

## Presence of the Newly Designated Powdery Mildew Resistance Landi in some Winter Barley Cultivars

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**Abstract:** In the last two decades, resistance to the powdery mildew pathogen has been investigated in a large number of barleys in the Czech Republic. Several tens of winter barley cultivars were identified with a resistance based on an unknown gene or unknown combinations of resistance genes. In this paper tests on 20 of these cultivars are presented. Thirty-two reference isolates of *Blumeria graminis* f.sp. *hordei* were used. All the 20 cultivars shared a particular, previously unknown resistance. Landi was the first cultivar registered with this resistance and it is recommended that this resistance be designated Ln. Isolates virulent to Ln were already found randomly in old European, and also in non-European pathogen populations, where cultivars possessing the resistance Ln were never grown. On the other hand, the resistance Ln has been highly effective even 13 years after Landi registration.

**Keywords:** *Blumeria graminis* f.sp. *hordei*; *Hordeum vulgare*; pathogen isolates; reaction types

Barley (*Hordeum vulgare* L.) annually grown on about 0.5 mil ha ranks among important crops in the Czech Republic. Barley grain is used for feeding farm animals and malt production for the brewing industry as well as for export. Production areas of winter barley stabilized on average at 124 000 ha in 2006–2010 and the proportion of two-rowed winter barley cultivars slightly exceeded 10%.

Powdery mildew caused by the ascomycetous fungus *Blumeria graminis* (DC.) Golovin ex Speer, f.sp. *hordei* Em. Marchal (= *Bgh*) is a dominant disease on winter barley (DREISEITL 2011a) and its susceptible cultivars are sources of inoculum for more widely grown spring barley. Resistance of plants to pathogens is an effective and environmentally friendly way of the disease control. Several concepts how to achieve and utilize the cultivar resistance have been developed. In winter barley cultivars specific-resistance genes to the powdery mildew pathogen predominate (DREISEITL 2007). To support breeding for mildew resistance, sources of specific resistance have been studied (DREISEITL *et al.* 2007; ŘEPKOVÁ

*et al.* 2006, 2009a, b; ŘEPKOVÁ & DREISEITL 2010; TETUROVÁ *et al.* 2010).

Many genes to the powdery mildew pathogen are known, however, individual resistances differ substantially in their effectiveness in the control of the pathogen population. Therefore, the identification of specific resistances in cultivars is a key to their effective utilization in breeding and growing. Specific resistances are often postulated on the basis of specific interactions of the host with pathogen isolates with known virulences. The number of resistances, and especially their combinations that can be postulated, depends on the availability of biological material, i.e. standard cultivars representing as the widest variability as possible in specific resistances of the given host as well as available isolates covering as the widest variability as possible in specific pathogenicity of the given pathogen.

A large number of unknown resistances have been found in barley cultivars over the last two decades (e.g. DREISEITL 2006). Those resistances that were detected in spring barley and were fully effective

to all *Bgh* isolates have been studied by DREISEITL (2011b). The objectives of this research were to use our present collection of reference isolates to repeat tests of winter barley cultivars that had a similar type of unknown powdery mildew resistance.

## MATERIALS AND METHODS

Tests on 20 cultivars with resistance to the powdery mildew pathogen based on an unknown gene or unknown combinations of resistance genes are reported (Table 1). Thirty-two reference isolates of *Bgh* held in the pathogen genebank at the Agricultural Research Institute in Kroměříž were used for reaction tests. Resistances were postulated by comparing the reactions of the tested cultivars with cultivars possessing genes from known resistance sources. This method is based on the assumption that a specific resistance gene of the host is matched by a virulence gene of the pathogen (FLOR 1955).

Further details of the method used including the testing procedure and evaluation can be found in DREISEITL (2011b).

## RESULTS

Twenty cultivars listed in Table 1 were tested and four similar resistance spectra (RSs) were detected (Table 2). To characterize these RSs, ten isolates including all five out of 32 isolates virulent on most of the tested cultivars were selected (Table 3). Resistance spectrum 1 was characterized by reaction type 4 (RT4) after inoculation with the five isolates and by a low RT, mostly RT1-2, after inoculation with the other 27 isolates. Resistance spectrum 2 was composed of RT2 after inoculation with three (3-33/03, A-G/05 and C-132/02) out of the five virulent isolates. Resistance spectrum 3 was characterized mainly by RT0 instead of RT4 after inoculation with two isolates, and RS4 was com-

Table 1. Twenty winter barley cultivars and their postulated resistances to the barley powdery mildew pathogen

Cultivar	No. of rows	Pedigree <sup>1</sup>	First tested	Resistance	
				spectrum	code
CH 58	2	(HVW 12803 × Pastorale) × Laguna	1999	3	Ln Ly
CH 76	2	Marna × KM 104	2005	1	Ln
CH 83	2	Target × Marna	2003	1	Ln
CH 84	2	Target × Marna	2003	1	Ln
CH 85	2	Target × Marna	2003	1	Ln
CH 134	2	FR 86/083/01A × Tiffany	2001	3	Ln Ly
CH 260	2	HVW 12803 × KM 103	1999	1	Ln
CH 264	2	Hanna × KM 104	2007	1	Ln
CH 671	2	Marna × KM 104	1999	1	Ln
CH 695	2	Marna × KM 104	2001	1	Ln
CH 1044	2	HVW 12803 × KM 103	2001	1	Ln
Florian (KM 999/04)	2	HVW 10803 × Monaco	2005	1	Ln
KM 999	2	HVW 10803 × Monaco	2000	1	Ln
KM 1318	2	HVW 12803 × Monaco	1996	1	Ln
KM 1318-303	2	HVW 12803 × Monaco	1998	1	Ln
KM 2030	2	Babylone × Florian	2008	4	Ln Sp
KM 2348	2	Ladoga × Vanessa	2009	4	Ln Sp
Landi	6	(LBP 818 × Tria) × Tapir	1995	2	Ln Ha
LP 6.627	6	HVW 2935 × Sorna	1997	1	Ln
NSL 98-6213	6	(Hilma × Marinka) × Opal	2002	3	Ln Ly

<sup>1</sup>KM 104 = HVW 12803, HVW 10803 is probably identical to HVW 12803

Table 2. Four resistance spectra produced by 20 winter barley cultivars inoculated with 10 isolates of *Blumeria graminis* f.sp. *hordei*

Resistance spectrum	code	Isolate of <i>Blumeria graminis</i> f.sp. <i>hordei</i>									
		Race I	3-33/03	A-G/05	KM 1875	21/97	C-38/01	I-248/99	J-20/04	I-162/09	C-132/02
1	Ln	0	4	4	4	4	1-2	1-2	1-2	1-2	4
2	Ln Ha	0	2	2	4	4	1-2	1-2	1-2	1-2	2
3	Ln Ly	0	0	0	4	4	1-2	1-2	1-2	1-2	4
4	Ln Sp	0	0	0	0	4	1-2	0	0	0	4

Table 3. Ten isolates of *Blumeria graminis* f.sp. *hordei* and their virulences (+) to selected barley resistance genes

Differential line/cultivar	Ml resistance gene(s)	Isolate of <i>Blumeria graminis</i> f.sp. <i>hordei</i>									
		Race I	3-33/03	A-G/05	KM 1875	21/97	C-38/01	I-248/99	J-20/04	I-162/09	C-132/02
Algerian-selection	<i>a1</i> <sup>1</sup>	+				+		+		+	+
P02	<i>a3</i> <sup>2</sup>							+	+		+
P03	<i>a6, a14</i> <sup>2</sup>						+				+
P04B	<i>a7, aNo3</i> <sup>2,3</sup>				+	+	+	+	+	+	+
P08B	<i>a9</i> <sup>2</sup>				+	+	+	+		+	
P10	<i>a12, aEm2</i> <sup>2,3</sup>				+	+	+	+	+	+	+
P11	<i>a13, aRu3</i> <sup>2</sup>					+		+			+
P17	<i>k1</i> <sup>2</sup> , <i>a8</i> <sup>1</sup>			+	+		+	+	+	+	
CH 666	<i>La</i> <sup>5</sup>	+					+	+	+	+	+
P21	<i>g, (CP)</i> <sup>2</sup> , <i>a8</i> <sup>4</sup>			+	+	+	+	+	+	+	+
P20	<i>at</i> <sup>2</sup> , <i>a8</i> <sup>4</sup>			+	+			+	+		+
Kompolti 4	<i>(Bw)</i> <sup>6</sup>	+	+			+		+	+	+	+
Weihenstephan 37/136	<i>h</i> <sup>7,1</sup>				+	+	+	+	+		

<sup>1</sup>DREISEITL (unpublished), <sup>2</sup>KØLSTER *et al.* (1986), <sup>3</sup>JØRGENSEN (1994), <sup>4</sup>JENSEN (1995), <sup>5</sup>DREISEITL (2005), <sup>6</sup>DREISEITL (2007), <sup>7</sup>WIBERG (1974)

posed of RT0 instead of RT4 after inoculation with three isolates (3-33/03, A-G/05 and KM 1875) and of RT0 instead of RT1-2 after inoculation with isolates I-248/99, J-20/04 and I-162/09. Fourteen cultivars exhibited RS1, one cultivar RS2, three cultivars RS3, and two cultivars RS4 (Table 1).

## DISCUSSION

In resistance tests performed in this research, the set of 20 cultivars exhibited four RSs. From the specific interactions shown in Table 3 and from the four observed spectra of interactions, it can be deduced that all the tested 20 cultivars share a so far unknown source of resistance present in 14 cultivars in the absence of other resistance genes (RS1), in one cultivar (Landi) in combination with Ha resistance (RS2), in three cultivars in combination with Ly resistance (RS3) and in two cultivars in combination with Sp resistance (RS4).

The oldest cultivar of the whole set tested herein is Landi, in which the relevant unknown resistance was detected for the first time. Landi was developed by I.G. Pflanzenzüchtung GmbH. and registered in 1995. Therefore, according to agreed guidelines (BOESEN *et al.* 1996), the resistance characterized by RS1 was designated as Ln. LBP 818 is a probable source of this resistance in Landi. However, no more detailed information about the origin of this resistance is known.

HVW 12803 is apparently the source of the resistance Ln in 12 out of 17 two-rowed cultivars. Though it is present in pedigrees of six of them only, HVW 10803 in the pedigrees of KM 999 and KM 999/04 (Florian) is probably identical to HVW 12803 and the different designation is erroneous rewriting only, and KM 104, which is in pedigrees of other four cultivars, is identical to HVW 12803 (breeding records of KM 104 show: HVW 12803 = KM 104).

RSs 1 and 2 indicate that RT0 developed after the inoculation of relevant cultivars with Race I could be determined by other resistance than Ln and Ha. Race I comes from Japan (HIURA & HETA 1955), and it is used to detect the resistance designated HH (Heil's Hanna) because no isolate avirulent to this resistance has been found in the European pathogen population. Thus, it can be assumed that the cultivars characterized by RSs 1 and 2 possess, besides the resistances mentioned, also the resistance HH. RT0-1 instead of RT1-2 was also obtained after inoculating LP 6.627 with two isolates (data not shown).

This difference is not significant enough, however, it indicates a possible presence of the resistance Ra in this cultivar.

Landi was registered in 1995, but the resistance Ln was still highly effective in 2008 because the relevant virulence was found only in two out of 160 isolates trapped from the aerial pathogen population in the Czech Republic (DREISEITL 2008). Therefore, Florian registered in 2008, which is the only cultivar carrying the resistance Ln in the Czech Republic, also belonged to the most resistant cultivars of winter barley.

The resistance Ln could not be fully effective against the pathogen population even before its use in commercial cultivars because domestic isolate D-10 from collections in 1963 (BRÜCKNER 1965) as well as isolate 58-74 found in Sweden before 1976 (JØRGENSEN, personal communication) were virulent to this resistance. Likewise, virulent isolate KM 1875, which was used for the tests herein, was found in Sweden already in 1976 (JØRGENSEN, personal communication). The current pathogen genebank in Kroměříž comprises 26 domestic isolates. Of them, only isolates 21/97 and C-132/02 originating from collections in 1997 and 2002, respectively, are characterized by the virulence *VLn*. However, the virulence to the resistance Ln occurs also outside Europe. It is documented by other virulent isolates used here: 3-33/03 from collections in China in 2003 (DREISEITL & WANG 2007) and A-G/05 obtained in Australia in 2005, as well as Israeli pathogen isolates collected on wild barley (*Hordeum vulgare* subsp. *spontaneum*) (DREISEITL *et al.* 2006). All of the presented European and non-European isolates have been included in the pathogen genebank due to other virulences/avirulences and the virulence to the resistance Ln, by which five isolates are characterized, was obtained by chance.

The randomly detected virulence to the resistance Ln in older European as well as in recent non-European pathogen populations documents that *VLn* was a typical unnecessary virulence. However, directional selection for virulence to the resistance Ln was very slow. Thus, the virulence *VLn* seems to be very stable because it was present in pathogen populations even without directional selection, and on the other hand, its frequency was low in the population with some, though probably not very strong directional selection. It is not common and offers an area for further investigations into this specific relationship of resistance Ln versus corresponding virulence *VLn*.

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