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Host responses of different Triticeae to species of the cereal cyst nematode complex in relation to breeding resistant durum wheat

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Summary – Twenty eight lines or cultivars of diploid (genomes A, D, S^I, U), tetraploid (genomes AB, D^vM^v, UM, US^v), and hexaploid (genome ABD) wheat were studied for their capacity to sustain the development of nine populations of *Heterodera avenae* originating from six countries (Algeria, France, Spain, Australia, India, and Israel), two populations of *Heterodera filipjevi* from Russia and Bulgaria, and one population of *Heterodera latipons* from Israel. Screenings were performed in artificial conditions using miniaturized tests. High resistance against populations of *H. avenae sensu stricto* occurred in the three levels of ploidy and in several genomes: S^1 (*T. longissimum*), D^vM^v (*T. ventricosum*), UM (*T. ovatum*), US^v (*T. variabile*), and ABD (*T. aestivum* AUS 4930). Total or intermediate resistance was found in genome D (*T. tauschii* CPI 110813 or AUS 18913) but their expression in synthetic hexaploid wheat was incomplete resistance. It was confirmed that the *Cre1* gene from wheat cv. Loros is ineffective against *H. avenae* populations from Australia, India, and Israel but also against *H. filipjevi.* Inter- and intraspecific differentiation within the cereal cyst nematode complex, based on (a)virulence to Triticeae and fitness, and the use of total and intermediate resistance in breeding programmes are discussed. © Orstom/Elsevier, Paris

Résumé – ***Réaction de différentes Triticeae à un complexe de nématodes à kystes des céréales, en vue de la sélection du blé dur résistant*** – Vingt-huit lignées ou cultivars de *Triticum* diploïdes (génomes A, D, S^I, U), tétraploïdes (génomes AB, D^vM^v, UM, US^v) et hexaploïdes (génome ABD) ont été étudiés pour leur capacité à permettre le développement de neuf populations d'*Heterodera avenae* originaires de six pays (Algérie, France, Espagne, Australie, Israël et Inde), deux populations d'*Heterodera filipjevi* provenant de Russie et de Bulgarie et une population d'*Heterodera latipons* provenant d'Israël. Les tests ont été conduits dans des conditions artificielles selon une technique miniaturisée. Les résultats ont montré une résistance élevée à l'encontre des populations d'*H. avenae sensu stricto* au sein des trois niveaux de ploïdie et dans les différents génomes S^1 (*T. longissimum*), D^vM^v (*T. ventricosum*), UM (*T. ovatum*), US^v (*T. variabile*) et ABD (*T. aestivum* AUS 4930). Des sources de résistance complète ou partielle ont été trouvées dans le génome D (*T. tauschii* CPI 110813 ou AUS 18913), mais leur expression dans les blés hexaploïdes synthétiques est incomplète. Il a été confirmé que le gène *Cre1* du blé cv. Loros est inefficace contre les populations d'*H. avenae* d'Australie, d'Inde et d'Israël, ainsi que contre les populations d'*H. filipjevi.* La différenciation inter- et intraspécifique dans ce complexe de nématodes à kystes des céréales pour leur (a)virulence vis-à-vis de Triticeae et leur capacité reproductive intrinsèque sont discutées, ainsi que l'utilisation de résistances complète et partielle dans les programmes de sélection. © Orstom/Elsevier, Paris

Keywords: cereal cyst nematodes, fitness, *Heterodera*, pathotype, resistance, Triticeae, virulence, wheat.

The cereal cyst nematode complex *Heterodera avenae* Wollenweber, *H. latipons* Franklin, and *H. filipjevi* Madzhidov, to which several 'Gotland-type' populations have recently been attached (Rumpenhorst *et al.*, 1996; Bekal, 1997; Bekal *et al.*, 1997), affects the yield of cereals (Rivoal & Cook, 1993). Damage from these nematodes occurs mostly in Mediterranean regions where agroclimatological conditions (heat, drought) worsen the moisture stress caused by the parasites, as demonstrated for *H. avenae* (Lili *et al.*, 1991). In these regions, widespread cereal monocropping and insufficient or absent weed control provide many potential hosts for a large increase in populations of parasitic nematodes, often including several species (Bekal *et al.*, 1997).

H. avenae is the best known species: it is widely distributed and damage has been assessed in many countries (Rivoal & Cook, 1993). Moreover, this species is polymorphous with many pathotypes (Andersen & Andersen, 1982; Cook & Rivoal, 1998) and at least two ecotypes, indicating an adaptation of its life cycle to the thermal hatching conditions in a Mediterranean climate or in a somewhat temperate oceanic climate (Banyer & Fischer, 1971; Rivoal, 1982).

Economic and environmental constraints make it necessary to develop integrated control systems to ensure a reasonable management of parasite populations, which must remain below their damage thresholds (Roberts, 1993). Resistant varieties are a necessary part of this type of plant protection system.

Table 1. *Origins of Triticum lines arranged by their ploidy level and type of genome.*

Lines	Origins
Diploids (2n=14)	
Genome A	
T. monococcum 157	J. Jahier, INRA, Le Rheu, France
T. monococcum 830	J. Jahier
Genome D	
T. tauschii ssp. *eusquarrosa* var. *meyeri* AUS 18913	R.F. Eastwood, Horsham, Australia
T. tauschii ssp. *eusquarrosa* var. *meyeri/typica* CPI 110813	R.F. Eastwood
Genome S^l	
T. longissimum 18	J. Valkoun, ICARDA, Alep, Syria.
Genome U	
T. umbellulatum 88	M. Zaharieva, IIPGR, Sadovo, Bulgaria
Tetraploids (2n=28)	
Genome AB	
T. durum 7655	J. Jahier
T. dicoccoides 829	J. Jahier
T. dicoccoides 4	J. Jahier
T. turgidum	
226	J. Jahier
cv. Kabir	M. Nachit, ICARDA, Alep, Syria
cv. Chaml	M. Nachit
cv. Oued Zenati	M. Nachit
cv. Korifla	M. Nachit
cv. Jennah Khotifa	M. Nachit
cv. Om Rabi	M. Nachit
cv. Bidi 17	J.C. Dussautoir, INRA, Montpellier, France
cv. Agathé	J.C. Dussautoir
cv. Capdur	J.C. Dussautoir
cv. Huguenot	J.C. Dussautoir
Genome D^vM^v	
T. ventricosum 11	J. Jahier
Genome UM	
T. ovatum 79	P. Monneveux, ENSA, Montpellier, France
Genome US^v	
T. variabile 1	J. Jahier
Hexaploids	
Genome ABD	
T. turgidum (Langdon)x *T. tauschii* AUS 18913	R.F. Eastwood
T. turgidum (Langdon)x *T. tauschii* CPI 110813	R.F. Eastwood
T. aestivum	
cv. Loros	J. MacKey, University of Uppsala, Sweden
cv. Arminda	J. Jahier
AUS 4930	F. Green, Field Crop Pathology, Plant Research Center, Urbvae, South Australia

Table 4. *Host responses of tetraploid* Triticum *species to populations of* Heterodera avenae, H. filipjevi, *and* H. latipons. *For each population, the table gives the mean and standard deviation of the numbers of white females or cysts per plant.*

Nematode populations	*T. durum*	*T. dicoccoides*	*T. dicoccoides*	*T. turgidum*		
	7655	829	4	226	cv. Kabir	cv. Cham1
	e	*d*	*e*	*e*	*e*	*b*
H. avenae						
E43	10.7 ± 3.70 *b*	15.5 ± 3.60 *a*	8.1 ± 2.70 *abc*	8.2 ± 2.50 *bc*	6.3 ± 2.50 *b*	21.8 ± 7.00 *ab*
E42	4.7 ± 1.30 *c*	8.2 ± 3.60 *ab*	5.8 ± 1.80 *bc*	8.5 ± 1.60 *bc*	7.4 ± 1.90 *b*	10.7 ± 4.00 *cd*
Fr1	8.5 ± 3.20 *bc*	16.3 ± 2.30 *a*	10.0 ± 2.80 *ab*	8.5 ± 2.30 *bc*	6.8 ± 1.60 *b*	24.6 ± 4.00 *a*
Fr2	5.0 ± 1.50 *c*	5.7 ± 2.04 *b*	5.3 ± 1.10 *bc*	5.6 ± 1.30 *bc*	6.1 ± 1.50 *b*	13.6 ± 3.00 *bcd*
Fr4	***1.0*** ± 0.79 *d*	10.2 ± 2.80 *ab*	4.5 ± 1.20 *bc*	4.5 ± 1.80 *c*	5.8 ± 3.40 *b*	9.4 ± 2.70 *d*
E48	5.2 ± 2.00 *c*	5.1 ± 1.56 *b*	3.6 ± 1.20 *bc*	6.3 ± 2.10 *bc*	10.4 ± 2.50 *b*	16.0 ± 3.70 *abc*
E57	22.4 ± 6.40 *a*	9.8 ± 3.31 *ab*	8.6 ± 2.30 *ab*	10.5 ± 2.00 *b*	20.2 ± 5.70 *a*	19.6 ± 3.80 *ab*
E50	11.8 ± 5.00 *ab*	10.1 ± 5.00 *ab*	9.4 ± 3.60 *ab*	9.0 ± 5.30 *bc*	8.4 ± 2.80 *b*	19.2 ± 3.80 *ab*
E83	19.3 ± 8.90 *a*	15.1 ± 3.90 *a*	5.8 ± 2.40 *bc*	19.4 ± 4.00 *a*	11.7 ± 4.20 *b*	17.4 ± 3.70 *ab*
H. filipjevi						
E88	7.5 ± 2.80 *bc*	7.7 ± 2.70 *ab*	6.0 ± 1.20 *bc*	7.2 ± 2.50 *bc*	6.6 ± 2.60 *b*	8.6 ± 1.00 *d*
A26	5.6 ± 3.10 *c*	6.0 ± 2.60 *b*	5.5 ± 1.50 *bc*	8.2 ± 2.40 *bc*	6.6 ± 2.30 *b*	10.5 ± 1.50 *cd*
H. latipons						
E69	6.4 ± 1.70 *bc*	7.2 ± 2.40 *ab*	17.0 ± 8.20 *a*	5.5 ± 4.00 *bc*	***3.0*** ± 2.00 *c*	4.6 ± 1.80 *e*

Nematode populations	*T. turgidum*				
	cv. Oued Zenati	cv. Korifla	cv. Jennah *Khotifa*	cv. Om Rabi	cv. Bidi 17
	b	*cd*	*e*	*b*	*bc*
H. avenae					
E43	19.1 ± 8.43 *ab*	18.1 ± 6.60 *ab*	17.7 ± 5.29 *ab*	9.5 ± 2.72 *c*	10.2 ± 2.74 *ab*
E42	11.1 ± 3.31 *cd*	10.1 ± 3.50 *ab*	8.0 ± 2.40 *c*	10.3 ± 1.67 *c*	11.5 ± 2.80 *ab*
Fr1	28.2 ± 4.89 *a*	10.8 ± 3.27 *ab*	19.5 ± 3.51 *a*	14.1 ± 3.48 *bc*	14.7 ± 3.76 *a*
Fr2	5.4 ± 1.53 *c*	15.3 ± 0.60 *ab*	7.4 ± 3.70 *c*	13.1 ± 4.88 *bc*	10.2 ± 2.56 *ab*
Fr4	12.0 ± 6.59 *bc*	9.3 ± 4.40 *ab*	5.4 ± 3.50 *c*	9.6 ± 2.70 *c*	9.2 ± 2.00 *ab*
E48	16.6 ± 3.13 *ab*	11.7 ± 2.80 *ab*	11.0 ± 3.70 *abc*	17.7 ± 3.25 *ab*	11.3 ± 3.04 *ab*
E57	26.7 ± 5.81 *a*	20.3 ± 7.10 *a*	8.0 ± 2.00 *c*	19.3 ± 3.76 *ab*	15.6 ± 5.38 *a*
E50	15.2 ± 2.64 *ab*	11.2 ± 1.80 *ab*	7.7 ± 2.61 *c*	18.7 ± 5.11 *ab*	11.8 ± 4.88 *ab*
E83	16.4 ± 4.72 *ab*	13.0 ± 4.00 *ab*	4.3 ± 1.72 *c*	22.1 ± 2.88 *a*	15.6 ± 4.28 *a*
H. filipjevi					
E88	21.6 ± 6.78 *ab*	11.1 ± 4.78 *ab*	9.6 ± 2.56 *bc*	14.9 ± 4.88 *abc*	11.2 ± 2.25 *ab*
A26	7.1 ± 1.59 *c*	6.8 ± 1.50 *b*	6.5 ± 1.79 *c*	10.6 ± 2.13 *c*	7.2 ± 2.17 *b*
H. latipons					
E69	7.5 ± 3.30 *c*	3.9 ± 2.12 *c*	***1.7*** ± 1.70 *d*	9.6 ± 3.00 *c*	7.2 ± 2.47 *b*

End of table 4 next page

Table 5. *Host responses of hexaploid* Triticum *species to populations of* Heterodera avenae, H. filipjevi, *and* H. latipons. *For each population, the table gives the mean and standard deviation of the numbers of white females or cysts per plant..*

Nematode populations	Langdon × AUS 18913 *e*	Langdon × CPI 110813 *g*	*T. aestivum* cv. Loros *i*	*T. aestivum* cv. Arminda *a*	*T. aestivum* AUS 4930 *j*
H. avenae					
E43	4.8 ± 2.16 *cd*	***2.4*** ± 1.35 *cd*	**0.2** ± 0.35 *e*	23.3 ± 6.84 *a*	**0.2** ± 0.28 *c*
E42	4.1 ± 1.74 *d*	***2.6*** ± 1.38 *cd*	**0.1** ± 0.15 *e*	17.3 ± 5.37 *abcd*	**0.2** ± 0.32 *c*
Fr1	6.4 ± 1.88 *bcd*	4.9 ± 2.35 *abcd*	**0.2** ± 0.36 *e*	21.0 ± 6.82 *ab*	**0.3** ± 0.48 *c*
Fr2	5.5 ± 1.30 *cd*	6.6 ± 1.78 *abc*	**0** *e*	12.4 ± 2.80 *cd*	**0** *c*
Fr4	***2.0*** ± 1.56 *e*	***1.7*** ± 1.53 *d*	**0.2** ± 0.32 *e*	21.0 ± 4.80 *ab*	**0** *c*
E48	11.6 ± 3.07 *b*	***2.5*** ± 1.73 *cd*	**0** *e*	11.1 ± 1.93 *de*	**0.1** ± 0.20 *c*
E57	11.5 ± 3.92 *b*	3.8 ± 1.59 *abcd*	6.4 ± 2.22 *bc*	21.0 ± 4.33 *ab*	*
E50	5.2 ± 1.36 *cd*	3.8 ± 1.84 *bcd*	4.2 ± 1.00 *cd*	17.7 ± 2.74 *abc*	*
E83	22.2 ± 3.56 *a*	4.1 ± 1.91 *abcd*	6.2 ± 1.50 *bc*	19.2 ± 5.12 *ab*	*
H. filipjevi					
E88	10.8 ± 3.84 *b*	13.5 ± 5.18 *a*	17.2 ± 7.75 *a*	14.0 ± 3.00 *bcd*	24.4 ± 5.97 *a*
A26	8.0 ± 1.90 *bc*	8.0 ± 1.17 *ab*	6.5 ± 1.38 *b*	11.0 ± 1.71 *de*	***1.3*** ± 1.00 *b*
H. latipons					
E69	11.5 ± 5.51 *b*	3.1 ± 1.66 *bcd*	3.2 ± 0.96 *d*	8.1 ± 1.63 *e*	**0.4** ± 0.49 *c*

The classification of the log (x+1) *transformed data, according to the Newman-Keuls test ($P \leq 0.05$), concerns both the nematode populations (letters placed under the mean data) and the plants (letters placed under the name of each line or cultivar). Means followed by the same letter are not significantly different. Means with bold or bold italic numbers indicate total (level 1) or intermediate (level 2) resistance, respectively.* * *Tests not carried out.*

of Triticeae tested also could have added a bias to the assessment of their respective resistance level. However, the virulence (seven females per plant on average) of the *H. avenae* population E57 from Israel on *T. tauschii* AUS 18913, a line that, like other diploids, is noted for its low root production, did not support this hypothesis or the reservations made by Eastwood *et al.* (1991) concerning the comparison of host responses of Triticeae with different ploidy levels.

Total resistance implies the presence of genes having a major effect on plant-nematode relationships and nematode populations that are homogeneous for virulence (Cook & Rivoal, 1998). This qualitative resistance was easily demonstrated for *T. ovatum* 79, *T. variabile* 1, and *T. ventricosum* 11, for most of the populations tested. Depending on populations, total and intermediate resistances were observed with *T. monococcum* 157 and 830, *T. tauschii* AUS 18913 and CPI 110813, *T. longissimum* 18, and *T. umbellulatum* 88. Resistance of the bread wheat cv. Loros was not universal with *H. avenae* and did not apply to *H. filipjevi* and *H. latipons.* The data on the resistance of AUS 4930 need to be completed.

The host responses of Triticeae to cyst nematodes were assessed from a small number of cases, considering the richness of this botanical family and the complexity and wide distribution of this group of nematodes (Ritter, 1982; Zaharieva, 1996). However, this evaluation did increase our knowledge on the potential virulence of the nematodes and defined host differentials at both inter- and intraspecific levels. If a mixture of species were suspected, it would be easy to extract *H. latipons* (E69) by culture on *T. ventricosum* 11. Similarly, the two populations A26 and E88 attributed to *H. filipjevi* would be isolated by culture on *T. aestivum* cv. Loros or AUS 4930, or even on *T. variabile* 1, as long as the identity of the nematode species is verified by RFLP on ribosomal DNA (Bekal *et al.*, 1997). Within *H. avenae*, population E57 from Israel is easily differentiated by its almost exclusive virulence to *T. ovatum* 79. The avirulence of both populations of *H. filipjevi* to *T. monococcum* 157 is noticeable. Differences in host responses of *T. monococcum* 830 differentiate pathotypes Fr1 and Fr4 in France, as already mentioned by Rivoal *et al.* (1986).

The tests, made under standard conditions and always with the same inoculum, confirmed that, for both *H. avenae* and *H. filipjevi*, populations can be differentiated by their (a)virulence, which defines distinct pathotypes, and by their reproduction capacity or 'fitness' (Rivoal & Person-Dedryver, 1982). Analysis of variance and/or correspondence factor analysis

Table 6. *Major sources of resistance to cereal cyst nematodes (*Heterodera*) in various* Triticum *lines or cultivars.*

Genotypes	Species and populations*		
	H. avenae	*H. filipjevi* E88, A26	*H. latipons* E69
Diploids			
T. monococcum 157	(-)** Fr2, E48	-	
T. monococcum 830	(-) Fr2, Fr4, E48	- A26 (-) E88	
T. tauschii AUS 18913	- Fr2, E50, E83 (-) E43, E42, Fr4, E48	(-) A26	
T. tauschii CPI 110813	- E42, E48, E50, E83 (-) E43, Fr2, Fr4, E57		(-)
T. longissimum 18	- E42, Fr2, Fr4, E48, E57, E50, E83 (-) Fr1	(-)	-
T. umbellulatum 88	(-) E42, E48	-	
Tetraploids			
T. ventricosum 11	- E43, E42, Fr1, Fr2, Fr4, E48, E57, E50, E83	-	
T. ovatum 79	- E43, E42, Fr1, Fr2, Fr4, E48, E50, E83	-	-
T. variabile 1	- E43, E42, Fr1, Fr2, Fr4, E48, E57, E83	(-)	(-)
Hexaploids			
T. aestivum cv. Loros	- E43, E42, Fr1, Fr2, Fr4, E48		
T. aestivum AUS 4930	- E43, E42, Fr1, Fr2, Fr4, E48	(-) A26	-

**Refer to Table 2 for the origin of the populations.*
***Resistance level: -, total; (-), intermediate.*

revealed marked differences between the Fr1 and Fr4 populations or between the E57 and E83 populations, depending on their development on various durum wheats such as Oued Zenati, Jennah Khotifa, or *T. turgidum* 226. Populations A26 and E88 of *H. filipjevi* were differentiated by their (a)virulence to AUS 4930 and by a difference in reproduction capacity on *T. tauschii* CPI 110813, the durum wheat cultivars Oued Zenati and Huguenot, and the bread wheat cultivar Loros.

As happened with oats and barley, the populations of *H. avenae* had different virulences toward the different Triticeae, which indicates a very high genetic variability in this species (Rivoal *et al.*, 1995; Lasserre *et al.*, 1996). This variability in virulence was well shown by the *Cre1* gene of cv. Loros, the efficacy levels of which clearly differentiated *H. avenae* populations from Europe and North Africa from those from Israel, India, and Australia. However, Fr1 from France and E57 from Israel were attributed to the same pathotype –Ha41– because of their identical (a)virulence characteristics toward oats or barley (Mor *et al.*, 1992; Cook & Rivoal, 1998). In *T. ovatum* 79, the addition of genome M, which includes the *Cre2* gene in *T. ventricosum 11* (Rivoal *et al.*, 1986; Delibes *et al.*, 1993; Jahier *et al.*, 1996), to the genome U cancelled the virulence of all *H. avenae* populations tested, except E57 from Israel. Two accessions with the two genomes UM also proved resistant to a population from Punjab, India (Singh *et al.*, 1991). Testing these genotypes with a larger sample of *H. avenae* populations would increase our knowledge of genetic relationships between this species and Triticeae, which would ease the choice of genotypes in resistance breeding programmes. The same situation was found in the species *H. filipjevi* and the related