza wariancji dla śmiertelności siewek świerka pospolitego powodowanej przez genotypy *H. parviporum*

Source of variance Źródło wariancji	SS	df	MS	F	p	Test F
Among groups Pomiędzy grupami	20.86308	9	2.31812	3.883903	0.000153	1.929689
Inside groups Wewnątrz grup	112.8053	189	0.596853			
Sum Razem	133.6683	198				

Table 3. Results of HSD Tukey test for comparison of Norway spruce seedlings mortality caused by *H. parviporum* genotypes
Tabela 3. Wyniki testu HSD Tukeya dla porównania śmiertelności siewek świerka pospolitego powodowanej przez genotypy *H. parviporum*

Genotypes Genotyp	8/3	8/4	9/1	10/1	11/1	12/1	13/2	14/1	Control Kontrola
8/1	0.01309	0.21313	0.02481	0.029673	0.15979	0.08038	0.00051	0.44357	0.02463
8/3		0.07406	0.35608	0.360826	0.10153	0.17777	0.17338	0.00796	0.00012
8/4			0.12702	0.13588	0.42435	0.28164	0.00696	0.15884	0.00791
9/1				0.5	0.16960	0.28008	0.08481	0.01523	0.00016
10/1					0.17864	0.28757	0.09348	0.01886	0.00035
11/1						0.34940	0.01102	0.11510	0.00423
12/1							0.02551	0.05385	0.00102
13/2								0.00026	9.01E-07
14/1									0.07898

Four months after the inoculation the mycelium of *H. parviporum* spread in wood of Norway spruce stems on average distance of 7.78 mm. The weakness mycelium development in the wood was observed for genotype 14/1 (distance 0.8 mm). The mycelium of genet 10/1 overgrew the wood on average distance of 11.8 mm (Fig. 2). The analysis of mycelium spread divided genotype into three groups, which mycelium spread in the wood on different distance (Table 4, $p < 0.05$). Looking into the genetic similarity, differences in genotypes aggressiveness occurred in all similarity groups. Average genetic similarity of isolates differed ($p < 0.05$) in mycelium growth in the Norway spruce wood was 36.5% (Table 5, Fig. 2). In the examined *H. parviporum* population five genotypes were characterized by the high virulence, two average and two low virulence (Fig. 3).

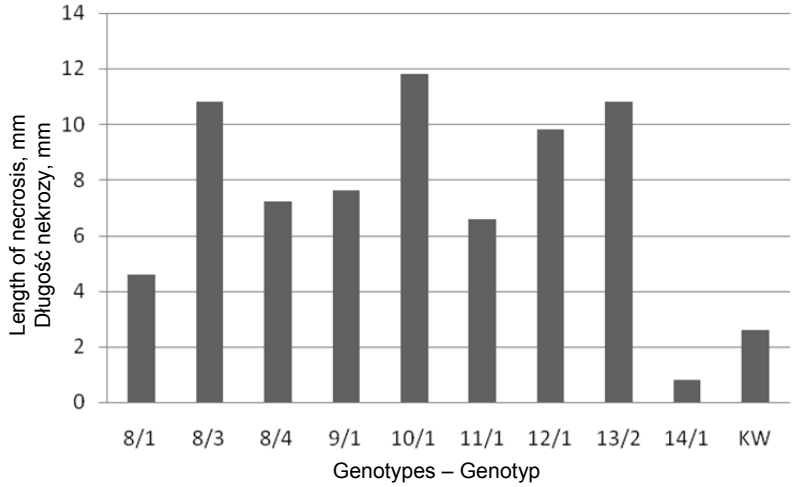

Fig. 2. Average of necrosis length in Norway spruce stems
Rys. 2. Średnia długość nekrozy w strzałkach świerka pospolitego

Table 4. Analysis of variance of necrosis in Norway spruce stems caused by *H. parviporum* genotypes
Tabela 4. Analiza wariancji dla nekrozy strzałek świerka pospolitego powodowanej przez genotypy *H. parviporum*

Source of variance Źródło wariancji	SS	df	MS	F	p	Test F
Among groups Pomiędzy grupami	580.1778	8	72.52222	12.87377	1.57E-08	2.208518
Inside groups Wewnątrz grup	202.8	36	5.633333			
Sum Razem	782.9778	44				

Table 5. Results of HSD Tukey test for comparison of necrosis of Norway spruce stems caused by *H. parviporum* genotypes
Tabela 5. Wyniki testu HSD Tukeya dla porównania nekrozy siewek świerka pospolitego powodowanej przez genotypy *H. parviporum*

Genotypes Genotyp	8/3	8/4	9/1	10/1	11/1	12/1	13/2	14/1	Control Kontrola
1	2	3	4	5	6	7	8	9	10
8/1	0.0077	0.10115	0.08912	0.00641	0.18719	0.0158	0.01097	0.03985	0.14975
8/3		0.012099	0.03393	0.30168	0.01818	0.21364	0.5	5.83E-05	0.00072
8/4			0.38712	0.0175	0.35303	0.01211	0.00237	8.52E-05	0.00096
9/1				0.03123	0.29040	0.06632	0.02127	0.001302	0.00606

Table 5 – cont. / Tabela 5 – cd.

Genotypes Genotyp	8/3	8/4	9/1	10/1	11/1	12/1	13/2	14/1	Control Kontrola
10/1					0.01661	0.13813	0.27996	0.000507	0.00201
11/1						0.03914	0.01426	0.00467	0.02067
12/1							0.09606	1.2E-06	4.58E-05
13/2								4.12E-07	1.78E-07
14/1									0.00834

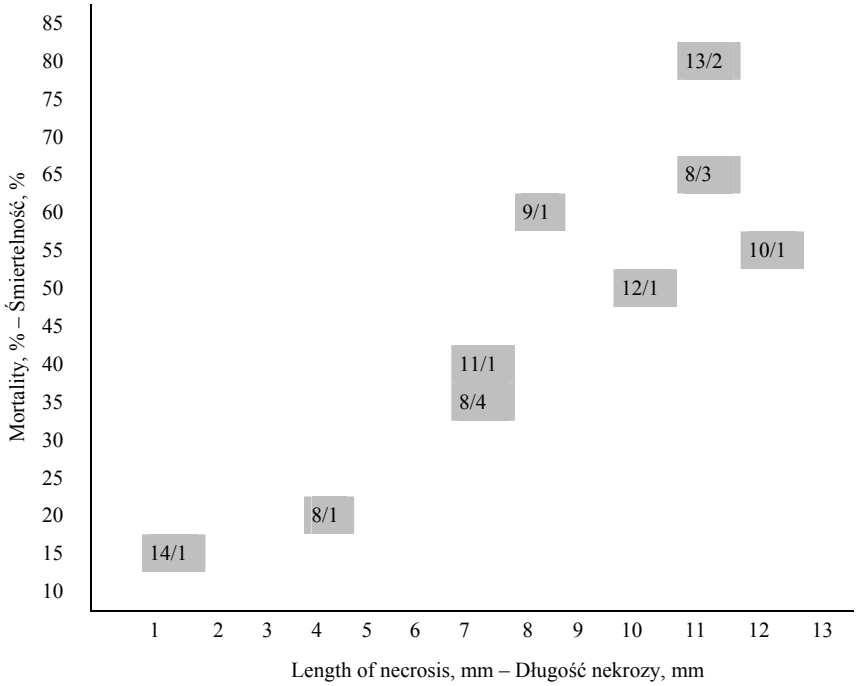

Fig. 3. Diagram of virulence of *H. parviporum* genotypes
Rys. 3. Diagram wirulencji genotypów *H. parviporum*

DISCUSSION

Stands might be colonized by *Heterobasidion* spp. in different degree. This is related to the importance of *Heterobasidion* root rot for stability of infected stands. Stenlid [1985] found that Norway spruce stands were colonized by pathogen in 12% and 17%. Rieger [1995] observed increase of infection from 7.5% to 33% during decade. On the other hand Stanlid and Wästerlund [1986] found infection of 77% of Norway spruces in the stand. Łakomy et al. [2001] analysed the stands destroyed by windstorms and

displayed that from 56% to 100% of the damaged trees had previously been colonized by pathogen. Łakomy et al. [2011] analysed colonization of *H. annosum* s str. and *H. parviporum* in ten stands – five Scots pine and five Norway spruce stands. Scots pine stands were infested in 42% – 100%, whereas infestation of Norway spruce stands was lower and estimated from 28% to 35%. Trees or stumps may be colonized by one or more *Heterobasidion* genets [Stenlid 1985, Piri et al. 1990, Piri 1996, Łakomy et al. 2007, Piri and Korhonen 2007, Dalke and Łakomy 2009]. Bodles et al. [2005] showed, that 45-year-old Sitka spruce stand (*Picea sitchensis* (Bong.) Carrière), that grew without thinning, was severly infected by *H. annosum* s. str. They found 25 pathogen's genotypes, and the biggest one spread on the distance of 22.5 m. At the other hand Dalke and Łakomy [2009] displayed bigger size of genets of *H. annosum* s. str. in Scots pine stand with beech undersorey. The biggest genotype cover area of 84 m^2 and occurred in three stumps and six young beeches. Stenlid and Redfern [1998] found that the biggest *H. annosum* sensu lato genet occupied area of 50 m in diameter. The age of this genet might be estimated to approx. 50 years, because there is assumption that *H. annosum* overgrows dead wood in average 50 cm per year [Rishbeth 1951, 1957, Slaughter and Parmeter 1995, Bendz-Hellgren et al. 1999]. Łakomy et al. [2011] found that the biggest genotype occurred on the area of 160.2 m^2 (about 28 m in diameter) and probable age of this genet was estimated to 28-years. They also showed a high genetic variability of the studied *H. annosum* s. str. populations expressed by low similarity among genotypes. Populations of *H. parviporum* were more similar. In all cases they found genets very closely related in 70-77% and also totally genetically different. Stenlid [1985] found that pathogen genotypes might be big and occupy even 15 trees in Norway spruce stands. However, Piri et al. [1990] estimated genets that colonized on the average, each three tree. In *Abies concolor* (Gordon et Glend) Lindl. ex Hildebr. stand [Garbelotto 1996] the longest distance between colonized by the same genet trees was 6 meters.

Pathogens may be varied in pathogenicity and aggressiveness between each other [Stenlid and Swedjemark 1988, Swedjemark et al. 1999, Werner and Łakomy 2002 b]. The health status of trees and damages caused by decay development in Norway spruce stems depended on virulence of existing pathogen's population in this stand. The aggressiveness and pathogenicity of genets belonging to different populations were tested in several studies [Stenlid and Swedjemark 1988, Swedjemark et al. 1999, Werner and Łakomy 2002 b]. Łakomy et al. [2005] investigating stumps from gap in Norway spruce stand found that 19 genotypes of *H. parviporum* differed in virulence. Forty two percent of genets caused mortality of less than 20% of seedlings *in vitro.* Moreover, 32% of genets were highly pathogenic (mortality over 50% of seedlings). In this study the patogenicity of *H. parviporum* population was higher. Only 22% of genets caused mortality less than 20% of seedlings, and 55% were highly pathogenic. These genets were both highly pathogenic and aggressive that was demonstrated by spread of necrosis in Norway spruce stems. Werner and Łakomy [2002 a, b] obtained similar results but they used only one (the same) isolate in both experiments *in vivo* and *in vitro.* The diversity of aggressiveness among isolates of *H. parviporum* was found in several studies [Stenlid and Swedjemark 1988, Swedjemark et al. 1999, Werner and Łakomy 2002 b]. The knowledge about virulence of *Heterobasidion* is very important in making a forecast of *Heterobasidion* spreading in a stand. This study showed that population that occurred in stand in the Suwałki Forest District could colonize the stand rather fast, because most of genets were highly virulent. La Porta et al. [1997] and Łakomy et al. [2005] suggested that size of damages in stand probably depended on genetic diversity

of pathogen's population. In addition Łakomy and Werner [unpublished] showed that sometimes the pathogenicity might be related with genetic diversity. Pathogen's populations differ on the base of genetic variability, areas occupied by genets and stand's age.

The recognition of genetic diversity, genets size and their virulence is very important in prediction of *Heterobasidion* root rot, economic damages and organizing the protection objectives. That is why the study on *Heterobasidion* populations, their virulence, spreading in stands, ability of trees infestation and host resistance should be continued.

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REFERENCES

Bendz-Hellgren M., Brandtberg P.-O., Johansson M., Swedjemark G., Stenlid J., 1999. Growth rate of *Heterobasidion annosum* in *Picea abies* established on forest land and arable land. Scand. J. For. Res. 14 (5), 402-407.

Bodles W.J.A., Beckett E., Zamponi L., Woodward S., Keča N., Capretti P., 2005. *Heterobasidion annosum* population recruitment and spread in a severely infected Sitka spruce stand in north east Scotland. In: Root and butt rots of forest trees. Proceedings of IUFRO Working Party 7.02.01. 11th International conference on root and butt rots. Eds M. Mańka, P. Łakomy. 16-22 Aug. 2004, Poznań-Białowieża, 83-93.

Boyce J.S., 1962. Greenhouse inoculations of coniferous seedlings with *Fome annosus*. Phytopathology 52, 4.

Dalke M., Łakomy P., 2009. Genetic diversity of *Heterobasidion annosum* sensu stricto populations in chosen Scots pine stands with beech in understorey. Acta Sci. Pol., Silv. Colendar. Rat. Ind. Lignar. 8 (2), 17-24.

Delatour C., 1982. Behavior of *Fomes annosus* in the stem of Norway spruce and in laboratory. In: Reistance to disease and pests in forest trees. Eds H.M. Heybreoek, B.R. Stephan, K. von Weissenberg. PUDOC, Wageningen, 268-274.

Dimitri L., 1969 a. Ein Beitrag zur Infektion der Fichtenwurzel durch den Wurzelschwamm *Fomes annosus* (Fr.) Cooke. Forstwissensch. Centralbl. 88, 72-80.

Dimitri L., 1969 b. Untersuchungen über die unterirdischen Eintrittspforten der wichtigsten Rotfäuleerreger bei der Fichte *Picea abies* (Karst.). Forstwissensch. Centralbl. 88, 281-301.

Garboletto M., 1996. The genetic structure of populations of *Heterobasidion annosum* (Fr.) Bref. from the global to local scale: implications for the biology, the epidemiology, and the evolution of a forest pathogen. Univ. Calif. Berkeley.

Ingestad T., 1979. Mineral nutrient requirements of *Pinus sylvestris* and *Picea abies* seedlings. Physiol. Plant 45, 373-380.

Korhonen K., Capretti P., Karjalainen R., Stenlid J., 1998. Distribution *Heterobasidion annosum* intersterility groups in Europe. In: *Heterobasidion annosum*. Biology, ecology, impact and control. Eds S. Woodward, J. Stenlid, R. Karjalainen, A. Hütermann. Univ. Press Cambridge, 93-104.

La Porta N., Capretti P., Kammiovirta K., Karjalainen R., Korhonen K., 1997. Geografhical cline of DNA variation within the F intersterility group of *Heterobasidion annosum* in Italy. Plant Pathol. 46, 773-784.

Łakomy P., Broda Z., Werner A., 2007. Genetic diversity of *Heterobasidion* spp. in Scots pine, Norway spruce and European silver fir stands. Acta Mycol. 42 (2), 203-210.

Łakomy P., Cieślak R., 2008. Early infection of *Fagus sylvatica* by *Heterobasidion annosum* sensu stricto. For. Path. 38, 314-319.

Łakomy P., Cieślak R., Rodak W., Kostrzewski T., 2001. Wpływ porażenia przez *Heterobasidion annosum* wybranych drzewostanów sosnowych i świerkowych na powstanie wiatrołomów i wiatrowałów w 1999 i 2000 roku [Influence of *Heterobasidion annosum* on wind damages in Scott pine and Norway spruce stands, in 1999 and 2000]. Sylwan 7, 43-54 [in Polish].

Łakomy P., Kowalski T., Werner A., 2000. Preliminary report on distribution of *Heterobasidion annosum* intersterylity groups in Poland. Acta Mycol. 35 (2), 303-309.

Łakomy P., Kwaśna H., Cieślak R., Molińska-Glura M., Dalke-Świderska M., 2011. Zróżnicowanie genetyczne populacji *Heterobasidion annosum* sensu stricto i *Heterobasidion parviporum* w wybranych drzewostanach sosnowych i świerkowych w Polsce [Genetic diversity of *Heterobasidion annosum* sensu stricto and *Heterobasidion parviporum* in chosen Scots pine and Norway spruce stands in Poland]. Sylwan 156 [in print; in Polish].

Łakomy P., Werner A., 2003. Distribution of *Heterobasidion annosum* intersterlility groups in Poland. For. Path. 33, 1-8.

Łakomy P., Werner A., Broda Z., 2005. Pathogenicity of *Heterobasidion annosum* S group clones to Norway spruce seedlings. In: Root and butt rots of forest trees. Proceedings of IUFRO Working Party 7.02.01. 11th International Conference on Root and Butt Rots. Eds M. Mańka, P. Łakomy. 16-22 Aug. 2004, Poznań-Białowieża, Poland, 94-100.

Mańka K., 2005. Fitopatologia leśna [Forest pathology]. PWRiL Warszawa [in Polish].

Piri T., 1996. The spreading of the S type of *Heterobasidion annosum* from Norway spruce stumps to the subsequent tree stand. Eur. J. For. Path. 26, 193-204.

Piri T., Korhonen K., 2007. Spatial distribution and persistence of *Heterobasidion parviporum* genets on a Norway spruce site. Forest Pathol. 37, 1-8.

Piri T., Korhonen K., Sairanen A., 1990. Occurrence of *Heterobasidion annosum* in pure and mixed spruce stands in Southern Finland. Scand. J. For. Res. 5, 113-125.

Rieger S., 1995. Infection of Norway spruce (*Picea abies*) by *Heterobasidion annosum* in relation to tree age. Eur. J. For. Path. 25, 357-365.

Rishbeth J., 1951. Observations on the biology of *Fomes annosus* with particular reference to East Anglian pine plantations. (II) Spore production, stump infection, and saprophytic activity in stumps. Ann. Bot. NS 15 (57), 1-21.

Rishbeth J., 1957. Some further observations on *Fomes annosus* Fr. Forestry 30, 69-89.

Sierota Z., 2001. Choroby lasu [Forest diseases]. Centr. Infor. Lasów Państw. Warszawa [in Polish].

Slaughter G.W., Parmeter J.R. Jr., 1995. Enlargement of tree-mortality centers surrounding pine stumps infected by *Heterobasidion annosum* in northeastern California. Can. J. For. Res. 25: 244-252.

Stenlid J., 1985. Population structure of *Heterobasidion annosum* as determined by somatic incompatibility, sexual incompatibility and isoenzyme patterns. Can. J. Bot. 63, 2268-2273.

Stenlid J., Redfern D.B., 1998. Spread within tree to stand. In: *Heterobasidion annosum*. Biology, ecology, impact and control. Eds S. Woodward, J. Stenlid, R. Karjalainen, A. Hütermann. Univ. Press Cambridge, 125-142.

Stenlid J., Swedjemark G., 1988. Differntial growth of S- and P-isolates of *Heterobasidion annosum* in *Picea abies* and *Pinus sylvestris*. Trans. Br. Mycol. Soc. 90 (2), 209-213.

Stenlid J., Wästerlund I., 1986. Estimating the frequency of stem rot in *Picea abies* using an increment borer. Scand. J. For. Res. 1, 303-308.

Swedjemark G., Johannesson H., Stenlid J., 1999. Intraspecific variation in *Heterobasidion annosum* for growth in sapwood of *Picea abies* and *Pinus sylvestris.* Eur. J. For. Path. 29, 249-258

Wagn O., 1980. Host plants of *Fomes annosus* in Denmark. In: Proc. of 5th conference on root and butt rots. Kassel, West Germany. Ed. L. Dimitri. Kassel: Intern. Union For. Res. Organ., 182-189.

Webb R., Alexander S., 1985. An update host index for *Heterobasidion annosum*. Inform. Ser. Coll. Agric. Life Sci. Virginia Polytech. Inst. State Univ. 2, 1-27.
Werner A., 1987. Responses in vitro grown pine seedlings to infection by four strains of *Heterobasidion annosum*. Eur. J. For. Path. 17, 93-101.
Werner A., 1991. Odporność sosny zwyczajnej na hubę korzeni i przebieg choroby siewek sosny zakażonych grzybem *Heterobasidion annosum* [Resistance of Scott pine to root rot and the disease development of pine seedlings infected by *Heterobasidion* annosum]. PWRiL Poznań, 1-168 [in Polish].
Werner A., Łakomy P., 2002 a. Intraspecific variation in *Heterobasidion annosum* (Fr.) Bref. for mortality rate on *Pinus sylvestris* L. and *Picea abies* (L.) Karst. seedlings grown in pure culture. Mycologia 94 (5), 855-860.
Werner A., Łakomy P., 2002 b. Host specialization of IS-group isolates of *Heterobasidion annosum* to Scots pine, Norway spruce and common fir in field inoculation experiments. Dendrobiology 47, 59-68.

WIRULENCJA POPULACJI *HETEROBASIDION PARVIPORUM* Z DRZEWOSTANU ŚWIERKOWEGO NADLEŚNICTWA SUWAŁKI

Streszczenie. Celem badań było określenie wirulencji izolatów *H. parviporum* reprezentujących różne genotypy o określonym pokrewieństwie i należące do populacji patogena zasiedlającej część drzewostanu świerkowego. Do badań wykorzystano dziewięć izolatów, o określonym podobieństwie genetycznym, reprezentujących odrębne genotypy populacji *H. parviporum* zasiedlające pniaki w drzewostanie świerka pospolitego w Nadleśnictwie Suwałki. Wykonano dwa doświadczenia infekcyjne. Doświadczenie infekcyjne *in vitro* przeprowadzono na pożywce agarowej zawierającej mikro- i makroskładniki. Po miesiącu wzrostu siewki były inokulowane grzybnią patogena. Patogeniczność określano na podstawie śmiertelności siewek po 9 miesiącach wzrostu. W doświadczeniu infekcyjnym *in vivo* użyto 3-letnich sadzonek świerka pospolitego posadzonych w donicach trzy miesiące przed założeniem doświadczenia. Strzałki świerkowe inokulowano grzybnią w ranę wykonaną nad szyją korzeniową. Po 4 miesiącach drewno strzałek analizowano pod kątem nekrozy powstałej w wyniku zasiedlenia i rozkładu drewna powodowanego przez patogena. Wirulencję genotypów określano na podstawie zdolności grzybni do zasiedlania żywego drewna oraz powodowanej śmiertelności siewek świerka pospolitego. Śmiertelność siewek wahała się od 16% do 80%. Analiza wariancji wykazała istotne różnice w śmiertelności siewek świerka pospolitego powodowanej przez poszczególne genotypy *H. parviporum.* Grzybnia poszczególnych genotypów patogena przerosła drewno strzałek sosnowych na średnią odległość od 0,8 mm do 11,8 mm. Różnice w agresywności genotypów występowały pomiędzy izolatami we wszystkich grupach pokrewieństwa. W badanej populacji *H. parviporum* pięć genotypów charakteryzowało się wysoką wirulencją, dwa średnią, a dwa niską.

Słowa kluczowe: *Heterobasidion parviporum,* genotypy, wirulencja, świerk pospolity

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