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STUDIES ON RESISTANCE TO DOWNY MILDEW IN SUNFLOWERS (*HELIANTHUS ANNUUS* L.)

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Downy mildew (*Plasmopara helianthi* Novot, is one of the important diseases of sunflowers in France. The first resistant variety, INRA 7702, was registered in 1970, and a second, earlier maturing variety, Rémil, was registered in 1974. Nevertheless, we are continuing a search for, and study of, new sources of resistance.

The first resistance gene to be reported, by VRANCEANU and STOENESCU in 1970, was Pl_1 in the line AD 66. We have this line in our breeding programme. INRA 7702 and Rémil have the resistance which comes from HA 61, a widely used line supplied by KINMAN, U.S.D.A. HA 61 contains two genes which give resistance to the downy mildew race in France, and which are different from Pl_1 (VEAR and LECLERCQ, 1971). Although they were named H1 and H2 at first, they should be known as Pl_2 and Pl_3 to conform with recognised nomenclature. According to ZIMMER and KINMAN (1972), one of these genes, and also Pl_1, are inactive against downy mildew in the United States.

In addition to AD 66 and HA 61, we have a series of resistant sunflowers of Argentinian origin in our breeding programme. They each contain a single resistance gene. Studies of the mildew reaction of the progenies from crosses between a number of these lines and HA 61 were made to determine whether they contained Pl_2 or Pl_3. The progenies from crosses between mildew susceptible lines and the F 1 progenies from HA 61 and CA 73 PI015 and 293 showed no segregation (table 1). The F1 hybrids must have been homosygote for mildew resistance and the resistance genes in the Argentinian lines must be Pl_2 or Pl_3. From observation of the progenies from crosses between these lines (table 1) it appears that all three contain the same gene.

We have a resistant line of French origin which was produced from a cross between *H. tuberosus*, a species resistant to downy mildew and *H. annuus* (LECLERCQ et al. 1970). After a programme of back cross to sunflowers, an exceptional plant, denoted HIR 34, was observed. It had

Table 1

Segregation of reaction to downy mildew in the progenies from crosses between resistant lines

Cross and generation	Number of plants		Total	Assumed ratio	χ^2 (Id : f)
	Resistant	Susceptible			
HA61 × P1015					
F1	27	0	27	1 : 0	
S × F_1	140	0	140		
293 × HA61					
F1	5	0	5	1 : 0	
S × F1	135	0	135	1 : 0	
CA73 × AD66					
F1	23	0	23		
S × F1	52	13	65	3 : 1	0.62
CA73 × 293					
F1	34	0	34		
F2	95	0	95	1 : 0	
S × F1	124	0	124	1 : 0	
CA 73 × P1015					
F1	36	0	36		
S × F1	43	0	43	1 : 0	

When $\chi^2 < 3,84$ $P > 0,05$ S = susceptible

the normal chromosome number of sunflowers, $2x = 34$, but had the mildew resistance of *H. tuberosus*. No chromosomal abnormalities of pairing or karyotype were visible. Since pairing did not normally occur between *H. annuus* chromosomes and the *H. tuberosus* chromosome carrying resistance, it must be supposed that a short length of *H. tuberosus* chromosome had been translocated on to an *H. annuus* chromosome. Morphologically, HIR 34 is a normal sunflower line, except for a few characteristics such as clustered leaves around the capitulum and a tendency to a concave head, which may have been transmitted from *H. tuberosus* with mildew resistance.

In the first generations of descendants from this plant, after both self and cross pollination, some irregularities of inheritance were observed (table 2). There was an excess of resistant plants in HIR 34-1 and an excess of susceptible plants in (Cm x HIR 34) F1 progeny. However, the 1 resistant : 1 susceptible ratio in the (♀ 5 xHIR 34) F1 progeny and in test cross progenies and the 3 resistant : 1 susceptible ratio in the F2 and some F3 progenies indicate the presence of a single dominant gene for resistance, present in heterozygous state in HIR 34. Reciprocal crosses between susceptible plants and descendants of HIR 34 were made to check the transmission of this gene via the pollen and the ovule. In conditions where the control HA61 gave ratios of 3 resistant : 1 susceptible in test crosses (table 3), the progenies from similar test crosses with HIR 34 gave ratios of 1 susceptible : 1 resistant. This was true whether the resistance was transmitted by the pollen or by the ovule. Thus, the resistance gene from *H. tuberosus* appears to be transmitted as part of the *H. annuus* genotype.

Table 2

Segregation of reaction to downy mildew in the descendants of HIR 34

Progeny and generation		Number of plants		Total	Assumed ratio	χ^2 (Id : f)
		Resistant	Susceptible			
HIR 34—1	S1	45	7	52	3 : 1	3.68
HIR 34—1—3	S2	12	0	12	1 : 0	
HIR 34—1—4		17	0	17	1 : 0	
HIR 34—1—5		4	1	5	3 : 1	
HIR 34—1—6		38	18	56	3 : 1	1,50
HIR 34—1—2—1	S3	39	20	59	3 : 1	3.82
HIR 34—1—5—3		32	10	42	3 : 1	0.32
(♀ 5 × HIR 34) a	F1	38	31	69	1 : 1	0.71
	F2	101	27	122	3 : 1	0.50
(Cm46 × HIR 34) a	F1	29	54	84	1 : 1	7.02
(Cm46 × HIR34) a—3	F2	26	5	31	3 : 1	1.5
(Cm 46 × HIR 34) a—1 —2	F3	12	5	17	3 : 1	0.3
Cm 32 × (Cm 46 × BC × F2 HIR 34) a—3		69	65	134	1 : 1	0.12

P > 0,05 when χ^2 < 3,84
R = resistant S = susceptible

Table 3

Reaction to downy mildew in a test for abnormalities in the inheritance of the mildew gene in HIR 34

Cross	Number of plants			Assumed ratio	χ^2 (Id : f)
	R	S	Total		
Cm 32 × (♀ 5 × HIR 34) F1	54	65	119	1 : 1	1.02
(♀ 5 × HIR34) F1 × Nain noir	9	11	20	1 : 1	0.2
Cm 32× (♀ 10 × HA61) F1	58	27	85	3 : 1	2.1
(♀ 10 × HA61) F1 Nain noir *	12	7	19	3 : 1	1.4

P < 0,05 when χ^2 < 3,84
R = resistant S = susceptible
*Nain noir contains a dominant gene T for red anthocyanin production; only the red plants in the progenies were scored.

To determine whether the resistance was due to Pl_1, Pl_2, Pl_3 or another gene, crosses were made between HIR 34 and most of the other resistant lines, and the mildew reactions of a number of progenies were observed (table 4). The progeny from the testcross susceptible x (HA61 x HIR 34) F1 gave a segregation ratio of 7 resistant : 1 susceptible, indicating the presence of three independent genes in the (HA61 x HIR34) F1 plants. The difference of the resistance gene in HIR 34 from those in HA 61 was confirmed by the crosses between

Table 4

Reaction to downy mildew in progenies form crosses between HIR 34 and other resistant lines

Progeny and generation	Number of plants			Assumed ratio	χ^2 (Id : f)
	R	S	Total		
HA61 × HIR34					
F1	36	0	36		
S × F1	76	11	87	7 : 1	
P1015 × HIR 34					
F1	18	0	18		
S × F1	152	48	210	3 : 1	0.52
293 × HIR 34					
F1	18	0	18		
S × F1	64	17	81	3 : 1	0.67
CA73 × HIR34					
F1	33	0	33		
S × F1	20	8	28	3 : 1	0.05
HIR34 × P1014					
F1	7	0	7		
F2	61	5	66		
S × F1	105	39	144	3 : 1	0.11
AD66 × HIR 34					
F1	0	48	48		
F2	98	131	229		
S × F1	51	48	99		

R = resistant S = susceptible
P > O , 05 when $\chi^2 < 3,84$

HIR 34 and CA 73, P 1015 and 293. A11 gave ratios of 3 resistant : 1 susceptible in the test cross progenies, indicationg that the two independent genes were present in each F1 hybrid (table 4). The AD 66 x HIR 34 cross gave anomalous F2 and testcross progenies, but the presence of segregation nevertheless indicates that Pl_1 is not present in HIR 34. We have therefore denoted the resistance gene in HIR 34 as Pl_4. ZIMMER has observed HIR 34 resistant in N. Dakota.

HIR 34 is thus a mildew resistant line of considerable interest. Last year, in Frech national trials, a hybrid with HIR 34 as the female parent gave and average of 30 q/ha, 90% of the control. In three places it had a better yield than the control. This is a useful result, but improvement of the agronomic characteristics of HIR 34 is necessary. In our breeding programme we are transfering Pl_4 in to other genetic backgrounds and also cytoplasmic male sterility in order to obtain good "female" lines. These will be crossed with male fertility restorer lines containing other resistance genes to give hybrids in which 3 or 4 resistance genes are combined. In addition, we are returning to material produced early in the *H. tuberosus x H. annuus* generations and hope to determine whether any further resistance genes can be obtained from *H. tuberosus*.

Thus in Europe at least, the immediate prospects for control of mildew attacks on the sunflower crop are good. Nevertheless, in the long term we must search for new sources of resistance. The use of genes giving complete resistance, often known as vertical resistance, is much debated. No changes in the pathogenicity of downy mildew have been reported, but the existance of different races in Europe and in N. America suggests that variation can occur. To protect the sunflower crop against pathogen variants, either a combination of many vertical resistance genes, or a form of horizontal, race non-specific resistance is necessary.

We are continuing our search for additional complete resistance genes, for example from crosses with other *Helianthus* species resistant to mildew. We have found one form of incomplete resistance, which could be of the horizontal type. Normally, plants which are susceptible to mildew show symptoms 15 days after inoculation. Where incomplete resistance occurs in the absence of any genes giving complete resistance, symtoms do no appear for four to six weeks on some plants (table 5). The rate of development of the pathogen appears to be reduced. The irregular expression of this character has meant that it has not get been possible to determine its mode of inheritance. Retarded susceptibility could be of use in the field if it would enable sunflower plants to reach a stage where mildew symptoms are attenauted and where seed production is little reduced by the appearence of downy mildew.

Table 5

Observations of retarded susceptibility in progenies from the sunflower line C46 in the absence of complete resistance genes

Progeny	Number of plants			% retarded susceptible	Incubation time (days)
	seedling susceptible	retarded susceptible	total		
C46	48	7	55	13	39±7.1
(C46 × HIR34) F1	82	32	114	28	39±8.4
(C46 × AD66) F1× SS	176	19	195	10	40±7.1
(C46 × SS) F1	20	2	22	9	27
(C46 × SS) F1 × SS	84	14	98	14	39

SS = seedling susceptible

This year we have made observations of mildew attack in the field. In addition to the discovery of complete resistance in a number of lines, we have found that some varieties are significantly less attacked than the controls, although they contain no vertical resistance genes (table 6). This resistance is being studied, for a reduction in the level of attack from 60% to 10% as shown by P58 HM3 for example, would make it possible to grow such varieties in mildew infested land. If this type of resistance does not depend on a specific reaction to a particular

Table 6

Observations of downy mildew infection in field trials in 1974

Trial	% mildew attack		Variety	
	control (1)	mean (2)		
41	61.4	49.1±14.2	INRA 7702	0
			353 BC 210	0
			P58 HM3	10.4
			1553 BC210	22,9
42	67.1	48.1±10.3	INRA 7702	0.3
			Rémil	4.2
			9353 BC210	23,5
			10153 BC210	28,8
			10253 BC210	38.8
			10653 BC210	0
43	69.0	56.3±15.1	INRA 7702	0
			Rémil	0
			A45 HM3	11,1
44	49.3	54.5±14.9	INRA 7702	1.3
			P 69	35.7
			61 B2	34.2
			77 B2	25.6
			71 B2	27,6
			52 E2	35,6

(1) control — Airelle
(1) Mean — mean of varieties lacking genes giving complete resistance.

Plasmopara genotype, it could provide, in addition, a long term defence against changes in the pathogenicity of *Plasmopara helianthi.*

In conclusion, one may say that to ensure an adequate control of downy mildew, use must be made of the different genes giving complete resistance and also of certain types of incomplete resistance.

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