Cereal cyst nematode resistance conferred by the *Cre7* gene from *Aegilops triuncialis* and its relationship with *Cre* genes from Australian wheat cultivars

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Abstract: Cereal cyst nematode (CCN; *Heterodera avenae* Woll.) is a root pathogen of cereal crops that can cause severe yield losses in wheat (*Triticum aestivum*). Differential host–nematode interactions occur in wheat cultivars carrying different CCN resistance (*Cre*) genes. The objective of this study was to determine the CCN resistance conferred by the *Cre7* gene from *Aegilops triuncialis* in a 42-chromosome introgression line and to assess the effects of the *Cre1*, *Cre3*, *Cre4*, and *Cre8* genes present in Australian wheat lines on Spanish pathotype Ha71. Inhibition of nematode reproduction was rank-ordered as $Cre1 \geq Cre4 \geq Cre7 \gg Cre8 > Cre3$. Lines carrying *Cre1*, *Cre4*, or *Cre7* exhibited a significantly higher level of resistance than those carrying *Cre8* or *Cre3*. Allelism tests indicated that *Cre7* segregated independently of *Cre1* on chromosome 2BL and *Cre4* on chromosome 2DL, and these genes could consistently be combined in the same genotype, inducing a more durable resistance. Tests to determine the chromosomal location of *Cre7* using addition lines were inconclusive.

Key words: *Heterodera avenae*, pyramiding, *Aegilops*, *Triticum aestivum*.

Résumé : Le nématode de l'avoine (« CCN », *Heterodera avenae* Woll.) est un pathogène racinaire chez les céréales et il peut entraîner d'importantes réductions de rendement chez le blé (*Triticum aestivum*). Des réactions différentielles en réponse à l'interaction hôte–pathotype du nématode sont observées chez les cultivars de blé portant différents gènes de résistance au nématode (*Cre*). L'objectif de ce travail était de mesurer la résistance au CCN conférée par le gène *Cre7* de l'*Aegilops triuncialis* chez une lignée d'introgression à 42 chromosomes et d'évaluer l'effet des gènes *Cre1*, *Cre3*, *Cre4* et *Cre8* (présents chez des blés australiens) sur le pathotype espagnol Ha71. Les gènes ont ainsi été classés en fonction du degré d'inhibition de la reproduction du nématode : $Cre1 \geq Cre4 \geq Cre7 \gg Cre8 > Cre3$. Les lignées portant les gènes *Cre1*, *Cre4* et *Cre7* affichaient un niveau de résistance significativement plus élevé que celles portant les gènes *Cre8* et *Cre3*. Des tests d'allélisme ont indiqué que *Cre7* présentait une ségrégation indépendante par rapport à *Cre1* (situé sur le chromosome 2BL) et *Cre4* (sur le chromosome 2DL). Il serait ainsi possible de combiner ces gènes au sein d'un même génotype pour induire une résistance plus durable. Les tests pour déterminer l'emplacement chromosomique de *Cre7* au moyen de lignées d'addition n'ont pas été concluants.

Mots-clés : *Heterodera avenae*, pyramidage, *Aegilops*, *Triticum aestivum*.

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Introduction

The cereal cyst nematode (CCN; *Heterodera avenae* Woll.) is an important cereal root pathogen that seriously affects many agricultural areas the world over. Since chemical control is expensive and toxic, the development of disease-resistant cultivars is at present the most acceptable alternative. Because current resistance genes may be overcome by

emerging virulent pathotypes, it is essential to search for new resistance specificities. Most CCN resistance (*Cre*) genes have been identified in wheat-related species within the tribe Triticeae, and only two of them (*Cre1* and *Cre8*) have been found in hexaploid wheat (Slootmaker et al. 1974; Ogonnaya et al. 2001; McIntosh et al. 2003). *Cre* genes from the genus *Aegilops* have been transferred to wheat: *Cre2*, *Cre5*, and *Cre6* from *Ae. ventricosa* (Delibes

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et al. 1993; Jahier et al. 2001; Ogonnaya et al. 2001), *Cre3* and *Cre4* from *Ae. tauschii* (Eastwood et al. 1994), *Cre7* from *Ae. triuncialis* (Romero et al. 1998), and *CreX* and *CreY* from *Ae. variabilis* (Barloy et al. 2007). All of these genes confer total or partial resistance to different CCN pathotypes.

The most widespread *Aegilops* species in the world, *Ae. triuncialis*, is an allotetraploid (UC genomes) in which neither the U nor the C genome seems to have undergone substantial modification compared with the U and C genomes in the diploids *Ae. umbellulata* and *Ae. caudata*, respectively (Vanichanon et al. 2003; Badaeva et al. 2004). Introgression wheat line TR-353 ($2n = 41$) carries genes *Cre7* (formerly *CreAet*) and *H30*, from *Ae. triuncialis*, which confer resistance to CCN and Hessian fly (*Mayetiola destructor*), respectively (Romero et al. 1998; Martín-Sánchez et al. 2003; McIntosh et al. 2003). *Cre7* confers high levels of resistance to several European CCN pathotypes, including the Spanish pathotype Ha71, as also revealed by enzyme activities associated with its expression in roots (Romero et al. 1998; Montes et al. 2004).

In this paper we first assess the effects of *Cre1*, *Cre3*, *Cre4*, and *Cre8* on resistance to the CCN pathotype Ha71. Our second aim was the genetic analysis of *Cre7* to determine its relationship with other *Cre* genes effective against Ha71.

Materials and methods

Biological materials

TR lines derived from the cross (*Triticum turgidum* H-1-1 $\times$ *Aegilops triuncialis* A-1) $\times$ *Triticum aestivum* H-10-15 have previously been described (Romero et al. 1998; Martín-Sánchez et al. 2003). Line TR-353 ($2n = 41$) was used as a donor of CCN resistance for breeding lines TR-3531 and TRrd-2 (both $2n = 42$). TR-3531 was obtained by 4 additional selfing rounds and TRrd-2 by backcrossing using different commercial wheat cultivars as recurrent parents (*T. aestivum* 'Osona', *T. aestivum* 'Cartaya', and *T. aestivum* 'Cajeme'). The disomic addition lines *T. aestivum* 'Alcedo'/*Ae. caudata* (C-lines) and *T. aestivum* 'Chinese Spring'/*Ae. umbellulata* (U-lines) and their corresponding amphiploids and parents were supplied by J. Raupp (Manhattan, Kansas, USA). Addition lines 3U and 5C were not available. *Triticum aestivum* 'Loros' and *T. aestivum* 'Iskamish' were gifts from S. Andersen (Copenhagen, Denmark). Synthetic hexaploids containing either *Cre3* (Langdon/AUS 18913 and Langdon/CPI 110809) or *Cre4* (AUS 10942/AUS 18914 and AUS 10942/CPI 110813) were supplied by R. Eastwood (Victoria, Australia). Finally, *T. aestivum* 'Goroke' and *T. aestivum* 'Frame', carrying *Cre1* and *Cre8*, were donated by M. Scurrah (Adelaide, Australia).

CCN resistance test

Soil from an infested field of wheat stubble at the "La Poveda" Experimental Station (Madrid, Spain) was used for routine resistance screening as described in Delibes et al. (1993). CCN pathotype Ha71 was used for all tests described in this report. Greenhouse tests were carried out as described by Ogonnaya et al. (2001) using 15 eggs per gram of soil. The collected soil was potted (250 g per pot) after storage for 6 weeks at 15 °C. Individual seeds were

Fig. 1. Evaluation under field conditions of susceptibility to the cyst nematode *Heterodera avenae* of wheat lines carrying different sources of resistance: *Cre1* (*T. aestivum* 'Loros', *T. aestivum* 'Iskamish', *T. aestivum* AUS 10894, and *T. aestivum* 'Goroke'), *Cre3* (Langdon/AUS 18913 and Langdon/CPI 110809), *Cre4* (AUS 10942/AUS 18914 and AUS 10942/CPI 110813), *Cre7* (TR-353, TR-3531, and TRrd-2), and *Cre8* (*T. aestivum* 'Frame'). Their available parents (open bars), *T. turgidum* 'Langdon', *T. turgidum* AUS 10942, and *T. aestivum* H-10-15, were also tested. Averages of 10–20 plants per stock are presented.

sown in a completely randomized design and seedlings were grown at 18 °C for 6 additional weeks. The degree of resistance was assessed by scoring the number of white female nematodes attached to the roots of each plant as described in Romero et al. (1998). Individual F_1 and F_2 plants were classified as resistant or susceptible using 20% of the maximum infestation level within the segregating population as a demarcation point. This is the most common criterion used in crosses between resistant cultivars. Chi-square tests were conducted on F_2 data to determine the goodness of fit of the experimental data to the expected ratios.

Results

Australian wheat lines carrying *Cre1*, *Cre3*, *Cre4*, or *Cre8*, and different wheat genotypes obtained in our laboratory with CCN resistance transferred from *Ae. triuncialis*, were screened for resistance in a naturally infested field. The results of these tests are summarized in Fig. 1. The available recipients of *Cre* genes (*T. turgidum* 'Langdon', *T. turgidum* AUS 10942, and *T. aestivum* H-10-15) were also tested and proved to be poor hosts for the nematode (16.6, 7.7, and 14 females/plant, respectively). Wheat cultivars with *Cre1*, *Cre4*, or *Cre7* displayed the highest levels of resistance, while those carrying *Cre3* or *Cre8* showed low to moderate resistance. U and C addition lines were also evaluated to determine the chromosomal location of CCN resistance in *Ae. triuncialis*. The wheat parent of the

Table 1. Evaluation under field conditions of susceptibility of *T. aestivum* 'Alcedo'/*Ae. caudata* disomic addition lines and their corresponding amphiploid and parents to the *Heterodera avenae* pathotype Ha71.

	Genome	Reaction to Ha71 (females/plant)
<i>Aegilops</i>		
<i>Ae. caudata</i>	C	0.33
<i>Ae. triuncialis</i>	UC	0.05
<i>T. aestivum</i>		
Anza	ABD	50.20
Alcedo	ABD	39.10
Alcedo/<i>Ae. caudata</i>		
Addition lines		
1C	ABD+1C	4.00
2C	ABD+2C	7.60
3C	ABD+3C	11.00
4C	ABD+4C	4.40
6C	ABD+6C	5.60
7C	ABD+7C	1.00
Amphiploid	ABDC	14.10

Note: *Aegilops triuncialis* and *T. aestivum* 'Anza' were used as resistant and susceptible controls, respectively. Averages of 10–20 plants per stock are presented.

U-lines, Chinese Spring, exhibited a low level of infection, making the U chromosome carrying the CCN resistance elusive (data not shown). The degree of resistance of the 6 available C-lines and their amphiploid was greater than that of the wheat parent Alcedo, which showed a high level of infection similar to that of the susceptible control cultivar, Anza, but lower than that of *Ae. caudata* (Table 1).

The introgression line TR-3531, under field and greenhouse conditions, showed a very high level of resistance very similar to that of the intermediate line TR-353 (Figs. 1, 2). The segregation of CCN resistance in an F₂ population (58 plants) from a cross between TR-3531 and its susceptible parent H-10-15 was studied in a greenhouse test (Fig. 2). All the F₁ plants showed high levels of resistance and the F₂ family segregated into 41 resistant and 17 susceptible plants. This ratio fits the theoretical Mendelian phenotypic ratio of 3:1 for a single dominant gene ($\chi^2 = 0.57$, df = 1, $p = 0.450$), suggesting that the resistance transferred from TR-353 to TR-3531 is likely determined by a monogenic dominant resistance factor (*Cre7*).

Genotypes carrying *Cre1*, *Cre4*, or *Cre7*, which are effective against the Ha71 pathotype, and one genotype carrying *Cre3*, which is ineffective (Fig. 1), were used for allicism studies under field conditions. F₂ progenies were obtained by crossing Australian wheat cultivars or lines carrying *Cre1* (Loros and Iskamish), *Cre3* (Langdon/CPI 110809), and *Cre4* (AUS 10942/CPI 110813) with introgression wheat lines carrying *Cre7* (TR-3531 and TRrd-2). The resistant/susceptible ratios summarized in Table 2 indicate that *Cre1* and *Cre4* segregated independently of *Cre7* in the 4 tested crosses.

Discussion

In this study we demonstrate the efficient transfer of CCN

Fig. 2. Distribution of cereal cyst nematode infection under greenhouse conditions of TR introgression lines with 41 (TR-353) or 42 (TR-3531) chromosomes and the F₂ generation (58 plants) from the cross TR-3531 × H-10-15. The average (vertical arrow) and the 99% confidence interval (horizontal line) are shown for TR-3531, TR-353, and *T. aestivum* H-10-15 (10 plants of each genotype) and for TR-3531 × H-10-15 F₁ (6 plants).

resistance from *Ae. triuncialis* to the introgression wheat lines TR-3531 and TRrd-2 (both $2n = 42$) (Figs. 1, 2). This is also the first time that the Australian wheat lines carrying the *Cre1* (Goroke), *Cre3* (Langdon/AUS 18913 and Langdon/CPI 110809), *Cre4* (AUS 10942/AUS 18914 and AUS 10942/CPI 110813), and *Cre8* (Frame) genes have been assayed against Spanish pathotype Ha71. As described for some *Cre* genes against other pathotypes (Safari et al. 2005), small variations of effectiveness were detected for *Cre1*, *Cre4*, and *Cre7* depending on the genetic background in which they were expressed (Fig. 1). The resistance level in Goroke was lower than that previously described and now confirmed for *Cre1* in Loros, Iskamish, and AUS 10894 (Delibes et al. 1993; Romero et al. 1998). The two genotypes carrying *Cre4* showed a high level of resistance to Ha71, although this gene has been shown to have partial dominance and poor penetrance in the heterozygous state against the Australian pathotype Ha13 (Eastwood 1995). However, we cannot rule out that these lines are carriers of *Cre4* together with a minor CCN resistance factor transferred from the progenitor AUS 10942, which displayed intermediate resistance to Ha71 (Fig. 1). In contrast, the two genotypes carrying *Cre3*, which are highly resistant to Ha13 and susceptible to European pathotypes Ha11 and Ha12 (Ogbonnaya et al. 2001), showed a level of susceptibility to Ha71 similar to that of their parent, Langdon. The cultivar Frame, carrying *Cre8*, which confers partial resistance to Ha13 (Ogbonnaya et al. 2001), was also not very resistant to Ha71. In conclusion, the different CCN resistance genes studied here exhibit varying degrees of efficacy in inhibiting Ha71 reproduction. According to these results and previous data (Ogbonnaya et al. 2001;

Table 2. Reaction to the cereal cyst nematode (CCN) under field conditions of different F₂ populations from crosses between Australian wheat lines carrying different CCN resistance genes (*Cre1*, *Cre3*, and *Cre4*) and introgression lines with *Cre7* (TR-3531 and TRrd-2).

Cross	Involved genes	F ₂ segregation	
		Disease reaction, resistant/susceptible	χ^2 (expected ratio)
TRrd-2 × <i>T. aestivum</i> 'Loros'	<i>Cre7</i> × <i>Cre1</i>	284/16	0.43* (15:1)
TRrd-2 × <i>T. aestivum</i> 'Iskamish'	<i>Cre7</i> × <i>Cre1</i>	246/22	3.27* (15:1)
Langdon/CPI 110809 × TR-3531	<i>Cre3</i> × <i>Cre7</i>	112/32	0.65* (3:1)
AUS 10942/CPI 110813 × TR-3531	<i>Cre4</i> × <i>Cre7</i>	204/7	3.12* (15:1)
AUS 10942/CPI 110813 × TRrd-2	<i>Cre4</i> × <i>Cre7</i>	164/17	3.05* (15:1)

* χ^2 value was 3.841 at $p = 0.05$, 1 df.

Montes et al. 2003), the efficacy of *Cre* genes against the Spanish pathotype Ha71 can be ranked as follows: *Cre1* ≥ *Cre4* ≥ *Cre5* ≥ *Cre2* ≥ *Cre7* >> *Cre8* > *Cre3* > *Cre6*.

The segregation of *Cre7* in the TR-3531 × H-10-15 F₂ family is what would be expected for a single Mendelian factor (Fig. 2). The chromosome in monosomic condition in the intermediate line TR-353 was surely the carrier of the CCN resistance factor because selfing of this line produced plants with 40 chromosomes, which were all susceptible, and plants with 41 and 42 chromosomes, which were all resistant (data not shown). The resistance level conferred by this gene was similar for double (lines TR-3531 and TRrd-2) and single doses (line TR-353 and F₁ progeny from cross TR-3531 × H-10-15), thus confirming that *Cre7* is a dominant gene. Moreover, the 3R:1S segregation in the F₂ population obtained from the cross *Cre3* × *Cre7* (Table 2) also confirms this result and the ineffectiveness of *Cre3* against pathotype Ha71.

Pyramiding different resistance genes (RGs) into a single genotype promotes durable resistance. The level of resistance of the pyramided wheat line carrying *CreX* and *CreY* was significantly higher than that of the single introgression lines (Barloy et al. 2007). Therefore, the combination of *Cre7* with other nonallelic *Cre* genes should be desirable. A total of 4 CCN resistance genes were mapped on homeologous group 2. *Cre1* and *Cre3* are mapped in the distal regions on the long arms of chromosomes 2B and 2D, respectively (Eastwood et al. 1994; Williams et al. 1994) and are homeoloci (de Majnik et al. 2003). *Cre4* and *Cre5* are located on chromosomes 2DL and 2AS, respectively (Eastwood et al. 1994; Jahier et al. 2001). The data summarized in Tables 1 and 2 do not indicate that *Cre7* was transferred to any of these chromosomes. The data in Table 1 suggest the presence of a major RG (or additive RGs) on chromosome 7C, whose corresponding addition line exhibited a resistance level similar to that of hexaploid lines TRrd-2 and TR-3531, and some minor factor(s) in each other available C-line. The unexpected susceptibility displayed by the amphiploid *T. aestivum* 'Alcedo'/*Ae. caudata* (ABDC, octoploid) suggests the possible occurrence of different events affecting gene expression in synthetic wheat polyploids, such as gene loss, gene silencing, modifier genes, and poor expression of RGs in backgrounds with a higher ploidy level (Hanušová et al. 1996; Kashkush et al. 2002). The results presented in Table 2 indicate that *Cre7* segregates independently of *Cre1* on the distal region of chromosome 2BL and *Cre4* on the proximal region of chro-

somosome 2DL. These data allow us to conclude that RGs in Loros (*Cre1*), Iskamish (*Cre1*), and AUS 10942/CPI 110813 (*Cre4*) are not alleles of the *Cre7* locus in TR-3531 or TRrd-2. However, we cannot rule out the location of *Cre7* in group 2 chromosomes at different loci from *Cre1* or *Cre4*. From all these data it can be concluded that *Cre1*, *Cre4*, and *Cre7* can be pyramided into a single genotype. Consequently, screening of F₂ plants carrying two different *Cre* genes derived from crosses between *Cre7* × *Cre1* and *Cre4* × *Cre7* is under way with the aim of producing wheat varieties with greater and lasting resistance to different CCN pathotypes.

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