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Structure of populations of wheat powdery mildew *(Erysiphe graminis* DC f.sp. *tritici* Marchal) in Central Europe in 1993–1996: I. Dynamics of virulence

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Abstract

In 1993–1996, the virulence of regional populations of the wheat powdery mildew pathogen (*Erysiphe graminis* DC f. sp. *tritici* Marchal) from the Czech Republic, Austria, Hungary and Slovakia against 13 resistance genes was investigated. The populations differed mainly at the regional level. Populations from the Czech Republic, mainly from the western regions, showed higher values of virulence against the *Pm4b* gene. Lower frequency of virulence against *Pm4b* was found in Austria, and the lowest value was observed in Hungary. The differences in frequencies of virulence against *Pm4a* and *Pm4b* showed a similar geographic pattern across the four countries: a continuous decline from west to east and from north to south. Virulence against *Pm2* decreased in all countries considered; virulence to *pm5, Pm6, Pm8* and *Mli* was high throughout. Genes and gene combinations that can ensure a relatively effective biological protection against this pathogen across Central Europe at present are *Pm3b, Pm2+Mld* and *Pm1+2+9*. Czech and Slovak populations were the most complex: virulence complexity reached a maximum in Slovakia in 1994. A similar evolution, though less significant, was observed in the Czech Republic. Data on complexity of isolates suggest that Central European populations of wheat powdery mildew tend to reach an intermediate level representing the optimal number of virulence genes. This process is probably a consequence of stabilizing selection.

Introduction

Powdery mildew is one of the most important fungal diseases of wheat and is caused by *Erysiphe graminis* f. sp. *tritici*. There are several ways to control this disease. The biological control is based on cultivating wheat varieties carrying specific or non-specific resistance or on growing mixtures of varieties. The history of intensive growing of wheat as a monoculture over the past years favoured rapid adaptation of the pathogen to overcome the resistance of host varieties formed by specific resistance genes as well as fungicides.

As powdery mildew conidia are spread by wind, populations in different countries can influence each other because there are no obstacles to this pathogen across different countries. For this reason, in the past years attention has been focused on the survey of the pathogen across extensive regions (Wolfe and Limpert, 1987; Felsenstein et al., 1991; Zeller and Fischbeck, 1992; Andrivon and Vallavieille-Pope, 1993).

The first survey on frequency of some virulence genes of wheat powdery mildew from Austria and the former Czechoslovakia was presented by Felsenstein (1991). Virulence in one locality in Hungary has been investigated over a longer period by Szunics and Szunics (1992, 1995). Survey of virulence of wheat powdery mildew in Slovakia began in 1992 (Švec et al., 1993). In 1993, this research was also extended to

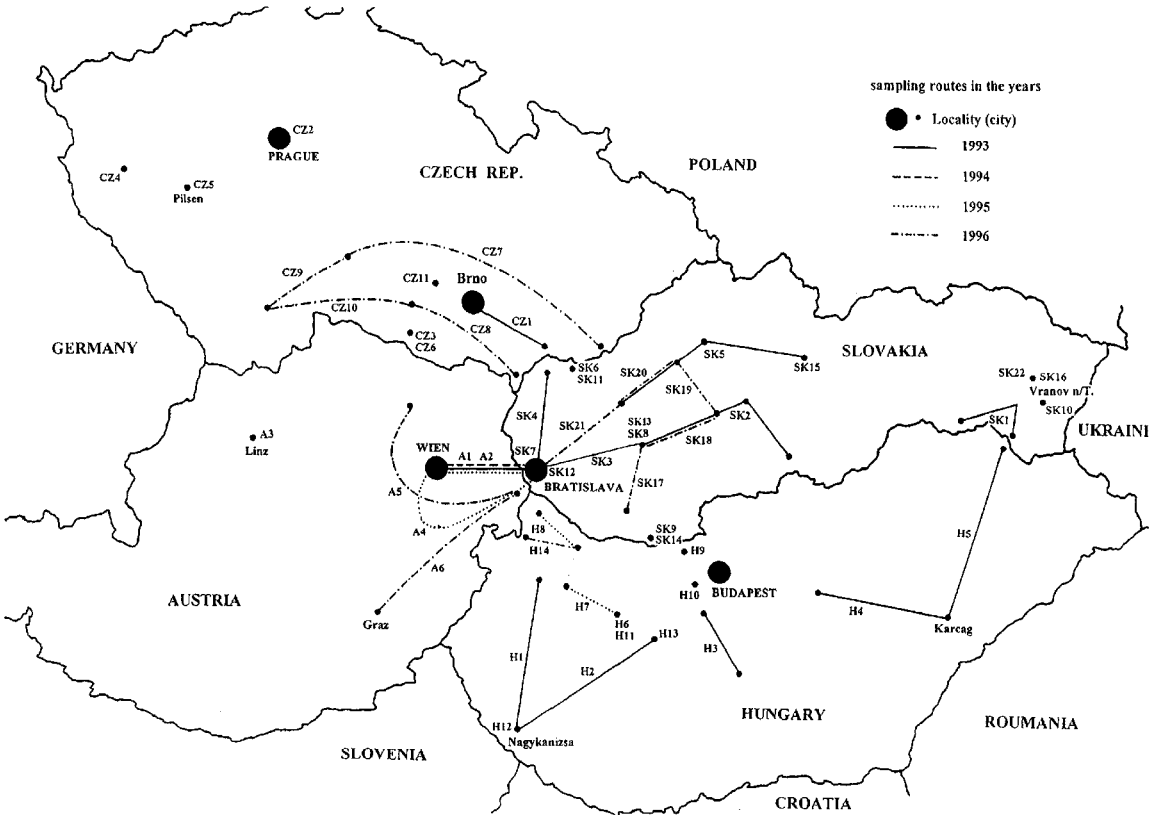

Figure 1. Network of spore sampling routes in 1993–1996.

the neighbouring countries. The aim of this survey is to provide information on actual effectiveness of major resistance genes and on development of virulence patterns in this part of Central Europe.

Materials and methods

Random spore samples of *Erysiphe graminis* DC f. sp. *tritici* were obtained from air by means of a jet spore sampler mounted on the roof of an automobile (Schwarzbach, 1979; Limpert and Schwarzbach, 1981; Limpert et al., 1984) while driving across regions of interest for epidemiology and wheat cultivation. The distance covered during sampling averaged 150 km (Figure 1). Trapped spores from the air fell onto segments of primary leaves of the susceptible wheat variety Košutka placed in Petri dishes containing 7% agar with 30 mg benzimidazole litre^{-1}. Additionally, spores were collected by means of a stationary nursery (seedlings of the susceptible variety Košútka grown in pots were exposed to air on high buildings) or by collecting them from plants of different varieties from the field. Sampling was carried out in May. Trapped spores were incubated in a climate chamber at 18 °C under continuous light (960 lux) . After 10 days of incubation each differential set was inoculated with the progeny of a single colony isolate by drawing spores into a pipet and blowing them into a miniature settling tower at the bottom of which the differential set was placed on agar in a Petri dish. Inoculum density was approximately 200 conidia cm^{-2}. Twelve days after inoculation the severity of attack on each leaf segment was scored relative to a susceptible check in the set. Single colonies of isolates were analysed on sets of leaf segments (length of 1.5 cm) cut from the first leaf of 8-10 day-old seedlings. Virulence tests were carried out on a differential set consisting of near-isogenic lines with Chancellor background and other varieties with single gene or combination of resistance genes: Axminster/8xCc (*Pm1*), Ulka/8xCc (*Pm2*), Asosan/8xCc (*Pm3a*), Chul/8xCc (*Pm3b*), Sonora/8xCc (*Pm3c*), Khapli/8xCc (*Pm4a*), Armada/8xCc (*Pm4b*), Hope (*pm5*), Timgalen (*Pm6*), Salzmuende 14 (*Pm8*),

Table 1. Distribution of the virulence against wheat resistance genes in 1993 (in %)

Locality[a]	SK1	SK2	SK3	SK4	SK5	CZ1	A1	H1	H2	H3	H4	H5
n	19	24	22	29	22	30	30	17	19	30	26	22
Resist. Genes												
Pm1	22	67	41	52	68	34	61	47	37	26	58	50
Pm2	(-)	96	50	90	86	63	65	53	100	29	69	68
Pm3a	26	71	41	42	27	44	39	20	47	42	50	59
Pm3b	5	17	10	3	14	39	14	30	27	23	8	23
Pm3c	32	83	68	66	55	53	71	60	40	31	50	82
Pm4a	74	58	59	52	41	75	77	47	21	65	39	64
Pm4b	58	33	55	41	27	56	19	6	11	53	31	41
Pm6	79	100	100	93	81	91	55	88	100	94	100	96
Pm2+Mld	5	(-)	18	(-)	23	17	29	10	5	15	27	32
Pm1+2+9	5	21	23	17	23	6	23	35	16	13	19	14

(–) = not evaluated, n = number of isolates, [a] see Figure 1.
SK1 = Moldava n/B. – Slov.N.Mesto, SK2 = Nitra - Kremnica - Lučenec, SK3 = Bratislava - Nitra, SK4 = Bratislava - Holíč, SK5 = Piešťany - Pov. Bystrica - Ružomberok, CZ1 = Strážnice - Brno, A1 = Bratislava - Wien, H1 = Sárvár - Nagykanizsa, H2 = Nagykanizsa - Siofok, H3 = Mártonvás.- Dunaföldvár, H4 = Jaszberény - Karcag, H5 = Karcag - Sátoraljaújhely.

Table 2. Distribution of the virulence against wheat resistance genes in 1994 (in %)

Locality[a]	SK6	SK7	SK8	SK9	SK10	CZ2	CZ3	A2
n	34	34	27	26	28	26	34	25
Resist. Genes								
Pm1	74	79	37	50	61	58	74	44
Pm2	68	65	74	50	61	46	74	44
Pm3a	65	56	33	65	29	42	21	32
Pm3b	38	35	7	23	11	1	21	36
Pm3c	100	94	59	77	40	89	77	60
Pm4a	74	77	89	62	68	92	71	72
Pm4b	56	53	63	42	61	76	44	40
Pm6	91	94	93	93	100	93	91	96
Pm2+Mld	15	24	37	8	7	15	3	24
Pm1+2+9	21	41	26	27	36	42	21	12

n = number of isolates, [a] see Figure 1.
SK6 = Skalica, SK7 = Bratislava, SK8 = Nitra, SK9 = Komárno, SK10 = Michalovce, CZ2 = Praha, CZ3 = Vranov n/D., A2 = Bratislava - Wien.

Milan (*Mli*), Maris Dove (*Pm2+Mld*), Normandie (*Pm1+Pm2+Pm9*) and Košutka as the susceptible control. Sporulation of more than 50 per cent was considered to be a virulent reaction. The differences in virulence frequency of the powdery mildew populations and the differences in mean number of virulence genes per isolate in particular countries between the years were calculated by use of a *t*-test.

Results

Regional differentiation

Regional populations differed from each other in their virulence pattern. Virulence frequencies to *pm5*, *Pm8* and *Mli* were close to 100%. Populations from Slovakia, where the highest number of samples was analysed, showed first an increase in virulence in 1994 followed by a subsequent decrease in later years (Tables 2, 3, 4). This pattern of virulence was typical mainly of the regional populations from western Slovakia (Figure 1, localities SK3, SK4, SK6, SK7, SK8, etc.). Regional populations from eastern Slovakia (localities SK1, SK10, SK16, SK22), mainly in 1993-1995, as compared to the other populations, showed a lower frequency of virulence against alleles of the *Pm3* locus and, in turn, an increased frequency against both alleles of the *Pm4* locus (Tables 1-3). The maximum increase in virulence of the total population of powdery mildew in Slovakia, recorded in 1994 in up to 9 virulence genes, was statistically significant in virulence genes against *Pm3b*, *Pm3c*, *Pm4a*, *Pm4b*, *pm5* and *Pm1+2+9* (Table 5).

Regional differences in virulence pattern of populations were also observed in the Czech Republic. In the eastern regions called Moravia (localities CZ1, CZ3, CZ6, CZ8, Figure 1), lower values of virulence against *Pm4b* were observed than in western regions. The population from the western regions from locality Bor (CZ4, Figure 1) differed from the others mainly by

Table 3. Distribution of the virulence against wheat resistance genes in 1995 (in %)

Locality[a]	SK11	SK12	SK13	SK14	SK15	SK16	CZ4	CZ5	CZ6	A3	A4	H6	H7	H8	H9	H10
n	34	26	25	38	38	40	25	30	36	23	15	35	27	31	31	34
Resist. Genes																
Pm1	41	46	32	34	84	58	40	43	33	48	60	46	67	32	29	44
Pm2	68	54	52	68	76	68	72	77	69	70	87	77	78	74	55	74
Pm3a	12	65	28	34	21	43	4	33	33	17	27	26	33	21	32	27
Pm3b	18	42	20	18	18	25	4	30	25	26	0	3	15	3	16	21
Pm3c	71	92	64	87	63	58	44	53	83	39	40	80	93	81	84	85
Pm4a	68	46	72	61	79	75	92	83	83	91	40	49	59	48	48	47
Pm4b	38	28	28	24	58	58	88	70	64	57	13	43	26	21	23	27
Pm6	100	100	76	100	100	95	92	97	97	96	93	100	96	100	100	100
Pm2+Mld	6	8	28	5	25	23	8	13	11	9	7	9	11	6	10	6
Pm1+2+9	12	12	21	11	26	15	20	17	19	13	13	17	37	16	16	27

n = number of isolates, [a] see Figure 1.
SK11 = Skalica, SK12 = Bratislava, SK13 = Nitra, SK14 = Komárno, SK15 = Ružomberok, SK16 = Vranov n/T., CZ4 = Bor, CZ5 = Plzeň, CZ6 = Vranov n/D., A3 = Linz, A4 = Bratislava - Wien - Kittsee, H6 = Veszprém, H7 = Veszprém - Pápa, H8 = Györ - Moson, H9 = Tatabánya, H10 = Székesfehérvár.

Table 4. Distribution of the virulence against wheat resistance genes in 1996 (in %)

Locality[a]	SK 17	SK 18	SK 19	SK 20	SK 21	SK 22	CZ 7	CZ 8	CZ 9	CZ 10	CZ 11	A 5	A 6	H 11	H 12	H 13	H 14
n	32	24	10	26	38	39	36	21	43	15	12	21	35	25	26	28	24
Resist. genes																	
Pm1	44	38	10	69	55	41	58	29	47	47	42	14	54	44	27	43	46
Pm2	25	46	70	77	50	41	50	52	40	53	75	38	29	40	42	39	29
Pm3a	53	25	20	42	45	26	36	33	37	33	25	24	46	36	42	36	54
Pm3b	16	13	20	42	11	5	8	0	21	7	33	10	17	16	12	11	13
Pm3c	94	75	100	73	68	69	78	71	79	67	58	62	77	72	85	85	88
Pm4a	66	83	80	73	71	62	78	86	93	73	67	52	80	64	77	71	63
Pm4b	31	46	50	31	34	28	53	62	72	47	67	10	66	44	46	36	33
Pm6	88	92	90	77	87	95	86	92	91	93	100	86	91	68	73	71	83
Pm2+Mld	16	13	0	21	21	8	17	24	14	33	17	5	11	0	4	4	17
Pm1+2+9	19	13	10	18	18	8	35	14	26	27	33	14	26	16	8	18	21

n = number of isolates, [a] see Figure 1.
SK17 = V. Meder - Nitra, SK18 = Nitra - Žiar n/H., SK19 = Žiar n/H. - Ilava, SK20 = Ilava - Piešťany, SK21 = Piešťany - Bratislava, SK22 = Vranov n/T., CZ7 = Stráně - Pelhřimov, CZ8 = Mor.Budějovice - Břeclav, CZ9 = Pelřimov - České Budějovice, CZ10 = České Budějovice - Moravské Budějovice, CZ11 = Velká Bíteš, A5 = Kittsee - Hollabrunn, A6 = Kittsee - Graz, H11 = Veszprém, H12 = Nagykanizsa, H13 = Siofók, H14 = Halma - Györ.

lower virulence against *Pm3*, and, in turn, by a higher virulence against *Pm4a* and *Pm4b*.

The population from Lower Austria (localities A1, A2, A4, A5), as compared to the others, except Hungary, was characterized mainly by lower occurrence of virulence against *Pm4b*. During 1993–1995, an increase in virulence against *Pm6* was noted in this population. In Upper Austria (locality Linz, A3), higher virulence against *Pm4a*, *Pm4b*, *pm5* and *Pm8* was seen compared to that in Lower Austria.

The population from Hungary in 1993 was characterized by lower values against *Pm1*, *Pm4a*, *Pm4b* and by higher values against the *Pm3* locus. This situation was maintained also in 1995 and 1996.

Table 5. Average values of the frequency of virulence in the countries in 1993 - 1996 and complexity of isolates

Country	SK	SK	SK	SK	SK	SK	SK	SK	CZ	CZ	CZ	CZ	CZ	CZ	CZ	CZ
Year	1993	1994	Signif.	1995	Signif.	1996	Signif.	Signif.	1993	1994	Signif.	1995	Signif.	1996	Signif.	Signif.
Signific.			93–94		94–95		95–96	93–96			93–94		94–95		95–96	93–96
Pm1	50.9	61.7	–	50.7	+	46.7	–	–	34.4	66.7	++	38.4	++	46.4	–	–
Pm2	81.4	64.4	++	65.7	–	47.9	++	++	62.5	61.7	–	72.5	–	49.6	++	–
Pm3a	42.2	50.3	–	32.8	++	37.3	–	–	44.0	29.9	–	25.2	–	34.6	–	–
Pm3b	9.6	24.2	++	22.9	–	16.0	+	–	38.8	11.7	++	20.8	–	13.4	–	++
Pm3c	62.1	75.8	+	71.6	–	76.9	–	++	52.9	81.7	++	62.6	++	74.0	–	+
Pm4a	56.0	73.8	++	67.6	–	70.4	–	+	75.0	79.9	–	85.6	–	82.7	–	–
Pm4b	42.2	55.0	+	40.4	++	34.3	–	–	56.3	58.1	–	72.5	–	61.4	+	–
pm5	91.4	100.0	++	97.0	–	100.0	+	++	93.7	100.0	+	98.9	–	99.2	–	+
Pm6	91.3	93.9	–	96.0	–	88.2	++	–	90.6	91.7	–	95.6	–	90.8	–	–
Pm8	100.	98.7	–	97.0	–	100.0	+	–	100.0	98.4	–	96.7	–	100.0	+	–
Mli	93.9	98.0	–	94.0	–	98.8	++	++	84.4	100.0	++	95.6	–	93.6	–	++
Pm2+Mld	15.9	18.1	–	15.6	–	15.4	–	–	16.6	8.3	–	11.0	–	18.9	–	–
Pm1+2+9	18.1	30.2	+	15.9	++	14.2	–	–	6.3	29.9	++	18.7	–	27.3	–	++
C (%)	58.1	64.9		59.0		57.4			58.1	62.9		61.1		60.9		
χ_i	7.48	8.37	++	7.65	++	7.45	–	–	7.53	8.18	–	7.95	–	7.89	–	–
$SD\chi_i$	0.15	0.16		0.12		0.13			0.29	0.18		0.17		0.16		
N	116	149		201		169			30	60		91		127		

Signif. = Significance of the difference between the years, ++ = significant difference at P = 0.01, + = significant difference at P = 0.05, - = no significant difference; SK = Slovakia, CZ = Czech Republic; *C (%)* = complexity of isolates (in per cent); χ_i = mean number of virulence genes per isolate; $SD\chi_i$ = standard error of mean; *n* = number of tested isolates.

Table 5. Continued: Average values of the frequency of virulence in the countries in 1993–1996 and complexity of isolates

Country	A	A	A	A	A	A	A	A	H	H	H	H	H	H
Year	1993	1994	Signif.	1995	Signif.	1996	Signif.	Signif.	1993	1995	Signif.	1996	Signif.	Signif.
Signific.			93–94		94–95		95–96	93–96			93–95		95–96	93–96
Pm1	61.3	44.0	–	52.6	–	39.3	–	+	42.8	43.0	–	39.8	–	–
Pm2	64.5	44.0	–	76.3	++	32.1	++	++	61.1	71.5	–	37.9	++	++
Pm3a	38.7	32.0	–	21.0	–	37.5	–	–	44.7	27.6	+	41.7	+	–
Pm3b	13.9	36.0	+	26.1	–	14.3	–	–	21.1	11.4	–	12.6	–	–
Pm3c	70.9	60.0	–	39.5	–	71.4	++	–	64.0	84.2	++	82.5	–	++
Pm4a	77.4	72.0	–	71.1	–	69.6	–	–	48.6	50.0	–	68.9	++	++
Pm4b	19.3	40.0	–	39.4	–	44.6	–	+	31.4	28.2	–	39.8	–	–
pm5	96.7	100.0	–	97.4	–	100.0	–	–	97.4	100.0	–	100.0	–	+
Pm6	54.8	96.0	++	94.7	–	89.3	–	++	95.7	99.4	–	73.8	++	++
Pm8	100.0	84.0	++	92.0	–	98.2	–	–	95.6	95.6	–	98.0	–	–
Mli	96.7	100.0	–	92.0	–	96.4	–	–	79.9	96.8	++	98.0	–	++
Pm2+Mld	29.0	24.0	–	7.9	–	8.9	–	+	18.7	8.2	+	5.8	–	++
Pm1+2+9	22.6	12.0	–	13.1	–	21.4	–	–	18.3	22.1	–	15.5	–	–
C (%)	57.4	57.2		55.6		55.6			55.3	56.7		54.9		
χ_i	7.53	7.44	–	7.21	–	7.16	–	–	7.21	7.37	–	7.10	–	–
$SD\chi_i$	0.31	0.44		0.28		0.25			0.16	0.13		0.16		
N	30	25		38		56			114	158		103		

Signif. = Significance of the difference between the years, ++ = significant difference at P = 0.01, + = significant difference at P = 0.05, - = no significant difference; A = Austria, H = Hungary; *C (%)* = complexity of isolates (in per cent); χ_i = mean number of virulence genes per isolate; $SD\chi_i$ = standard error of mean; *n* = number of tested isolates.

Evolution of virulence frequencies

During 1993–1996, a decline was recorded in virulence against *Pm2* in all four countries. A moderate increase in virulence was found against *Pm4b*, mainly in the Czech Republic and Hungary, and a similar situation was also recorded against *Pm4a*. The differences in frequencies of both virulences showed an identical geographic pattern across the four countries: a consistent decline from west to east and from north to south. The highest values of these virulences were found in western Czech Republic and Upper Austria; generally the lowest values were seen in Hungary. During the investigated years, a change was recorded in effectiveness of the *Pm6* gene. While in eastern Slovakia and in Lower Austria virulence in 1993 reached 80% and 55%, respectively, in subsequent years this virulence exceeded 90%, thus reclassifying this resistance gene as ineffective. During the whole investigated period, the *pm5*, *Pm8*, and *Mli* genes were ineffective. Gene combinations *Pm2+Mld* and *Pm1 + 2 + 9* belonged to the most effective resistance genes in all four countries and during the whole investigated period. Average values of virulence against them varied in the respective countries from 5% to 30% (Table 5). The severity of infection of genotypes carrying *Pm1* and *Pm3* genes varied considerably, mainly in a region-dependent manner. The ratio of effectiveness among alleles of *Pm3* was always maintained: the most effective allele being *Pm3b* and the least effective being *Pm3c*.

Complexity

The value of complexity of the isolates was calculated in two ways: as the average value of all virulences (in%) for the respective country (C%, Table 5), and also on the basis of pathotype data, i.e. based on the mean number of virulence genes per isolate (x_i, Table 5).

Statistically significant changes in complexity were found only in data from Slovakia. Here, the average value reached the maximum in 1994, but during the next two years its value again declined to the original level. Similar trends in development of complexity, although below the level of significance, were observed in the Czech Republic. The Austrian and Hungarian populations were in equilibrium, although since 1993 the mean number of virulence genes in Austria has shown a slight decrease.

Discussion

It is evident that the development of powdery mildew populations in Central Europe does not occur at the same time and equally in all countries of this region. Nevertheless, certain tendencies common to all populations may be noted. If the resistance genes are divided into several groups according to their effectiveness, the classification will be identical in all countries. For example, in each country, genes *pm5*, *Pm6*, *Pm8* and *Mli* have been overcome. Despite high values of virulence against these genes, varieties carrying *pm5*, *Pm6* and *Pm8* resistance genes still posses an intermediate mildew control in the field and are being cultivated in large areas (ÚKSUP, 1996). This difference between high virulence on primary leaf segments (under laboratory conditions) and the low degree of infection in the field may be a consequence of higher resistance of adult plants depending on non-specific resistance gene expression. A comparison of our four-year results with earlier data (Felsenstein, 1991) revealed that effectiveness changed mainly for genes *Pm4b* and *Pm6* over the years. It is probable that in eastern Slovakia the pathogen population's adaptation to the presence of resistance gene *Pm6* is delayed, as in 1992 (Švec et al., 1993) the frequency of virulence in this region reached about 30% while in western Slovakia virulence reached 100% as early as in 1993 (Table 1). A contrary virulence dynamics was observed against *Pm2*, despite the fact that gene combination *Pm2+6* is often incorporated into wheat varieties (Hanušová, 1993; Limpert et al., 1994). This fact provides evidence for different durable effectiveness of these two genes, in conformity with the division of genes into strong and weak ones (Vanderplank, 1968).

Against *Pm4b* gene, which during 1989-1992 was one of the most effective genes (Felsenstein, 1991; Švec et al., 1993], a considerable increase in virulence was observed mainly in the Czech Republic where, of 18 registered varieties, 8 carried this gene. *Pm4b* seems to have been incorporated first into varieties which are recommended for growing either in intermediate or in higher altitudes (beet- and potato-growing regions) with a more humid climate, where the virulence values against this gene are high (Prague - CZ2, Bor – CZ4, Plzeň – CZ5, Ružomberok – SK15, Figure 1, Tables 2, 3). With regard to variability of virulence against *Pm4a*, certain tendencies can be observed: a clear decline in virulence from west to east (CZ – SK, A – H) and from north to south. The sim-

ilar pattern of virulence against these two resistance genes suggests the possibility of a closer genetic relationship of the respective genes. From data on the complexity of isolates in Slovakia and the Czech Republic, a similar increase was seen in 1994 and a decrease during the following years (Table 5). It is probable that the development of the pathogen populations in agroecosystems takes place in certain cycles with different amplitude depending on the environmental conditions, as described by Clarke (1976) for natural host-pathogen systems. Similar cycles can be deduced also from data representing the development of virulence of wheat powdery mildew in Switzerland in 1980–1989 (Winzeler et al., 1991). A repeating cyclical pattern of virulence against *Mla 12* in barley was described by Wolfe (1984) in Great Britain. The existence of cycles, i.e. increase and decrease in individual or complex virulence, may be the result of action of directional and stabilizing selection. Vanderplank (1963; 1968) considers vertical resistance in host populations and stabilizing selection in pathogen populations as closely connected. Due to stabilizing selection the races of pathogens are as simple as possible. Marshal and Pryor (1978) stated that the evolution towards complex races depends, among others, on the strength of stabilizing selection against unnecessary virulence genes. The population of powdery mildew in Austria seems to be more stabilized than that in other countries. The value of complexity of isolates in 1995 and 1996 was the same (55.6%). However, this population is maintained in a state of dynamic equilibrium: i.e. if in one year the frequency of necessary virulence genes increased and the frequency of unnecessary genes decreased (or vice versa), the complexity of virulence remained at the same level. Also, the results of Hovmoller (1993) show that selection for one virulence gene may result in decline in frequency of another virulence gene, even in a case with no direct selection against unnecessary virulence genes. However, Parlevliet (1981) assumed that vertical resistance does not necessarily imply stabilizing selection and that if this type of selection seems to be an empty concept in crop pathosystems, it needs to be so in wild plant pathosystems. The fitness of the pathogen population is greatest at some intermediate level of virulence. This tendency toward intermediate values is stabilizing selection in the sense of Vanderplank.

The results concerning the complexity of pathotypes suggest that the Central European populations of wheat powdery mildew also tend to reach an intermediate level representing the optimal number of virulence genes depending on the strength of directional and stabilizing selection. Although in Slovakia there was a decrease in the selection pressure due to the reduction in varieties with resistance genes *Pm4b* and *pm5* in 1995 (Švec et al., 1997), virulence against *Pm4b* correspondingly decreased firstly in western Slovakia, while virulence against *pm5* remained at the same high level.

The problem of stabilizing selection in crop pathosystems is not definitely resolved in our opinion and in the future it will be necessary to observe the virulence and its complexity (necessary and unnecessary genes) contemporaneously in a long temporal period and over an extensive area.

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