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MicroReview

Advances in understanding recessive resistance to plant viruses

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SUMMARY

Recent work carried out to characterize recessive mutations which render experimental hosts non-permissive to viral infection (loss-of-susceptibility mutants) seems to be converging with new data on natural recessive resistance in crop species, and also with functional analyses of virus avirulence determinants. Perhaps the most well known examples are the studies that identified the eukaryotic translation initiation factors 4E(iso) (eIF(iso)4E) and 4E(eIF4E) as the host factors required for potyvirus multiplication within experimental and natural hosts, respectively, and the potyviral genome-linked protein (VPg) as the viral factor that directly interacts with eIF4E to promote potyvirus multiplication. The purpose of this paper is to review the available information on the characterization of loss-of-susceptibility mutants in experimental hosts, natural recessive resistances and virus avirulence factors, and also to comment on possible implications for the design of new sources of sustainable virus resistance.

INTRODUCTION

Plants protect themselves against pathogens using a wide variety of mechanisms, among them the establishment of pre-formed defence barriers and the use of basal defence responses; although the focus of the most extensive studies has been the well-known gene-for-gene resistance response (Dangl and Jones, 2001). This type of resistance depends on the ability of a plant to recognize a pathogen and to quickly mount a range of defensive measures often associated with the development of a hypersensitive response (HR). This recognition process was revealed by analysis of the race or pathovar specific resistances conferred by single dominant resistance genes (R-genes), and was formulated according to the gene-for-gene hypothesis (Flor, 1971). At the biochemical level, recent evidence suggests that the products of R-genes function as 'guards', detecting the activity of the avirulence effectors bound to a host target factor rather than the avirulence factor itself (Axtell and Staskawicz, 2003; MacKey *et al.*, 2003). Several viral R-genes have been characterized to date, and all belong to the nucleotide binding site leucine-rich repeat (NBS-LRR) super family of R-genes (Hammond-Kosak and Parker, 2003).

Research into the genetic resistance to plant pathogenic viruses is also providing important basic information on different plant biological processes. Perhaps the most recent example consists of work carried out to analyse the phenomenon of post-transcriptional gene silencing (PTGS), which, for some research groups, was initially undertaken to understand different aspects related to transgenic or natural resistance to plant viruses (e.g. Covey *et al.*, 1997). Today, it is clear that RNA silencing (PTGS in plants, RNA interference in animals and quelling in fungi) is a fundamental process of the regulation of eukaryotic gene expression (Voinnet, 2002). Both R-mediated disease resistance and RNA silencing have been the subject of many excellent reviews (e.g. Hammond-Kosak and Parker, 2003; Voinnet, 2002; Waterhouse *et al.*, 2001) and will not be covered further here.

In contrast to these two examples, the information available on incompatible interactions between viruses and plants controlled in the host by recessive resistance genes is less in quantity and more disperse. There are two generally accepted hypotheses to explain the mechanisms of recessive resistance (Fraser, 1990, 1999). The first hypothesis proposes that resistance might be the result of a passive mechanism that makes a plant resistant due

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to the lack of a specific host factor required by the virus to complete its cycle, or due to the presence of a mutated version of that factor. According to the second hypothesis, resistance might be the result of an active mechanism, in which the resistant plant produces an inhibitor that interferes with some stage of the virus cycle or contains a factor which recognizes some virus-encoded molecule and therefore switches on a resistance response; susceptibility, in this case, is due to the presence of a repressor of the inhibitor or of a resistance response repressor. Recent results on recessive resistance to potyviruses are better explained in the conceptual framework of the first hypothesis (Lellis *et al.*, 2002; Nicaise *et al.*, 2003; Ruffel *et al.*, 2002), whereas the second scenario has only been found to apply to plant–fungal interactions, such as that controlled by the *Mlo* gene in barley (Buschges *et al.*, 1997). Importantly, work carried out to characterize loss-of-susceptibility mutants obtained through mutagenesis in experimental hosts is converging with work on natural recessive resistance in crop species, and also with functional analyses of virus avirulence determinants. Moreover, non-host resistance (i.e. resistance at species level) probably shares many basic mechanisms with the cultivar resistance conferred by recessive genes. Here, we review some of these latter aspects and their implications for the identification of new sources of resistance.

LOSS-OF-SUSCEPTIBILITY MUTANTS IN EXPERIMENTAL HOSTS

Viruses depend on the host biochemical machinery to complete their biological cycle. Thus, the successful infection of a plant by a virus requires a series of compatible interactions between host and viral factors along a complex multi-step process that includes the expression and replication of the viral genome, cell-to-cell movement and long distance translocation through the plant vascular system (Carrington *et al.*, 1996; Maule *et al.*, 2002). Characterization of some of these host factors has been achieved through the analysis of large collections of mutagenized hosts and the consequent identification of several genes required for virus multiplication (Table 1). Perhaps one of the most powerful approaches has been that of Ahlquist and co-workers, who used yeast genetics to identify the host factors required for *Brome mosaic virus* (BMV) gene expression and replication. This was possible due to the unique ability of BMV to direct RNA replication, subgenomic mRNA synthesis and encapsidation in *Saccharomyces cerevisiae,* recapitulating all known features of BMV replication in plant cells (Janda and Ahlquist, 1993; Quadt *et al.*, 1995). Thus, the host factors required for specific translation of genomic RNAs (Noueiry *et al.*, 2000), coordination of RNA translation and replication (Diez *et al.*, 2000; Noueiry *et al.*, 2003), initiation of negative strand RNA synthesis (Tomita *et al.*, 2003) and RNA replication (Ishikawa *et al.*, 1997; Lee *et al.*, 2001) were identified by this research group (reviewed in Ahlquist *et al.*, 2003) (Table 1).

In *Arabidopsis thaliana*, host factors required for virus gene expression or replication (i.e. factors that affect virus multiplication in protoplasts) have also been identified through the screening of mutants (Table 1). Thus, TOM1 (and its homologue TOM3) and TOM2A have been shown to be required for efficient *Tobacco mosaic virus* (TMV) multiplication in *Arabidopsis* protoplasts. They are host integral membrane proteins which interact with each other and also with viral replication factors (Hagiwara *et al.*, 2003; Ishikawa *et al.*, 1991, 1993; Ohshima *et al.*, 1998; Tsujimoto *et al.*, 2003; Yamanaka *et al.*, 2002). Following a similar approach, the eukaryotic translation initiation factor 4E(iso) (eIF(iso)4E) has been shown to be necessary for efficient multiplication of the potyviruses *Turnip mosaic virus* (TuMV) and *Tobacco etch virus* (TEV) in single cells (Duprat *et al.*, 2002; Lellis *et al.*, 2002; Whitham *et al.*, 1999). In addition, several *Arabidopsis* mutants in which virus movement is restricted have been identified: these include *cum1-1* and *cum2-1*, which affect the local spread of *Cucumber mosaic virus* (CMV) (Yoshii *et al.*, 1998a) and that of CMV and *Turnip crinkle virus* (TCV) (Yoshii *et al.*, 1998b), respectively, and *vsm1*, in which the systemic movement of a tobamovirus is specifically restricted (Lartey *et al.*, 1998) (Table 1). The cloning and characterization of these latter genes has the potential to provide very useful information regarding the mechanisms governing virus movement within their hosts.

NATURAL RECESSIVE RESISTANCE GENES

The screening of *Arabidopsis* ecotypes for differential susceptibility to viruses has also been carried out in a number of laboratories. Such studies have contributed to the identification of several natural recessive genes that affect virus multiplication in the plant (Table 1). Again, the cloning and characterization of these genes is an exciting area that can provide important information on mechanisms governing host–virus interactions. However, most of the natural recessive resistances to viruses identified thus far are from crop species. Interestingly, recessive resistances seem to be more frequent for plant viruses than for other plant pathogens, for which resistances appear to be predominantly inherited as monogenic dominant characters (Fraser, 1990). Moreover, recessive resistances are more frequent for potyviruses than for viruses of other families (Table 2): 63.8% of the examples listed in Table 2 correspond to potyviruses. This may simply reflect the relative importance of the viruses of this family or, perhaps, other specific peculiarities of the potyvirus biology.

Unfortunately, basic research on the control and mechanisms underlying recessive resistance in crop species often did not go as far as it might. In some cases, even the genetics of the resistance remains unclear. The expression of the resistance was analysed in some detail in only one-quarter of the crop/virus combinations listed in Table 2. For most of the cases in which this aspect was studied, the resistances seemed to be active at the single cell

Table 1 Recessive resistances in experimental hosts.

Host	Virus	Gene	Phenotype of mutant	Function of protein	Selected references
Saccharomyces cerevisiae	*Brome mosaic virus*	ded1	Mutations block BMV replication without inhibiting cell growth. This is associated with selective inhibition of RNA2 translation.	RNA helicase required for translation initiation on all yeast mRNAs. The RNA2 5′ non-coding region is required for this inhibition.	Noueiry et al. (2000)
		lsm1	Mutant defective in coordination of translation and replication. Strongly reduced translation of genomic RNAs destined for replication.	Forms part of a complex that facilitates deadenylation-dependent mRNA turnover. Contains the conserved Sm motif. Required for efficient 1a recruitment of viral RNAs.	Diez *et al.* (2000)
		ydj1	Mutant shows no initiation of negative-strand RNA synthesis.	Chaperone involved in forming BMV replication complexes, possibly by directing 2a polymerase folding or a step required for RNA synthesis.	Tomita *et al.* (2003)
		ole1	Mutation blocks BMV RNA replication in an early step due to the reduction in unsaturated fatty acid levels in membranes.	β fatty acid desaturase, key enzyme for converting saturated to unsaturated fatty acids.	Lee *et al.* (2001)
		mab1-1 *mab2-1* *mab3-1*	Mutants inhibit accumulation of positive and negative-strand RNA3 and subgenomic mRNA.		Ishikawa *et al.* (1997)
Arabidopsis thaliana	*Tobacco mosaic virus*	*tom1* *(tom3)* *(thh1)*	Mutation affects amplification of TMV-related RNAs in a single cell (not valid for CMV or TCV).	Transmembrane protein localized in tonoplast. It interacts with helicase domain of tobamovirus-encoded replication proteins and is an essential constituent of the tobamoviral replication complex.	Ishikawa *et al.* (1991) Ishikawa *et al.* (1993) Yamanaka *et al.* (2000) Yamanaka *et al.* (2002) Hagiwara *et al.* (2003)
		tom2A	Mutation affects accumulation of TMV-related RNAs in protoplasts in a tobamovirus specific manner.	Transmembrane protein localized in tonoplast, interacts with TOM1 and facilitates formation of tobamoviral RNA replication complex.	Ohshima *et al.* (1998) Tsujimoto *et al.* (2003)
	Cucumber mosaic virus *Turnip crinkle virus*	*cum1-1*	Mutation affects spreading of CMV (not TCV and TMV) within an infected leaf, possibly due to the cell-to-cell movement of CMV in a virus specific manner.		Yoshii *et al.* (1998a)
		cum2-1	Mutation affects the local spreading and cell- to-cell movement of both CMV and TCV.		Yoshii *et al.* (1998b)
	Turnip vein clearing virus	*vsm1*	Mutation affects systemic movement of TVCV.		Lartey *et al.* (1998)
	Tobacco etch potyvirus *Turnip mosaic potyvirus*	*lsp1*	Mutants are defective in supporting TuMV and TEV genome expression and/or replication.	Translation factor eIF(iso)4E with cap-binding activity. It interacts with VPg.	Lellis *et al.* (2002) Whitham *et al.* (1999) Wittmann *et al.* (1997)
	Lettuce mosaic potyvirus	?	Complete resistance to all LMV isolates of the Cape Verde islands.		Revers *et al.* (2003)
	Beet curly top virus	?	Resistance to BCTV.		Lee *et al.* (1994)
	Tobacco mosaic virus	?	Resistance to TMV.		Dardick *et al.* (2000)

Table 2 Recessive resistances in crop species.

Host	Virus (Genus; Family)	Gene*	Expression†	Selected references
Apium graveolens	*Celery mosaic virus* (*Potyvirus*; *Potyviridae*)	*cmv*	Apical leaves	D'Antonio *et al.* (2001)
Arachis hipogaea	*Groundnut rosette virus* (*Umbravirus*)	Two genes	Escape to infection/ No symptoms	Olorunju *et al.* (1992)
Brassica campestris	*Turnip mosaic virus* (*Potyvirus*; *Potyviridae*)	Two genes	Apical leaves	Yoon *et al.* (1993)
Brassica rapa *PI 418957c* and *Jin G 55*	*Turnip mosaic virus* (*Potyvirus*; *Potyviridae*)	Monogenic	Apical leaves	Hughes *et al.* (2002)
Capsicum annum	*Potato virus Y* (*Potyvirus*; *Potyviridae*)	*pvr2*^{1}, *pvr2*^{2}, *pvr3*, *pvr5*	Inoculated leaves; apical leaves	Dogimont *et al.* (1996); Ruffel *et al.* (2002)
	Cucumber mosaic virus (*Cucumovirus*; *Bromoviridae*)	At least two major genes	Escape to infection	Grube *et al.* (2000)
	Pepper mottle virus (*Potyvirus*; *Potyviridae*)	*pvr3*	Apical leaves	Murphy and Kyle (1995)
	Pepper veinal mottle virus (*Potyvirus*; *Potyviridae*)	*pvr2*^{2}, *pvr6*	Apical leaves	Caranta *et al.* (1996)
	Tobacco etch virus (*Potyvirus*; *Potyviridae*)	*pvr2*^{2}	Protoplasts	Deom *et al.* (1997)
Capsicum chinense	*Pepper mottle virus* (*Potyvirus*; *Potyviridae*)	*pvr1*	Protoplasts	Murphy *et al.* (1998)
	Potato virus Y (*Potyvirus*; *Potyviridae*)	*pvr1*	Ioculated leaves	Boiteux *et al.* (1996)
	Tobacco etch virus (*Potyvirus*; *Potyviridae*)	*pvr1*	Protoplasts	Murphy *et al.* (1998)
Capsicum frutescens	*Cucumber mosaic virus* (*Cucumovirus*; *Bromoviridae*)	At least two major genes	Escape to infection	Grube *et al.* (2000)
Citrullus lanatus	*Zucchini yellow mosaic virus* (*Potyvirus*; *Potyviridae*)	Monogenic	Apical leaves	Provvidenti (1991a)
Cucumis melo	*Cucumber green mottle mosaic virus* (*Tobamovirus*)	Polygenic	No symptoms	Rajamony *et al.* (1990)
	Cucurbit aphid borne yellows virus (*Polerovirus*; *Luteoviridae*)	*cab-1*, *cab-2*	Apical leaves	Dogimont *et al.* (1997)
	Melon necrotic spot virus (*Carmovirus*; *Tombusviridae*)	*nsv*	Protoplasts	Díaz *et al.* (2004)
Cucumis sativus	*Papaya ring spot virus* (*Potyvirus*; *Potyviridae*)	Monogenic	Reduced accumulation	Grumet *et al.* (2000)
	Watermelon mosaic virus (*Potyvirus*; *Potyviridae*)	*wmv-2*	No symptoms	Wai and Grumet (1995)
	Moroccan watermelon mosaic virus (*Potyvirus*; *Potyviridae*)	*mwm*	No symptoms	Kabelka and Grumet (1997)
	Zucchini yellow fleck virus (*Potyvirus*; *Potyviridae*)	*zyf*	Apical leaves	Gilbert-Albertini *et al.* (1995)
	Zucchini yellow mosaic virus (*Potyvirus*; *Potyviridae*)	*zym*	Reduced accumulation	Ullah and Grumet (2002)
Cucurbita moschata cv. *Nigerian*	*Papaya ring spot virus* (*Potyvirus*; *Potyviridae*)	*prv*	No symptoms	Brown *et al.* (2003)
Glycine max	*Cowpea chlorotic mottle virus* (*Bromovirus*; *Bromoviridae*)	Two genes	Reduced accumulation	Goodrick *et al.* (1991)
Hordeum vulgare	*Barley mild mosaic virus* (*Bymovirus*; *Potyviridae*)	*rym1* to *rym12*	?	Kuhne *et al.* (2003)
	Barley yellow mosaic virus (*Bymovirus*; *Potyviridae*)	*rym1* to *rym12*	?	Kanyuka *et al.* (2003)

Table 2 *continued.*

Host	Virus (Genus; Family)	Gene*	Expression†	Selected references
Lactuca sativa	*Beet Western yellows virus* (*Polerovirus*; *Luteoviridae*)	*bwy*	Reduced accumulation	Pink *et al.* (1991)
	Bidens mottle virus (*Potyvirus*; *Potyviridae*)	?	?	Provvidenti and Hampton (1992)
	Lettuce mosaic virus (*Potyvirus*; *Potyviridae*)	*mol1, mol2*	Reduced accumulation	Nicaise *et al.* (2003)
Lycopersicon esculentum	*Peru tomato virus* (*Potyvirus*; *Potyviridae*)	?	?	Provvidenti and Hampton (1992)
	Tomato spotted wilt virus (*Tospovirus*; *Bunyaviridae*)	?	?	Maluf *et al.* (1991)
Lycopersicon hirsutum	*Tomato yellow leaf curl virus* (*Begomovirus*; *Geminiviridae*)	Two to three genes	No symptoms	Vidavsky and Czosnek (1998)
	Tobacco etch virus (*Potyvirus*; *Potyviridae*)	*pot-1*	Inoculated leaves	Parrella *et al.* (2002)
	Potato virus Y (*Potyvirus*; *Potyviridae*)	*pot-1*	Inoculated leaves	Parrella *et al.* (2002)
Manihot esculenta	*African cassava mosaic virus* (*Begomovirus*; *Geminiviridae*)	?	Escape to infection	Legg and Thresh (2000)
Manihot glaziovii	*African cassava mosaic virus* (*Begomovirus*; *Geminiviridae*)	Polygenic	Reduced accumulation	Legg and Thresh (2000)
Matthiola incana	*Turnip mosaic virus* (*Potyvirus*; *Potyviridae*)	?	?	Provvidenti and Hampton (1992)
Nicotiana tabacum	*Potato virus Y* (*Potyvirus*; *Potyviridae*)	*va*	Inoculated leaves	Masuta *et al.* (1999)
	Tobacco etch virus (*Potyvirus*; *Potyviridae*)	Two genes	Apical leaves	Schaad and Carrington (1996)
	Tobacco vein mottling virus (*Potyvirus*; *Potyviridae*)	*va*	Inoculated leaves	Nicolas *et al.* (1997)
Oryza glaberrima	*Rice yellow mottle virus* (*Sobemovirus*)	?	Inoculated leaves	Albar *et al.* (2003)
Oryza sativa	*Rice yellow mottle virus* (*Sobemovirus*)	Monogenic	Inoculated leaves	Albar *et al.* (2003)
Oryza sativa TKM6 *O. sativa* Utri Merah	*Rice tungro spherical virus* (*Waikavirus*; *Sequiviridae*)	*tsv-1*/*tsv-1*, *tsv-2*	Apical leaves/ Escape to infection	Azzam *et al.* (2000)
Phaseolus vulgaris	*Bean common mosaic virus* (*Potyvirus*; *Potyviridae*)	*bc-l*, *bc-1*^{2}, *bc-2*, *bc-2*^{2}, *bc-3*, *bc-u*	?	Miklas *et al.* (2000)
	Bean yellow mosaic virus (*Potyvirus*; *Potyviridae*)	*cyv*	?	Park and Tu (1991)
	Clover yellow vein virus (*Potyvirus*; *Potyviridae*)	?	Protoplasts	Sato *et al.* (2003)
Pisum sativum	*Bean common mosaic virus* (*Potyvirus*; *Potyviridae*)	*bcm*	Apical leaves	Provvidenti (1991b)
	Bean leafroll virus (*Enamovirus*; *Luteoviridae*)	*lr*	No symptoms	Baggett and Hampton (1991)
	Bean yellow mosaic virus (*Potyvirus*; *Potyviridae*)	*mo*	Apical leaves	Schroeder and Provvidenti (1971)
	Clover yellow vein virus (*Potyvirus*; *Potyviridae*)	?	?	Provvidenti and Hampton (1992)
	Passionfruit woodiness virus (*Potyvirus*; *Potyviridae*)	*pwv*	?	Provvidenti and Niblett (1994)
	Watermelon mosaic virus (*Potyvirus*; *Potyviridae*)	*mo*	Apical leaves	Schroeder and Provvidenti (1971)
	White lupin mosaic virus	*wlv*	Apical leaves	Provvidenti and Hampton (1993)

Table 2 *continued.*

Host	Virus (Genus; Family)	Gene*	Expression†	Selected references
	(*Potyvirus*; *Potyviridae*)			
	Pea seedborne mosaic virus (*Potyvirus*; *Potyviridae*)	*sbm1*, *sbm2*, *sbm3*, *sbm4*	Protoplasts	Johansen *et al.* (2001)
Rubus idaeus	*Raspberry bushy dwarf virus* (*Idaeovirus*)	?	?	Knight and Barbara (1981)
Vicia faba	*Bean yellow mosaic virus* (*Potyvirus*; *Potyviridae*)	?	?	Provvidenti and Hampton (1992)
Vigna mungo PDM 116	*Bean yellow mosaic virus* (*Potyvirus*; *Potyviridae*)	Two genes	No symptoms	Pal *et al.* (1991)
Vigna radiata	*Mungbean yellow mosaic virus* (*Begomovirus*; *Geminiviridae*)	?	?	Malik *et al.* (1986)
Vigna unguiculata	*Blackeye cowpea mosaic virus* (*Potyvirus*; *Potyviridae*)	?	?	Provvidenti and Hampton (1992)
	Cowpea severe mosaic virus (*Comovirus*; *Comoviridae*)	Three genes	No symptoms	Umaharan *et al.* (1997)

*In cases labelled with a question mark, the genetic control of the resistance has not been clarified. In other cases, resistance alleles have not been named.
†In cases labelled with a question mark, the expression of the resistance has not been analysed. For the rest of the cases, the methodology for studying this aspect has been quite variable. We have categorized the following: Protoplasts = virus accumulation was not detected in inoculated protoplasts; inoculated leaves = virus accumulation was not detected either in inoculated or in non-inoculated leaves; apical leaves = virus accumulation was not detected in non-inoculated leaves; reduced accumulation = virus accumulation was reduced compared to susceptible controls; escape to infection = a smaller proportion of plants were infected compared to susceptible controls; no symptoms = there was no symptom expression even if virus accumulated at normal levels.

level, and only in one case did the resistance appear to act during the cell-to-cell movement of the virus (Nicolas *et al.*, 1997; Table 2). To our knowledge, the characterization of recessive genes conferring resistance to viruses in crop species has only been reported for pepper and lettuce. Interestingly, these resistances are against two different potyviruses, but they were shown to be controlled by the same host factor, the eukaryotic initiation factor 4E (eIF4E) (Nicaise *et al.*, 2003; Ruffel *et al.*, 2002). In both cases, *eIF4E* was identified as the resistance gene by using a candidate gene approach in which *eIF4E* was chosen as a candidate based on previous results obtained with the *Arabidopsis*/TEV-TuMV systems (see above), and also based on a number of results related to the properties of the viral genome-linked protein, VPg, which was shown to act as a potyviral avirulence factor for several host/potyvirus combinations (see below).

AVIRULENCE DETERMINANTS

The durability of resistance deployed in the field may be compromised by the appearance of resistance-breaking strains. This fact, however, may be of great value in research: genetic analyses comparing resistance-breaking and non-resistance-breaking viral strains have produced interesting results regarding the identification of determinants for pathogen avirulence and mechanisms of resistance (Harrison, 2002). Resistance-breaking strains have been identified in around one-third of the crop/virus combinations listed in Table 2, but the avirulence determinants have been characterized in only half of them, perhaps because of the limited availability of infectious cDNA clones for the corresponding viruses. Notably, seven of the nine avirulence determinants already characterized correspond to potyviral VPgs (Keller *et al.*, 1998; Kuhne *et al.*, 2003; Nicolas *et al.*, 1997; Sato *et al.*, 2003; Schaad and Carrington, 1996), one corresponds to the P3-6K1 potyviral genes (Johansen *et al.*, 2001), and only one avirulence determinant has been characterized for a non-potyvirus, *Melon necrotic spot virus* (MNSV) (Díaz *et al.*, 2004). Interestingly, in this last case the avirulence determinant probably consists of an RNA sequence on its own (the MNSV 3′-untranslated region) and the resistance of melon to MNSV appears to depend on the same viral genetic determinant as the non-host resistances of *Nicotiana benthamiana* and *Gomphrena globosa* (Fig. 1) (Díaz *et al.*, 2004). As regards the involvement of the potyviral VPg as the avirulence determinant in several crop/potyvirus combinations, TEV and TuMV VPgs have been shown to interact with eIF4E and eIF(iso)4E factors from different hosts. Additionally, it has been shown that these VPg–eIF4E interactions up-regulate genome amplification and are necessary for infectivity (Leonard *et al.*, 2000; Schaad *et al.*, 2000; Wittmann *et al.*, 1997). These results prompted use of the candidate gene approach mentioned above to identify eIF4E from lettuce and pepper as the factor controlling recessive resistances to potyviruses in these hosts, and illustrate very well how research on the 'virus side' can provide valuable

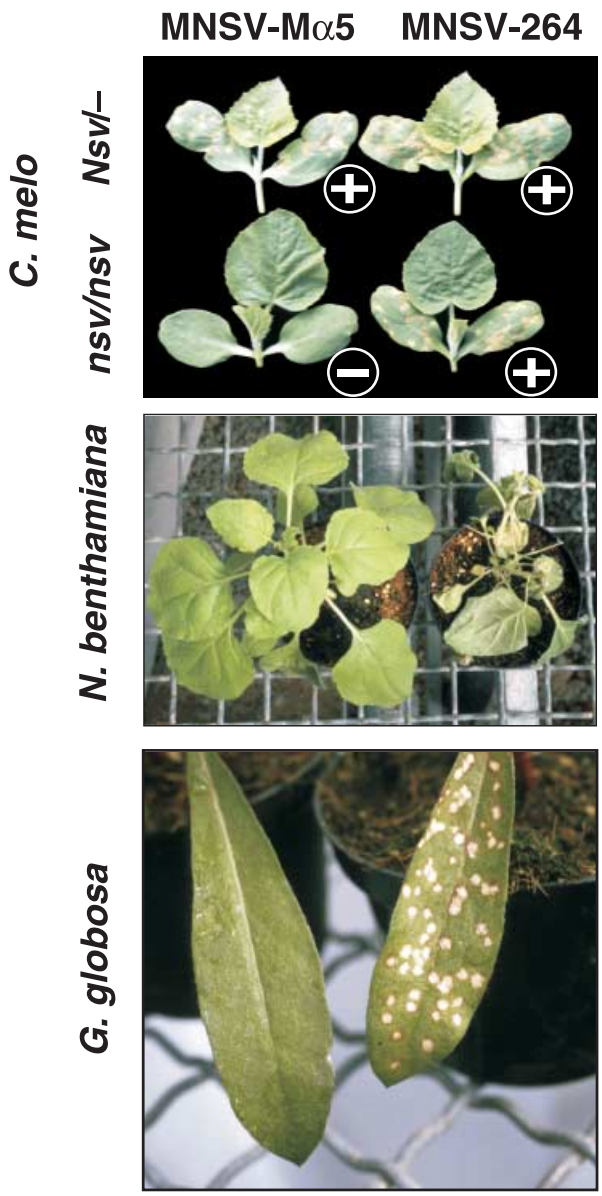

Fig. 1 A *Melon necrotic spot virus* (MNSV) strain is able to overcome the resistance in melon and non-host plants. Seedlings of the melon cultivars Planters Jumbo (*nsv/nsv* genotype) and PMR-45 (*Nsv/–* genotype) as well as *Nicotiana benthamiana* and *Gomphrena globosa* plants were inoculated with two different viral strains, MNSV-264 and MNSV-Mα5. MNSV-264 is able to overcome the melon resistance conferred by *nsv* (+ indicates infection, – indicates no infection) and also the non-host resistance of *N. benthamiana* and *G. globosa*, whereas MNSV-Mα5 is not. Interestingly, the avirulence determinants for both types of resistance seem to reside in the same region of the MNSV genome (Díaz *et al.*, 2004).

information relevant to the molecular characterization of recessive resistance genes. The identification of eIF4E as the host factor controlling recessive resistance to different potyviruses in two different hosts, together with the high frequency of identification of VPg as an avirulence determinant, point to the possibility that the same host factor might control recessive resistance to potyviruses in other plant species.

NON-HOST RESISTANCE

One intriguing phenomenon is that most plant species are resistant to the majority of plant viruses. Resistance at the plant species level is termed non-host resistance. As in the case of recessive resistance described above, passive mechanisms could govern non-host resistance (Dawson and Hilf, 1992; Fraser, 1990). Passive resistance could operate if a plant species lacks or contains a modified version of a factor that is essential for the viral infection cycle. If the modified gene leads to immunity to viruses without affecting the fitness of the plant, then it can be predicted that the new allele will be selected at the species level, leading to non-host resistance. The case of MNSV/*N. benthamiana* and *G. globosa*, described above (Fig. 1), might be used as an example to illustrate this hypothesis. Alternatively, the virus could have evolved so that the pathogenicity factors were non-functional in non-host plants. Two examples involving TMV and *Cauliflower mosaic virus* (CaMV) provide precedents for this proposed pattern of evolution. It was shown that isolates of TMV that developed the ability to overcome different resistance genes in *Capsicum* have lost their ability to multiply in tomato and tobacco, hosts that were the original source of infection (Fraser, 1990). The lack of a specific function in the virus was also described as governing the host range for CaMV (Wintermantel *et al.*, 1993): the CaMV isolate D4 systemically infects solanaceous and cruciferous species, whereas the CaMV isolate CM1841 is unable to systemically infect any solanaceous host but can infect cruciferous species. Transgenic *Nicotiana bigelovii* expressing the D4 gene VI product extended the host range of CM1841 to solanaceous species (Schoelz and Wintermantel, 1993; Wintermantel *et al.*, 1993). Very probably, understanding the mechanisms underlying non-host resistance will be crucial for the development of stable virus resistant crops.

CONCLUSIONS AND FUTURE CHALLENGES

Genetic studies using experimental model hosts, such as yeast and *Arabidopsis,* are providing fundamental information on how viruses intimately interact with their hosts. The potential of these models is very high and their exploitation is just beginning. The range of viruses considered will probably broaden, and new mutants will be identified through the screening of germplasm collections or large numbers of mutants. For example, the recent development of an experimental system based on yeast as a host for the replication of tombusvirus satellite RNAs (Panavas and Nagy, 2003) will surely render important results in the future. Once genes responsible for these mutations are identified, research groups will face biological questions that will have to be analysed

using biochemical and cellular biology approaches. The availability of extensive information on the determinants of functions coded by viral genomes is critical to providing complementary information that would be highly valuable regarding these last aspects. On the other hand, the potential to extract basic information by using crop species as experimental hosts should not be under-estimated. One of the simplest reasons for this is that a significant number of monogenic recessive resistances have already been identified in crop species and some of these resistances have been characterized, at least partially. Another reason for this is that nature often produces interesting tools for the dissection of the mechanisms controlling these resistances, such as resistance-breaking virus strains. Three groups of crop/virus combinations which may deserve differential attention can be identified: (i) resistances to potyviruses in which the viral VPg appears to be the avirulence factor and/or that are expressed at the single cell level; in such cases, a candidate gene approach to analysing the possible involvement of eIF4E in the resistance may be applicable, (ii) resistances to potyviruses for which an avirulence factor different from the viral VPg has been identified and/or that are not expressed at the single-cell level, and (iii) resistances to viruses other than potyviruses. Particularly, in those cases where the resistance gene has been genetically characterized and positioned in a genetic map, an alternative candidate approach may be used in order to identify genes co-segregating with the resistance gene. Genes previously identified as host factors in other systems (e.g. *Arabidopsis* or yeast) could be candidates for undertaking such an approach, as well as genes shown to respond to viral infection (Aranda *et al.*, 1996; Whitham *et al.*, 2003).

Finally, but not less importantly, we must consider the practical implications of research in this area. For example, studies using crop species as experimental hosts may provide information relevant to the likelihood of appearance of resistance breaking viral strains and estimations of their fitness, and hence, data relevant to the estimation of the durability of resistance in the field. In addition, studies using model species may provide information on new targets for interfering with the virus multiplication cycle in the host and, therefore, on the design of new strategies for the control of virus-induced diseases. As examples, newly developed technologies such are those based on RNA interference (Voinnet, 2002; Wang and Waterhouse, 2001) or high throughput methods for the identification of allelic variants in collections of mutagenized individuals based on TILLING (Targeting Induced Local Lesions IN Genomes) (McCallum *et al.*, 2000) seem promising for the transfer of information from model to crop species in order to engineer virus resistance.

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REFERENCES

Ahlquist, P., Noueiry, A., Lee, W., Kushner, D. and Dye, B. (2003) Host factors in positive-strand RNA virus genome replication. *J. Virol.* **77**, 8181–8186.

Albar, L., Ndjiondjop, M.N., Esshak, Z., Berger, A., Pinel, A., Jones, M., Fargette, D. and Ghesquiere, A. (2003) Fine genetic mapping of a gene required for *Rice yellow mottle virus* cell-to-cell movement. *Theor. Appl. Genet.* **107**, 371–378.

Aranda, M.A., Escaler, M., Wang, D. and Maule, A.J. (1996) Induction of HSP70 and polyubiquitin expression associated with plant virus replication. *Proc. Natl Acad. Sci. USA*, **93**, 15289–15293.

Axtell, M.J. and Staskawicz, B.J. (2003) Initiation of *RPS2*-specific disease resistance in *Arabidopsis* is coupled to the avrRpt2-directed elimination of RIN4. *Cell*, **112**, 369–377.

Azzam, O., Yambao, M.L.M., Muhsin, M., McNally, K.L. and Umadhay, K.M.L. (2000) Genetic diversity of *Rice tungro spherical virus* in tungro-endemic provinces of the Philippines and Indonesia. *Arch. Virol.* **145**, 1183–1197.

Baggett, J.R. and Hampton, R.O. (1991) Inheritance of viral bean leaf roll tolerance in peas. *J. Am. Soc. Hortic. Sci.* **116**, 728–731.

Boiteux, L.S., Cupertino, F.P., Silva, C., Dusi, A.N., MonteNeshich, D.C., vanderVlugt, R.A.A. and Fonseca, M.E.N. (1996) Resistance to *Potato virus Y* (pathotype 1-2) in *Capsicum annuum* and *Capsicum chinense* is controlled by two independent major genes. *Euphytica*, **87**, 53–58.

Brown, R.N., Bolanos-Herrera, A., Myers, J.R. and Jahn, M.M. (2003) Inheritance of resistance to four cucurbit viruses in *Cucurbita moschata*. *Euphytica*, **129**, 253–258.

Buschges, R., Hollricher, K., Panstruga, R., Simons, G., Wolter, M., Frijters, A., van Daelen, R., van der Lee, T., Diergaarde, P., Groenendijk, J., Topsch, S., Vos, P., Salamini, F. and Schulze-Lefert, P. (1997) The barley Mlo gene: a novel control element of plant pathogen resistance. *Cell*, **88**, 695–705.

Caranta, C., Palloix, A., GebreSelassie, K., Lefebvre, V., Moury, B. and Daubeze, A.M. (1996) A complementation of two genes originating from susceptible *Capsicum annuum* lines confers a new and complete resistance to *Pepper veinal mottle virus*. *Phytopathology*, **86**, 739–743.

Carrington, J.C., Kasschau, K.D., Mahajan, S.K. and Schaad, M.C. (1996) Cell-to-cell and long-distance transport of viruses in plants. *Plant Cell*, **8**, 1669–1681.

Covey, S.N., Al-Kaff, N.S., Langara, A. and Turner, D.S. (1997) Plants combat infection by gene silencing. *Nature*, **385**, 781–782.

D'Antonio, V., Falk, B. and Quiros, C.F. (2001) Inheritance of resistance to *Celery mosaic virus* in celery. *Plant Dis.* **85**, 1276–1277.

Dangl, J.L. and Jones, J.D.G. (2001) Plant pathogens and integrated defence responses to infection. *Nature*, **411**, 826–833.

Dardick, C., Golem, S. and Culver, J. (2000) Susceptibility and symptom development in *Arabidopsis thaliana* to *Tobacco mosaic virus* is influenced by virus cell-to-cell movement. *Mol. Plant-Microbe Interact.* **13**, 1139–1144.

Dawson, W.O. and Hilf, M.E. (1992) Host-range determinants of plant-viruses. *Annu. Rev. Plant Phys.* **43**, 527–555.

Deom, C.M., Murphy, J.F. and Paguio, O.R. (1997) Resistance to *Tobacco etch virus* in *Capsicum annuum*: inhibition of virus RNA accumulation. *Mol. Plant-Microbe Interact.* **10**, 917–921.

Díaz, J.A., Nieto, C., Moriones, E., Truniger, V. and Aranda, M.A. (2004) Molecular characterization of a *Melon necrotic spot virus* strain that overcomes the resistance in melon and non-host plants. *Mol. Plant-Microbe Interact.*, in press.

Diez, J., Ishikawa, M., Kaido, M. and Ahlquist, P. (2000) Identification and characterization of a host protein required for efficient template selection in viral RNA replication. *Proc. Natl Acad. Sci. USA*, **97**, 391–3918.

Dogimont, C., Bussemakers, A., Martin, J., Slama, S., Lecoq, H. and Pitrat, M. (1997) Two complementary recessive genes conferring resistance to Cucurbit aphid borne yellows luteovirus in an Indian melon line (*Cucumis melo* L.). *Euphytica*, **96**, 391–395.

Dogimont, C., Palloix, A., Daubze, A.M., Marchoux, G., Selassie, K.G. and Pochard, E. (1996) Genetic analysis of broad spectrum resistance to potyviruses using doubled haploid lines of pepper (*Capsicum annuum* L.). *Euphytica*, **88**, 231–239.

Duprat, A., Caranta, C., Revers, F., Menand, B., Browning, K.S. and Robaglia, C. (2002) The Arabidopsis eukaryotic initiation factor (iso) 4E is dispensable for plant growth but required for susceptibility to potyviruses. *Plant J.* **32**, 927–934.

Flor, H.H. (1971) Current status of the gene-for-gene concept. *Annu. Rev. Phytopatol.* **9**, 275–276.

Fraser, R.S.S. (1990) The genetics of resistance to plant viruses. *Annu. Rev. Phytopathol.* **28**, 179–200.

Fraser, R.S.S. (1999) Plant resistance to viruses. In: *Encyclopedia of Virology* (Granoff A. and Webster, R.G., eds). San Diego, CA: Academic Press, pp. 1300–1307.

Gilbert-Albertini, F., Pitrat, M. and Lecoq, H. (1995) Inheritance of resistance to *Zucchini yellow fleck virus. Cucumis sativus* L. *Hortscience*, **30**, 336–337.

Goodrick, B.J., Kuhn, C.W. and Hussey, R.S. (1991) Restricted systemic movement of *Cowpea chlorotic mottle virus* in soybean with non-necrotic resistance. *Phytopathology*, **81**, 1426–1431.

Grube, R.C., Zhang, Y.P., Murphy, J.F., Loaiza-Figueroa, F., Lackney, V.K., Provvidenti, R. and Jahn, M.K. (2000) New source of resistance to *Cucumber mosaic virus* in *Capsicum frutescens*. *Plant Dis.* **84**, 885–891.

Grumet, R., Kabelka, E., McQueen, S., Wai, T. and Humphrey, R. (2000) Characterization of sources of resistance to the watermelon strain of *Papaya ringspot virus* in cucumber: allelism and co-segregation with other potyvirus resistances. *Theor. Appl. Genet.* **101**, 463–472.

Hagiwara, Y., Komoda, K., Yamanaka, T., Tamai, A., Meshi, T., Funada, R., Tsuchiya, T., Naito, S. and Ishikawa, M. (2003) Subcellular localization of host and viral proteins associated with tobamovirus RNA replication. *EMBO J.* **22**, 344–353.

Hammond-Kosak, K.E. and Parker, J.E. (2003) Deciphering plant-pathogen communication: fresh perspectives for molecular resistance breeding. *Curr. Opin. Biotechnol.* **14**, 177–193.

Harrison, B.D. (2002) Virus variation in relation to resistance-breaking in plants. *Euphytica*, **124**, 181–192.

Hughes, S.L., Green, S.K., Lydiate, D.J. and Walsh, J.A. (2002) Resistance to *Turnip mosaic virus. Brassica rapa* and *B. napus* and the analysis of genetic inheritance in selected lines. *Plant Pathol.* **51**, 567–573.

Ishikawa, M., Diez, J., Restrepo-Hartwig, M. and Ahlquist, P. (1997) Yeast mutations in multiple complementation groups inhibit Brome mosaic virus RNA replication and transcription and perturb regulated expression of the viral polymerase-like gene. *Proc. Natl Acad. Sci. USA*, **94**, 4892–4896.

Ishikawa, M., Naito, S. and Ohno, T. (1993) Effects of the *tom1* mutation of *Arabidopsis thaliana* on the multiplication of Tobacco mosaic virus RNA in protoplasts. *J. Virol.* **67**, 5328–5338.

Ishikawa, M., Obata, F., Kumagai, T. and Ohno, T. (1991) Isolation of mutants of *Arabidopsis thaliana* in which accumulation of Tobacco mosaic virus coat protein is reduced to low levels. *Mol. Gen. Genet.* **230**, 33–38.

Janda, M. and Ahlquist, P. (1993) RNA-dependent replication, transcription and persistence of Brome mosaic virus RNA replicons in *S. cerevisiae*. *Cell*, **72**, 961–970.

Johansen, I.E., Lund, O.S., Hjulsager, C.K. and Laursen, J. (2001) Recessive resistance in *Pisum sativum* and potyvirus pathotype resolved in a gene-for-cistron correspondence between host and virus. *J. Virol.* **75**, 6609–6614.

Kabelka, E. and Grumet, R. (1997) Inheritance of resistance to the Moroccan watermelon mosaic virus in the cucumber line TMG-1 and cosegregation with *Zucchini yellow mosaic virus* resistance. *Euphytica*, **95**, 237–242.

Kanyuka, K., Ward, E. and Adams, M.J. (2003) Polymyxa graminis and the cereal viruses it transmits: a research challenge. *Mol. Plant Pathol.* **4**, 393–406.

Keller, K.E., Johansen, I.E., Martin, R.R. and Hampton, R.O. (1998) Potyvirus genome-linked protein (VPg) determines *Pea seed-borne mosaic virus* pathotype-specific virulence in *Pisum sativum*. *Mol. Plant-Microbe Interact.* **11**, 124–130.

Knight, V.H. and Barbara, D.J. (1981) Susceptibility of red raspberry varieties to *Raspberry bushy dwarf virus* and its genetic control. *Euphytica*, **30**, 803–811.

Kuhne, T., Shi, N., Proeseler, G., Adams, M.J. and Kanyuka, K. (2003) The ability of a bymovirus to overcome the rym4-mediated resistance in barley correlates with a codon change in the VPg coding region on RNA1. *J. Gen. Virol.* **84**, 2853–2859.

Lartey, R., Ghoshroy, S. and Citovsky, V. (1998) Identification of an *Arabidopsis thaliana* mutation (*vsm1*) that restricts systemic movement of tobamoviruses. *Mol. Plant-Microbe Interact.* **11**, 706–709.

Lee, W., Ishikawa, M. and Ahlquist, P. (2001) Mutation of host Δ9 fatty acid desaturase inhibits Brome mosaic virus RNA replication between template recognition and RNA synthesis. *J. Virol.* **75**, 2097–2106.

Lee, S., Stenger, D., Bisaro, D. and Davis, K. (1994) Identification of loci in *Arabidopsis* that confer resistance to geminivirus infection. *Plant J.* **6**, 525–535.

Legg, J.P. and Thresh, J.M. (2000) Cassava mosaic virus disease in East Africa: a dynamic disease in a changing environment. *Virus Res.* **71**, 135–149.

Lellis, A.D., Kasschau, K.D., Whitham, S.A. and Carrington, J.C. (2002) Loss-of-susceptibility mutants of *Arabidopsis thaliana* reveal an essential role for eIF(iso)4E during potyvirus infection. *Curr. Biol.* **12**, 1046–1051.

Leonard, S., Plante, D., Wittmann, S., Daigneault, N., Fortin, M.G. and Laliberte, J.F. (2000) Complex formation between potyvirus VPg and translation eukaryotic initiation factor 4E correlates with virus infectivity. *J. Virol.* **74**, 7730–7737.

MacKey, D., Belkhadir, Y., Alonso, J., Ecker, J.R. and Dangl, J.L. (2003) *Arabidopsis* RIN4 is a target of the type II virulence effector AvrRpt2 and modulates RPS2-mediated resistance. *Cell*, **112**, 379–389.

Malik, I.A., Sarwar, G. and Ali, Y. (1986) Genetic-studies in mung bean (*Vigna radiata* (L.) Wilczek).1. Inheritance of tolerance to mung bean yellow mosaic-virus and some morphological characters. *Pakistan J. Bot.* **18**, 189–198.

Maluf, W.R., Tomabraghini, M. and Corte, R.D. (1991) Progress in breeding tomatoes for resistance to tomato spotted wilt. *Rev. Brasileira Genetica*, **14**, 509–525.

Masuta, C., Nishimura, M., Morishita, H. and Hataya, T. (1999) A single amino acid change in viral genome-associated protein of *Potato virus Y* correlates with resistance breaking in 'Virgin A Mutant' tobacco. *Phytopathology*, **89**, 118–123.

Maule, A.J., Leh, V. and Lederer, C. (2002) The dialogue between viruses and hosts in compatible interactions. *Curr. Opin. Plant Biol.* **5**, 279–284.

McCallum, C.M., Comai, L., Greene, E.A. and Henihoff, S. (2000) Targeting induced local lesions in genomes (TILLING) for plant functional genomics. *Plant Physiol.* **123**, 439–442.

Miklas, P.N., Larsen, R.C., Riley, R. and Kelly, J.D. (2000) Potential marker-assisted selection for bc-1(2) resistance to Bean common mosaic potyvirus in common bean. *Euphytica*, **116**, 211–219.

Murphy, J.F., Blauth, J.R., Livingstone, K.D., Lackney, V.K. and Jahn, M.K. (1998) Genetic mapping of the pvr1 locus in *Capsicum* spp. and evidence that distinct potyvirus resistance loci control responses that differ at the whole plant and cellular levels. *Mol. Plant-Microbe Interact.* **11**, 943–951.

Murphy, J.F. and Kyle, M.M. (1995) Alleviation of restricted systemic spread of Pepper mottle potyvirus in *Capsicum annuum* cv avelar by coinfection with a cucumovirus. *Phytopathology*, **85**, 561–566.

Nicaise, V., German-Retana, S., Sanjuan, R., Dubrana, M.P., Mazier, M., Maisonneuve, B., Candresse, T., Caranta, C. and LeGall, O. (2003) The eukaryotic translation initiation factor 4E controls lettuce susceptibility to the potyvirus *Lettuce mosaic virus*. *Plant Physiol.* **132**, 1272–1282.

Nicolas, O., Dunnington, S.W., Gotow, L.F., Pirone, T.P. and Hellmann, G.M. (1997) Variations in the VPg protein allow a potyvirus to overcome *va* gene resistance in tobacco. *Virology*, **237**, 452–459.

Noueiry, A., Chen, J. and Ahlquist, P. (2000) A mutant allele of essential, general translation initiation factor *DED1* selectively inhibits translation of a viral mRNA. *Proc. Natl Acad. Sci. USA*, **97**, 12985–12990.

Noueiry, A., Diez, J., Falk, S., Chen, J. and Ahlquist, P. (2003) Yeast Lsm1p-7p/Pat1p deadenylation-dependent mRNA-decapping factors are required for Brome mosaic virus genomic RNA translation. *Mol. Cell. Biol.* **23**, 4094–4106.

Ohshima, K., Taniyama, T., Yamanaka, T., Ishikawa, M. and Naito, S. (1998) Isolation of a mutant of *Arabidopsis thaliana* carrying two simultaneous mutations affecting *Tobacco mosaic virus* multiplication within a single cell. *Virology*, **243**, 472–481.

Olorunju, P.E., Kuhn, C.W., Demski, J.W., Misari, S.M. and Ansa, O.A. (1992) Inheritance of resistance in peanut to mixed infections of Groundnut rosette virus (grv) and Groundnut rosette assistor virus and a single infection of grv. *Plant Dis.* **76**, 95–100.

Pal, S.S., Dhaliwal, H.S. and Bains, S.S. (1991) Inheritance of resistance to *Yellow mosaic virus* in some vigna species. *Plant Breed*, **106**, 168–171.

Panavas, T. and Nagy, P.D. (2003) Yeast as a model host to study replication and recombination of defective interfering RNA of *Tomato bushy stunt virus*. *Virology*, **314**, 315–325.

Park, S.J. and Tu, J.C. (1991) Inheritance and allelism of resistance to a severe strain of *Bean yellow mosaic virus* in common bean. *Can. J. Plant Pathol.* **13**, 7–10.

Parrella, G., Ruffel, S., Moretti, A., Morel, C., Palloix, A. and Caranta, C. (2002) Recessive resistance genes against potyviruses are localized in colinear genomic regions of the tomato (*Lycopersicon* spp.) and pepper (*Capsicum* spp.) genomes. *Theor. Appl. Genet.* **105**, 855–861.

Pink, D.A.C., Walkey, D.G.A. and McClement, S.J. (1991) Genetics of resistance to *Beet western yellows virus* in lettuce. *Plant Pathol.* **40**, 542–545.

Provvidenti, R. (1991a) Inheritance of resistance to the florida strain of *Zucchini yellow mosaic virus* in watermelon. *Hortscience*, **26**, 407–408.

Provvidenti, R. (1991b) Inheritance of resistance to the nl-8 strain of *Bean common mosaic virus* in *Pisum sativum. J. Hered.* **82**, 353–355.

Provvidenti, R. and Hampton, R.O. (1992) Sources of resistance to viruses in the potyviridae. *Arch. Virol.* **Suppl. 5**, 189–211.

Provvidenti, R. and Hampton, R.O. (1993) Inheritance of resistance to *White lupin mosaic virus* in common pea. *Hortscience*, **28**, 836–837.

Provvidenti, R. and Niblett, C.L. (1994) Inheritance of resistance to a strain of *Passionfruit woodiness virus* in pea (*Pisum-sativum* L.). *Hortscience*, **29**, 901–902.

Quadt, R., Ishikawa, M., Janda, M. and Ahlquist, P. (1995) Formation of Brome mosaic virus RNA-dependent RNA polymerase in yeast requires coexpression of viral proteins and viral RNA. *Proc. Natl Acad. Sci. USA*, **92**, 4892–4896.

Rajamony, L., More, T.A. and Seshadri, V.S. (1990) Inheritance of resistance to *Cucumber green mottle mosaic virus* in muskmelon (*Cucumis melo* L.). *Euphytica*, **47**, 93–97.

Revers, F., Guiraud, T., Houvenaghel, M., Mauduit, T., Le Gall, O. and Candresse, T. (2003) Multiple resistance phenotypes to *Lettuce mosaic virus* among *Arabidopsis thaliana* accessions. *Mol. Plant-Microbe Interact.* **16**, 608–616.

Ruffel, S., Dussault, M.H., Palloix, A., Moury, B., Bendahmane, A., Robaglia, C. and Caranta, C. (2002) A natural recessive resistance gene against *Potato virus Y* in pepper corresponds to the eukaryotic initiation factor 4E (eIF4E). *Plant J.* **32**, 1067–1075.

Sato, M., Masuta, C. and Uyeda, I. (2003) Natural resistance to *Clover yellow vein virus*. beans controlled by a single recessive locus. *Mol. Plant-Microbe Interact.* **16**, 994–1002.

Schaad, M.C., Anderberg, R.J. and Carrington, J.C. (2000) Strain–specific interaction of the Tobacco etch virus NIa protein with the translation initiation factor eIF4E in the yeast two-hybrid system. *Virology*, **273**, 300–306.

Schaad, M.C. and Carrington, J.C. (1996) Suppression of long-distance movement of *Tobacco etch virus* in a non-susceptible host. *J. Virol.* **70**, 2556–2561.

Schoelz, J.E. and Wintermantel, W.M. (1993) Expansion of viral host-range through complementation and recombination in transgenic plants. *Plant Cell*, **5**, 1669–1679.

Schroeder, W.T. and Provvidenti, R. (1971) A common gene for resistance to *Bean yellow mosaic virus* and *Watermelon mosaic virus* 2 in *Pisum sativum. Phytopathology*, **61**, 846–848.

Tomita, Y., Mizuno, T., Diez, J., Naito, S., Ahlquist, P. and Ishikawa, M. (2003) Mutation of host *dnaJ* homolog inhibits Brome mosaic virus negative-strand RNA synthesis. *J. Virol.* **77**, 2990–2997.

Tsujimoto, Y., Numaga, T., Ohshima, K., Yano, M., Ohsawa, R., Goto, D., Naito, S. and Ishikawa, M. (2003) *Arabidopsis tobamovirus* multiplication (TOM) 2 locus encodes a transmembrane protein that interacts with TOM1. *EMBO J.* **22**, 335–343.

Ullah, Z. and Grumet, R. (2002) Localization of Zucchini yellow mosaic virus to the veinal regions and role of viral coat protein in veinal chlorosis conditioned by the zym potyvirus resistance locus in cucumber. *Physiol. Mol. Plant P.* **60**, 79–89.

Umaharan, P., Ariyanayagam, R.P. and Haque, S.Q. (1997) Resistance to *Cowpea severe mosaic virus*, determined by three dosage dependent genes in *Vigna unguiculata* L. Walp. *Euphytica*, **95**, 49–55.

Vidavsky, F. and Czosnek, H. (1998) Tomato breeding lines resistant and tolerant to *Tomato yellow leaf curl virus* issued from *Lycopersicon hirsutum*. *Phytopathology*, **88**, 910–914.

Voinnet, O. (2002) RNA silencing: small RNAs as ubiquitous regulators of gene expression. *Curr. Opin. Plant Biol.* **5**, 444–451.

Wai, T. and Grumet, R. (1995) Inheritance of resistance to *Watermelon mosaic virus* in the cucumber line tmg-1—Tissue-specific expression and relationship to *Zucchini yellow mosaic virus* resistance. *Theor. Appl. Genet.* **91**, 699–706.

Wang, M.-B. and Waterhouse, P.M. (2001) Application of gene silencing in plants. *Curr. Opin. Plant Biol.* **5**, 146–150.

Waterhouse, P.M., Wang, M.-B. and Lough, T. (2001) Gene silencing as an adaptive defence against viruses. *Nature*, **411**, 834–842.

Whitham, S.A., Quan, S., Chang, H.S., Cooper, B., Estes, B., Zhu, T., Wang, X. and Hou, Y.M. (2003) Diverse RNA viruses elicit the expression of common sets of genes in susceptible *Arabidopsis thaliana* plants. *Plant J.* **33**, 271–283.

Whitham, S., Yamamoto, M. and Carrington, J. (1999) Selectable viruses and altered susceptibility mutants in *Arabidopsis thaliana*. *Proc. Natl Acad. Sci. USA*, **96**, 772–777.

Wintermantel, W.M., Anderson, E.J. and Schoelz, J.E. (1993) Identification of domains within gene-VI of *Cauliflower mosaic virus* that influence systemic infection of *Nicotiana bigelovii* in a light-dependent manner. *Virology*, **196**, 789–798.

Wittmann, S., Chatel, H., Fortin, M.G. and Laliberte, J.F. (1997) Interaction of the viral protein genome linked of Turnip mosaic potyvirus with the translational eukaryotic initiation factor (iso) 4E of *Arabidopsis thaliana* using the yeast two-hybrid system. *Virology*, **234**, 84–92.

Yamanaka, T., Imai, T., Satoh, R., Kawashima, A., Takahashi, M., Tomita, K., Kubota, K., Meshi, T., Naito, S. and Ishikawa, M. (2002) Complete inhibition of tobamovirus multiplication by simultaneous mutations in two homologous host genes. *J. Virol.* **76**, 2491–2497.

Yamanaka, T., Ohta, T., Takahashi, M., Meshi, T., Schmidt, R., Dean, C., Naito, S. and Ishikawa, M. (2000) TOM1, an Arabidopsis gene required for efficient multiplication of a tobamovirus, encodes a putative transmembrane protein. *Proc. Natl Acad. Sci. USA*, **97**, 10107–10112.

Yoon, J.Y., Green, S.K. and Opena, R.T. (1993) Inheritance of resistance to *Turnip mosaic virus* in chinese cabbage. *Euphytica*, **69**, 103–108.

Yoshii, M., Yoshioka, N., Ishikawa, M. and Naito, S. (1998a) Isolation of an *Arabiopsis thaliana* mutant in which accumulation of Cucumber mosaic virus coat protein is delayed. *Plant J.* **13**, 211–219.

Yoshii, M., Yoshioka, N., Ishikawa, M. and Naito, S. (1998b) Isolation of an *Arabiopsis thaliana* mutant in which the multiplication of both *Cucumber mosaic virus* and *Turnip crinkle virus* is affected. *J. Virol.* **72**, 8731–8737.