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Characterisation of resistance to turnip mosaic virus in oilseed rape (*Brassica napus*) and genetic mapping of *TuRB01*

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Abstract Turnip mosaic virus (TuMV) is the major virus infecting *Brassica* crops. A dominant gene, *TuRB01*, that confers extreme resistance to some isolates of TuMV on *Brassica napus* (oilseed rape), has been mapped genetically. The mapping employed a set of doubled-haploid lines extracted from a population used previously to develop a reference RFLP map of the *B. napus* genome. The positioning of *TuRB01* on linkage group N6 of the *B. napus* A–genome indicated that the gene probably originated from *Brassica rapa*. Resistance phenotypes were confirmed by indirect plate-trapped antigen ELISA using a monoclonal antibody raised against TuMV. The specificity of *TuRB01* was determined using a wide range of TuMV isolates, including representatives of the European and American/Taiwanese pathotyping systems. Some isolates of TuMV that did not normally infect *B. napus* plants possessing *TuRB01* produced mutant viruses able to overcome the action of the resistance gene. *TuRB01* is the first gene for host resistance to TuMV to be mapped in a *Brassica* crop. A second locus, *TuRB02*, that appeared to control the degree of susceptibility to the TuMV isolate CHN 1 in a quantitative manner, was identified on the C-genome linkage group N14. The mapping of other complementary genes and the selective combining of such genes, using marker-assisted breeding, will make durable resistance to TuMV a realisable breeding objective.

Key words *Brassica* · TuMV Resistance · Genetic mapping · Mutation · Plant breeding

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Introduction

Turnip mosaic virus (TuMV) is a member of the *Potyvirus* genus which is the largest genus of plant viruses, with 180 members (Shukla et al. 1994). In an international survey of economically significant field-vegetable viruses (Tomlinson 1987), TuMV was found to be second only to cucumber mosaic virus in importance. TuMV has a wide experimental host range infecting 318 species from 156 genera (Edwardson and Christie 1991). It naturally infects horticultural *Brassica* crops (including cauliflower, broccoli, cabbage, Brussels sprout and Chinese cabbage), other horticultural crops (including artichoke, peas, rhubarb, chicory and lettuce), arable *Brassica* crops (including oilseed rape and turnip rape), ornamentals (including stocks and wall flowers) and a wide range of weed species. TuMV is transmitted in a non-persistent manner by over 40 aphid species and occurs in the temperate and tropical regions of Africa, Asia, Australia, Europe, India and North and South America. TuMV is particularly damaging in parts of the world where horticultural and arable *Brassica* crops are grown all year round, such as Canada (Stobbs et al. 1991), China (Liu et al. 1996), Taiwan (Yoon et al. 1993), Korea (Choi et al. 1992), and the UK (Hardwick et al. 1994).

Attempts to control vector and TuMV spread with insecticides have been unsuccessful (Evans and MacNeil 1983; Niu et al. 1983) and the deployment of virus-resistant crop varieties is likely to be the most effective, environmentally friendly and sustainable approach to control. The plant genes that confer resistance against viruses mediate a wide range of interactions from reduction or restriction of infection through to complete immunity to disease development (Ponz and Bruening 1986; Fraser 1990). Several resistances effective against TuMV have been identified and partially characterised in *Brassica napus* (Shattuck and Stobbs 1987; Walsh 1989) and *Brassica rapa* (Suh et al. 1995). However, the only gene for resistance to TuMV that had been mapped previous to the current report was the *Tu* gene of lettuce (*Lactuca sativa*) (Robbins et al. 1994).

B. napus (oilseed rape) is an amphidiploid species equivalent to a chromosome-doubled interspecies hybrid between *B. rapa* (the A genome) and *Brassica oleracea* (the C genome) (U 1935). The linkage groups of the *B. napus* genetic map have recently been assigned to the A and C genomes (Parkin et al. 1995) and trait mapping using aligned RFLP maps of the *B. napus* genome is becoming routine (Parkin et al. 1994; Fray et al. 1997). With the advent of marker-assisted breeding in *Brassica* crops (Lydiate et al. 1995) the rational combining of individual resistance genes to create durably resistant varieties has become a realisable objective. This paper describes the mapping of a gene for resistance to TuMV in *B. napus* and the interactions of this gene with a wide range of TuMV isolates representing different pathotypes.

Materials and methods

Plant material and RFLP analysis

The *B. napus* lines 165, S1, R4 and S6 are four differentials from the European system for pathotyping isolates of TuMV (Jenner and Walsh 1996). The spring oilseed rape (SOSR) cultivar "Westar" gave identical phenotypes to line R4 when challenged with five different pathotypes of TuMV (Jenner and Walsh 1996) and N–o–1 is a doubled-haploid (DH) line of SOSR derived from "Westar" via microspore culture (Sharpe et al. 1995). As described in Sharpe et al (1995), N–o–1 was crossed with N-o–9 (a DH line of winter oilseed rape) and one of the resulting F_1 plants was subjected to microspore culture to produce a segregating population of DH lines (N–o–72–8). The N–o–72–8 population was assayed at 277 RFLP-defined loci and the resulting data were used to construct a genetic map of *B. napus* (Sharpe et al. 1995).

Turnip mosaic virus isolates and propagation

The geographic origin, propagation in *B. juncea*, and phenotypes on the *B. napus* differentials of the European pathotyping scheme of all 20 TuMV isolates used in this study (see Table 1) were described by Jenner and Walsh (1996). The UK 1M isolate originated from the UK 1 isolate by mutation (Jenner and Walsh 1996).

Disease assays

The phenotypes of virus-plant interactions were determined using young *B. napus* plants at the two true-leaf stage. The plants were mechanically inoculated (all leaves) as described by Jenner and Walsh (1996) and the plant phenotypes were assessed visually at weekly intervals. Indirect plate-trapped antigen (PTA) ELISA was used as described by Jenner and Walsh (1996) to confirm lack of infection where no symptoms were observed, except for the following modifications: leaf sap was diluted 1:1 in 0.05 *M* sodium carbonate buffer; antibodies were diluted in phosphate-buffered saline (pH 7.3) containing Tween 20 (0.05%) and bovine serum albumin (0.5 g/l); the first antibody was a mouse monoclonal (EMA 67) produced against TuMV isolate CZE 1 and shown to be capable of recognising all isolates of TuMV used in these experiments (Jenner et al. 1999); the second antibody was goat anti-mouse IgG conjugated to alkaline phosphatase (Sigma Chemical Co., Poole, UK) which was incubated for 3 h at room temperature; the substrate was made up in 10% diethanolamine. Complete resistance was verified by performing ELISA tests on inoculated leaves and partial resistance (i.e. the infection of inoculated leaves but the absence of systemic spread) was verified by performing ELISA tests on the fourth (and uninoculated) true leaf.

Fig. 1 Leaves of N-o-1 (*left*) and N-o-9 (*right*) plants illustrating the phenotypes of the parental lines after inoculation with TuMV isolate UK 1

Results

Mapping a single dominant resistance gene in *B. napus*

The resistance/susceptibility phenotypes of the two parental lines (N–o–1 and N–o–9) and of F_1 plants resulting from the N-o-9×N-o-1 cross were tested. All N-o-1 plants were completely resistant to TuMV pathotype 1 (UK 1), no symptoms of virus infection were observed and no virus was detected by ELISA after inoculation (Table 1; Fig 1). N-o–1 was susceptible to pathotypes 3 (CZE 1) and 4 (CDN 1) and the second parental line N-o-9 was susceptible to all three TuMV pathotypes (Table 1). Eight F_1 plants were tested and all were resistant to UK 1 TuMV, demonstrating that resistance was dominant.

A random sample of 28 DH lines from the N–o–72–8 mapping population of *B. napus*, along with the parental lines (N–o–1 and N–o–9), were inoculated (4–5 plants/line per isolate) with TuMV isolates UK 1, CZE 1 and CDN 1. All the lines were completely susceptible to CZE 1 and CDN 1 and the phenotypes after inoculation with UK 1 are presented in Fig. 2. The segregation of resistance (15 lines) and susceptibility (13 lines), in the 28 lines tested, allowed the resistance gene to be localized to two map intervals flanking the pO120b cluster on linkage group N6 (Fig. 2). The resistance gene was called *TuMV RESISTANCE IN BRASSICA 01* (*TuRB01*). Phenotypes were very clear; susceptible plants developed severe systemic mosaic-type symptoms whereas resistant plants showed no symptoms at all.

Stability and specificity of the resistance

Westar (N-o-1) was completely resistant to four of the TuMV isolates (UK 1, CHN 1, JPN 1 and JPN 2), par-

Table 1 Phenotypes of the interactions between 20 isolates of TuMV and various *B. napus* lines. UK 1, CZE 1, CDN 1 and GK 1 represent pathotypes 1, 3, 4 and 9 respectively of the European pathotyping scheme (Jenner and Walsh 1996), CHN 1–5 represent strains C1–5 of the American/Taiwanese pathotyping scheme (Provvidenti 1980; Green and Deng 1985) and CHN 6–12 represent strain groups Tu1–7 of the Chinese strain typing scheme (Liu et al. 1990)

[a] Only inoculated leaves infected, i.e. no systemic spread
[b] Virus appeared to mutate giving atypical phenotype on the R4 differential and the line containing *TuRB01*

Virus isolate (European pathotype) [original strain designation]	Number of plants infected systemically/number inoculated					
	N-o-1	N-o-9	Differentials of European pathotyping scheme			
			165	S1	R4	S6
UK 1(1)	0/10	9/9	0/2	2/2	0/2	2/2
UK 1M(3)	5/5	5/5	0/2	2/2	2/2	2/2
CZE 1(3)	10/10	8/8	0/2	2/2	2/2	2/2
CDN 1(4)	10/10	8/8	2/2	2/2	2/2	2/2
CDN 2(3)	5/5	5/5	0/2	2/2	2/2	2/2
GK 1(9)	3/5[a]	5/5[a]	0/2	1/2[a]	2/2[a]	2/2
CHN 1(1)[C1]	0/4	4/4	0/2	2/2[a]	0/2	2/2
CHN 2(3)[C2]	5/5	4/4	0/2	2/2	2/2	2/2
CHN 3(3)[C3]	5/5	5/5	0/2	2/2	2/2	2/2
CHN 4(3)[C4]	5/5	5/5	0/2	2/2	2/2	2/2
CHN 5(3)[C5]	5/5	4/4	0/2	2/2	2/2	2/2
CHN 6(3)[Tu1]	5/5	4/4	0/2	2/2	2/2	2/2
CHN 7(3)[Tu2]	5/5	5/5	0/2	2/2	2/2	2/2
CHN 8(3)[Tu3]	5/5	4/4	0/2	2/2	2/2	2/2
CHN 9(3)[Tu4]	5/5	4/4	0/2	2/2	2/2	2/2
CHN 10(3)[Tu5]	5/5	4/4	0/2	2/2	2/2	2/2
CHN 11(1[b])[Tu6]	3/5	4/4	0/2	2/2	2/2	2/2
CHN 12(3)[Tu7]	5/5	4/4	2/2	2/2	2/2	2/2
JPN 1(7)	0/5	3/4	0/2	2/2[a]	0/2	2/2
JPN 2(7)	0/5	0/4	0/2	2/2[a]	0/2	2/2

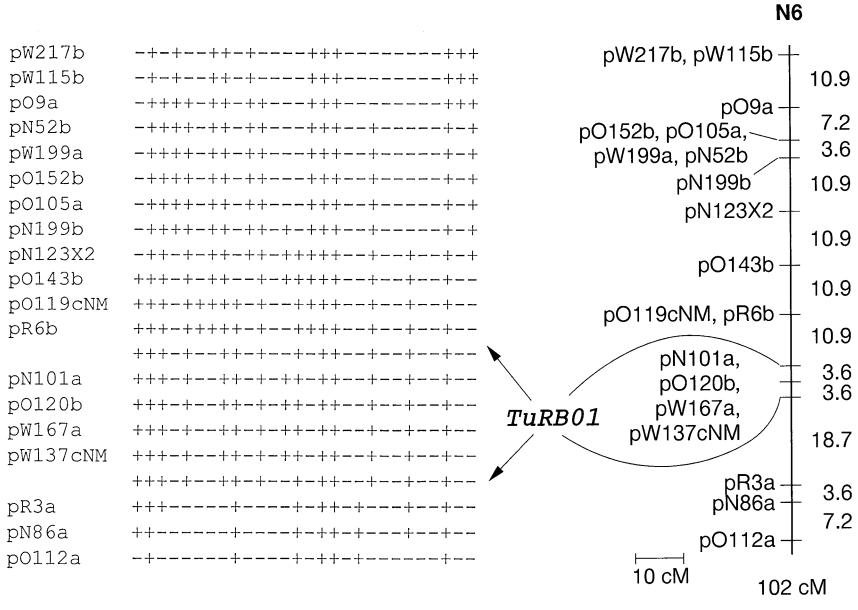

Fig. 2 Genetic scoring data at RFLP-defined loci and at the *TuRB01* locus on linkage group N6 of the *B. napus* genome in 28 DH lines from the N-o-72–8 population: + inheritance of the N-o-9 parental allele; – inheritance of the N-o-1 parental allele; *columns*, DH lines; *rows*, loci. The order of the loci on linkage group N6 is based on the map derived from the segregation data of all 92 DH lines of the N-o-72–8 population (Sharpe et al. 1995). The genetic distances shown are calculated from the segregation data of the 28 DH lines

tially resistant to two isolates (GK 1 and CHN 11) and completely susceptible to 14 isolates (Table 1). The specificity of the resistance in N–o–1 was almost identical to that of the oilseed rape line R4 (Table 1). N–o–1 only differed from R4 with respect to its reaction with GK 1; all of the R4 plants inoculated with GK 1 were infected, whereas only three of the five N–o–1 plants were infected (Table 1). This non-uniform infection by GK 1 was also observed in the DH lines of the N-o–72–8 population that carried the *TuRB01* gene, those lines that lacked the *TuRB01* gene were completely sensitive to GK 1 (Table 2), suggesting that this partial resistance was associated, perhaps by linkage, with the *TuRB01* gene. The pathotype 1 isolate, CHN 11, appeared to mutate to pathotype 3 in that it infected systemically all the R4 plants, some of the N-o-1 plants (Table 1), and some of the plants in the DH lines of the N-o-72–8 population that had the *TuRB01* gene (Table 2).

Additional resistance genes

Some lines not carrying the *TuRB01* gene were partially (or totally) resistant to TuMV isolates CHN 1, JPN 1 and JPN 2 (Tables 1 and 2) suggesting the existence of an additional resistance gene. The resistance was quantitative

Table 2 Phenotypes of the interactions between six isolates of TuMV and DH lines from the N-o-72–8 population

Virus isolate (European pathotype) [original strain designation]	N–o–72–8–									
	48	69	126	152	241	12	54	77	137	178
UK 1(1)	0/5	0/5	0/4	0/5	0/5	5/5	5/5	5/5	5/5	5/5
GK 1(9)	4/4[a]	5/5[a]	1/5[a]	5/5[a]	3/5[a]	5/5[a]	5/5[a]	5/5[a]	5/5[a]	5/5[a]
CHN 1(1)[C1]	0/4	0/5	0/5	0/5	0/5	4/5	4/5	0/5	2/5	4/5
CHN 11(1[b])[Tu6]	2/4	0/5	2/5	0/5	1/5	5/5	5/5	5/5	5/5	5/5
JPN 1(7)	0/5	0/5	0/5	0/5	0/5	5/5	5/5	5/5	3/5	4/5
JPN 2(7)	0/4	0/5	0/5	0/5	0/5	2/5	3/5	0/5	0/5	3/5

[a] Only inoculated leaves infected, i.e. no systemic spread
[b] Virus appeared to mutate giving atypical phenotype on the lines containing *TuRB01*

Table 3 Phenotypes of the interactions between TuMV isolates CHN 1, JPN 2 and UK 1 and 22 microspore-derived lines from plant N-72–8 (F_1 plant from a cross between N-o-1 and N-o-9)

Plant line	Number of plants infected systemically/number inoculated with TuMV isolate as determined by ELISA:		
	UK 1	CHN 1	JPN 2
N-o-72–8-4	5/5	9/10(+1)[a]	0/10
–8	5/5	1/10	0/10
–24	5/5	0/10	0/10
–31	5/5	4/10	0/10
–34	4/4	3/10	0/9
–53	5/5	0/10	9/10
–55	5/5	0/10	0/10
–67	5/5	4/10	3/10
–77	5/5	3/10(+2)	0/10
–78	5/5	2/10(+2)	1/10
–82	5/5	4/10(+6)	0/10
–83	5/5	1/10	0/10
–84	5/5	1/10	1/10
–89	5/5	5/10(+1)	0/10
–90	5/5	2/10	0/10
–101	5/5	3/10	0/10
–115	5/5	6/10(+2)	0/10
–116	5/5	7/10(+1)	0/10
–136	5/5	9/10	0/10
–137	5/5	5/10(+1)	0/10
–147	5/5	0/10	0/10
–191	5/5	10/10	5/10(+2)
S6	5/5	5/5	5/5
R4	0/5	0/5	0/5
S1	5/5	0/5	0/5
165	0/5	0/5	0/5

[a] Numbers in brackets refer to plants where virus symptoms were seen but virus was not detected by ELISA

rather than absolute and was more effective against JPN 2 than either CHN 1 or JPN 1. Segregation for this partial resistance was again observed when an increased number of DH lines from the N–o–72–8 population (all lacking the *TuRB01* gene) were inoculated with the CHN 1 and JPN 2 isolates of TuMV (Table 3). Some of the 22 lines exhibited different segregation patterns for quantitative resistance to TuMV CHN 1 compared with that for quantitative resistance to TuMV JPN 2. This suggested that different genes might be involved, although the experiment was somewhat compromised by the low levels of infection achieved by JPN 2.

Mapmaker QTL (Lincoln et al. 1992) was used to probe for the existence of a gene controlling quantitative resistance to CHN 1 using existing genetic marker data, and the number of plants susceptible to CHN 1 (out of the ten plants tested) as a quantitative measure of susceptibility for each of the 22 DH lines tested. This analysis identified a locus (*TuRB02*) on the lower part of the C-genome linkage group N14, flanked by pW133a and pR113bNM (Fig. 1 in Sharpe et al. 1995), with a LOD score of 2.4 for association with the trait. While the LOD score was too low to give complete confidence in the existence of a resistance gene, the locus appeared to have an appreciable effect. In the eight DH lines where the whole interval was derived from the N-o-9 parent, the mean number of infected plants was 1.5 out of 10. In the seven DH lines where the corresponding interval was derived from the N-o-1 parent, the mean number of infected plants was 6 out of 10. Confirmation of the effect of *TuRB02* will await the analysis of an increased number of lines from the N-o-72–8 population lacking *TuRB01*.

Discussion

Visual inspection of symptoms following inoculation of *B. napus* with TuMV UK 1 proved an accurate assay of

phenotype (confirmed by ELISA) because of the severe infection induced in susceptible individuals and the absence of symptoms in resistant individuals. The segregation of the resistance phenotype was consistent with a single dominant resistance gene, *TuMV RESISTANCE IN BRASSICA 01* (*TuRB01*), mapping to a locus on linkage group N6 of *B. napus* (Sharpe et al. 1995). *TuRB01* is the first gene for resistance to a virus to be mapped in a *Brassica* species. TuMV resistance in the oilseed rape variety "Rafal" (Walsh 1989), with a very similar specificity to that of *TuRB01*, might result from the same gene. The "Rafal" resistance was classified as immunity based on the definition of Cooper and Jones (1983) and the inability to detect virus in inoculated leaves using a range of techniques. It is possible that *TuRB01* induces an extreme form of hypersensitivity where single infected cells are killed (localising infection to these cells and preventing spread to adjacent cells) or operational immunity where cell-to-cell movement is impaired (Arroyo et al. 1996). Further research is required to thoroughly define the nature of resistance mediated by *TuRB01*.

The *TuRB01* gene was only effective against one of the five American/Taiwanese TuMV pathotypes, one of the seven Chinese strain types and two of the five European pathotypes. TuMV pathotype 1 (against which *TuRB01* is effective) is the most abundant pathotype in Europe although pathotypes 3 and 4 (both of which overcome *TuRB01*) are also common (Jenner and Walsh 1996). Widespread deployment of *TuRB01* in *Brassica* cultivars would obviously select for resistance-breaking isolates of TuMV where they exist alongside isolates unable to overcome the resistance. In an earlier study (Jenner and Walsh 1996), the type pathotype 1 isolate of TuMV, UK 1, infected a proportion of plants of the cultivar Westar, which possesses *TuRB01*. When virus was isolated from these plants and inoculated to plants of the line R4, all were infected. The coat-protein coding region of this variant (UK 1M) was subsequently sequenced and found to differ from UK 1 by a single nucleotide (Lehmann et al. 1997). This suggests very strongly that UK 1M was a mutant form of the UK 1 isolate. Jenner and Walsh (1996) also observed the infection by 16 pathotype 1 isolates (including CHN 11) in a proportion of what should have been resistant R4 plants. When virus was subsequently recovered from these plants, their identity confirmed as TuMV and inoculated to further R4 plants, these all became infected. The apparent propensity of TuMV isolates to mutate and overcome the *TuRB01* resistance, as also suggested by CHN 11 in this study, is another potential problem in utilising the gene in the development of resistant crop varieties. These mutant TuMV viruses could be particularly damaging because the new/altered phenotype was necrotic with severe symptoms often leading to plant death. However, it is impossible to predict whether such mutations would be common in vivo. In tests where the resistance gene in "Rafal" was challenged by aphid-inoculated TuMV, no such mutations were observed (Walsh 1989). A number of extreme forms of resistance to TuMV have been identified in swede forms of *B. napus* (Tomlinson and Ward 1982; Shattuck and Stobbs 1987) and in *B. rapa* (Provvidenti 1980; Suh et al. 1995). Genetic mapping of the individual resistance genes carried by these cultivars and the definition of their precise resistance profiles would make possible the design of genotypes likely to exhibit durable resistance to TuMV and the development of such genotypes using marker-assisted breeding techniques.

No extreme forms of resistance to TuMV have been identified in *B. oleracea*, although quantitative resistance has been reported on a number of occasions (Pound et al. 1965; Pink and Walkey 1988; Walkey and Pink 1988). The putative resistance gene *TuRB02* on the C-genome linkage group N14 might represent one of the loci controlling quantitative resistance in *B. oleracea* and *B. napus*. Genes for extreme forms of resistance might not exist in the *Brassica* C-genome and the damage caused to *B. oleracea* crops by TuMV makes the introgression of genes for resistance to TuMV from *B. rapa* into *B. oleracea* desirable. Marker-assisted selection will accelerate this intergenomic gene transfer (Lydiate et al. 1995) and research on the mapping of a range of genes for resistance to TuMV in *B. rapa* has already begun.

Circumstantial evidence derived from the interaction between *TuRB01* and the sequenced isolates of TuMV (Nakashima et al. 1991; Nicolas and Laliberté 1992; Sano et al. 1992) supports the hypothesis that it interacts with the coat protein coding region of the TuMV genome (Lehmann et al. 1997). Cloning *TuRB01* and identifying the gene product would increase the understanding of virus recognition and the processes defining susceptibility and resistance.

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References

Arroyo R, Soto MJ, Martinez-Zapater JM, Ponz F (1996) Impaired cell-to-cell movement of potato virus Y in pepper plants carrying the y^a ($pr2^1$) resistance gene. Mol Plant-Microbe Interact 9:314–318

Choi CW, Ryu KH, Choi SR, Park WM, Yoon KE (1992) Mixed infection of turnip mosaic virus and cucumber mosaic virus identified from vegetables in the Daekwalleong area. Korean J Plant Pathol Newslett 3:85–86

Cooper JI, Jones AT (1983) Responses of plants to viruses: proposals for the use of terms. Phytopathology 73:127–128

Edwardson JR, Christie RG (1991) The potyvirus group. Volumes I–IV, Fla Agric Exp Stn Tech Bull Monograph 16

Evans IR, MacNeil BH (1983) Virus disease of rutabagas (turnips). Ontario Ministry of Agriculture and Food Factsheet 73–061

Fraser RSS (1990) The genetics of resistance to plant viruses. Annu Rev Phytopathol 28:179–200

Fray MJ, Puangsomlee P, Goodrich J, Coupland G, Evans EJ, Arthur AE, Lydiate DJ (1997) The genetics of stamenoid petals in oilseed rape (*Brassica napus*) and a candidate gene from *Arabidopsis thaliana*. Theor Appl Genet 94:731–736

Green SK, Deng TC (1985) Turnip mosaic virus strains in cruciferous hosts in Taiwan. Plant Dis 69:28–31

Hardwick NV, Davies JML, Wright DM (1994) The incidence of three virus diseases of winter oilseed rape in England and Wales in the 1991/92 and 1992/93 growing seasons. Plant Pathol 43:1045–1049

Jenner CE, Walsh JA (1996) Pathotypic variation in turnip mosaic virus with special reference to European isolates. Plant Pathol 45:848–856

Jenner CE, Keane GJ, Jones JE, Walsh JA (1999) Serotypic variation in turnip mosaic virus. Plant Pathol 48:101–108

Lehmann P, Petrzik K, Jenner C, Greenland A, Špak J, Kozubek E, Walsh JA (1997) Nucleotide and amino-acid variation in the coat protein coding region of turnip mosaic virus isolates and possible involvement in the interaction with the brassica resistance gene *TuRB01*. Physiol Mol Plant Pathol 51:195–208

Lincoln S, Daly M, Lander E (1992) Mapping genes controlling quantitative traits with MAPMAKER/QTL 1.1. Whitehead Institute Technical Reports, 2nd edn, Cambridge, Massachusetts

Liu XP, Lu WC, Liu YK, Li JL (1990) A study on TuMV strain differentiation of cruciferous vegetables from ten provinces in China. Chinese Sci Bull 35:1734–1739

Liu XP, Lu WC, Liu YK, Wei SQ, Xu JB, Liu JB, Liu ZR, Zhang HJ, Li JL, Ke GL, Yao WY, Cai YS, Wu, FY, Cao SC, Li YH, Xie SD, Lin BX, Zhang CL (1996) Occurrence and strain differentiation of turnip mosaic potyvirus and sources of resistance in Chinese cabbage in China. Acta Hort 407:431–440

Lydiate D, Dale P, Lagercrantz U, Parkin I, Howell P (1995) Selecting the optimum genetic background for transgenic varieties, with examples from *Brassica*. Euphytica 85:351–358

Nakashima H, Sako N, Joh K, Hori K, Nonaka F (1991) Nucleotide sequences of the coat protein genes of aphid transmissible and non-transmissible isolates of turnip mosaic virus. Ann Phytopathol Soc Jpn 57:549–557

Nicolas O, Laliberté JF (1992) The complete nucleotide sequence of turnip mosaic potyvirus RNA. J Gen Virol 73:2785–2793

Niu X, Leung H, Williams PH (1983) Sources and nature of resistance to downy mildew and turnip mosaic virus in Chinese cabbage. J Am Soc Hort Sci 108:775–778

Parkin I, Magrath R, Keith D, Sharpe A, Mithen R, Lydiate D (1994) Genetics of aliphatic glucosinolates. 2. Hydroxylation of alkenyl glucosinolates in *Brassica napus*. Heredity 72:594–598

Parkin IAP, Sharpe AG, Keith DJ, Lydiate DJ (1995) Identification of the A and C genomes of amphidiploid *Brassica napus* (oilseed rape). Genome 38:1122–1131

Pink DAC, Walkey DGA (1988) The reaction of summer- and autumn-maturing cauliflowers to infection by cauliflower and turnip mosaic viruses. J Hort Sci 63:95–102

Ponz F, Bruening G (1986) Mechanisms of resistance to plant viruses. Annu Rev Phytopathol 24:355–381

Pound GS, Williams PH, Walker JC (1965) Mosaic and yellows resistant inbred cabbage varieties. Wis Agric Exp St Res Bull 259

Provvidenti R (1980) Evaluation of Chinese cabbage cultivars from Japan and the People's Republic of China for resistance to turnip mosaic virus and cauliflower mosaic virus. J Am Soc Hort Sci 105:571–573

Robbins MA, Witsenboer H, Michelmore RW, Laliberté JF, Fortin MG (1994) Genetic mapping of turnip mosaic virus resistance in *Lactuca sativa*. Theor Appl Genet 89:583–589

Sano Y, van der Vlugt R, de Haan P, Takahashi A, Kawakami M, Goldbach R, Kojima M (1992) On the variability of the 3' terminal sequence of the turnip mosaic virus genome. Arch Virol 126:231–238

Sharpe AG, Parkin IAP, Keith DJ, Lydiate DJ (1995) Frequent nonreciprocal translocations in the amphidiploid genome of oilseed rape (*Brassica napus*). Genome 38:1112–1121

Shattuck VI, Stobbs LW (1987) Evaluation of rutabaga cultivars for turnip mosaic virus resistance and the inheritance of resistance. HortScience 22:935–937

Shukla DD, Ward CW, Brunt AA (1994) The Potyviridae. CAB International, Wallingford, UK

Stobbs LW, Shattuck VI, Shelp BJ (1991) Effect of turnip mosaic virus infection on the development, virus titer, glucosinolate concentrations and storability of rutabaga roots. Plant Dis 75:575–579

Suh SK, Green SK, Park HG (1995) Genetics of resistance to five strains of turnip mosaic virus in Chinese cabbage. Euphytica 81:71–77

Tomlinson JA (1987) Epidemiology and control of virus diseases of vegetables. Ann Appl Biol 110:661–681

Tomlinson JA, Ward CM (1982) Selection for immunity in swede (*Brassica napus*) to infection by turnip mosaic virus. Ann Appl Biol 101:43–50

U N (1935) Genome analysis in *Brassica* with special reference to the experimental formation of *B. napus* and peculiar mode of fertilisation. Jpn J Bot 7:389–452

Walkey DGA, Pink DAC (1988) Reactions of white cabbage (*Brassica oleracea* var. *capitata*) to four different strains of turnip mosaic virus. Ann Appl Biol 112:273–284

Walsh JA (1989) Genetic control of immunity to turnip mosaic virus in winter oilseed rape (*Brassica napus* ssp. *oleifera*) and the effect of foreign isolates of the virus. Ann Appl Biol 115:89–99

Yoon JY, Green SK, Opeña, RT (1993) Inheritance of resistance to turnip mosaic virus in Chinese cabbage. Euphytica 69:103–108