Eur J Plant Pathol (2009) 123:281–289
DOI 10.1007/s10658-008-9365-3

Reduced BYDV–PAV transmission by the grain aphid in a *Triticum monococcum* line

S. Tanguy · C.-A. Dedryver

Received: 16 April 2008 / Accepted: 11 August 2008 / Published online: 28 August 2008

Abstract The luteovirus *Barley yellow dwarf virus*–PAV (BYDV–PAV) and its vector, the aphid *Sitobion avenae* are two major sources of yield losses in cereal crops. We report in this paper the effects of a *Triticum monococcum* line (TM44), resistant by antibiosis to *S. avenae,* on the different steps of transmission of one BYDV–PAV isolate by the aphids. First, it was shown that TM44 is strongly resistant to BYDV–PAV transmission, but exclusively when *S. avenae* is the vector. Second, that TM44 is resistant (1) to BYDV–PAV acquisition by *S. avenae* and (2) to its inoculation, whatever the respective duration of these two periods. Third, that both resistances have partially additive effects. In the discussion, several lines of evidence are given to support the hypothesis that resistance of TM44 to PAV transmission is due to the same disturbances to *S. avenae* feeding behaviour that are involved in its antibiosis against this aphid species. Reasons for caution in releasing resistant material in the field are presented.

Keywords Wheat · Antibiosis · *Luteoviridae* · *Sitobion avenae* · Acquisition · Inoculation

S. Tanguy · C.-A. Dedryver (✉)
INRA/Agrocampus/Université Rennes 1, UMR 1099,
BiO3P Domaine de la Motte, BP 35327, 35653 Le Rheu,
France
e-mail: charles-antoine.dedryver@rennes.inra.fr

Introduction

Aphids and aphid-borne viruses are two major sources of crop losses in temperate climates (Tatchell 1989). Their control is mainly based on insecticide treatments against aphids in order to avoid direct damage due to feeding and indirect damage due to virus spread. However, breeding cultivars resistant to aphids and/or viruses could be an alternative control method in the context of sustainable agricultural development.

In recent decades, considerable efforts have been made in order to find and characterise plant resistance sources to insects and/or to viruses, and many attempts have been made to incorporate them into new cultivars, with variable degrees of success. At the end of the twentieth century, more than 200 insect-resistant cultivars were grown around the world (Smith 1989). In antixenosis, a plant is rapidly recognised as a poor host by a pest that subsequently moves away. In antibiosis, survival and fecundity of the pest are affected by feeding on the plant. Both types of resistance have been reported in a large number of aphid-crop interactions (Webster 1991), as well as resistance to virus movement (Palloix et al. 1997), virus multiplication (Lecoq and Pitrat 1982) and virus transmission (Chen et al. 1996). However, only some of these genes have been successfully introduced into commercial varieties, e.g. (1) the single dominant *Vat* gene conferring melon resistance

to *Cucumber mosaic virus* (CMV) transmission by *Aphis gossypii*, (2) a resistance gene to *Zucchini yellow mosaic virus* (ZYMV) in zucchini squash (Lecoq et al. 2004) and (3) the single dominant gene *Byd2* for resistance to *Barley yellow dwarf virus* (BYDV) in the wheat cv. MacKellar (Laurence and Banks 2003).

Aphids cause crop damage both by direct feeding and by virus transmission. As resistance genes to aphids or to plant viruses are rare and generally difficult and costly to introduce into commercial varieties, the most economical way consists in focusing on resistance genes acting both on the vectors and their associated viruses during the transmission step. Indeed, resistance to aphids (especially by antibiosis) and resistance to virus transmission are not independent in many cases, because both of them target aphid feeding function. In an extensive review, Jones (1987) cited more than 20 examples of different crop/virus combinations where resistance to the vector was found to decrease virus incidence in the field or/and in laboratory experiments. However, until now, the relationships between the two processes have not been precisely described in the case of persistently transmitted viruses, unlike for non-persistently transmitted viruses (Martin et al. 2003).

In Europe the 'grain aphid' *Sitobion avenae* causes frequent direct damage to cereals between May and July, especially on winter wheat with losses averaging 1.2 tons ha^{-1}, (Tatchell 1989), due to its huge multiplication between heading and milky-ripening stage of the crop. Moreover, the three major aphid species occurring on cereals (*Rhopalosiphum padi*, *S. avenae*, *Metopolophium dirhodum*) also transmit in a persistent manner several viral species belonging to the *Barley/Cereal yellow dwarf virus* (B/CYDV) complex (D'Arcy and Mayo 1997). In barley and wheat, B/CYDV can lower the yield by >50% (Lister and Ranieri 1997). With persistently transmitted viruses such as B/CYDV, virus acquisition from a source plant, and virus inoculation to a target plant, are entirely dependent on vector feeding behaviour.

Some sources of resistance to BYDV (mostly against BYDV–PAV) have been identified from wild relatives of barley and wheat. In barley, the most efficient source of resistance, associated with the *Yd2* gene from Ethiopian wild barley (Rasmusson and Schaller 1959), has been extensively used in breeding programmes. Resistance sources to BYDV–PAV multiplication have been found in perennial *Triticineae* (genera *Thinopyrum* and *Lophopyrum*) and transferred to bread wheat genotypes. The resulting wheat lines are partly resistant, but have been shown to quickly induce the selection of virus variants with increased infection abilities (Chain et al. 2006).

Resistance (antibiosis) to cereal aphids, mainly *S. avenae*, has been found (1) in plants with high concentrations of secondary compounds like DIMBOA (Niemeyer 1991), (2) in the genera *Agropyron*, *Elytrigia* and *Pascopyrum* (Triticineae; Shukle et al. 1987), and (3) in the diploid wheat *Triticum monococcum* (Di Piétro et al. 1998). The genotype *T. monococcum* REB81044 (TM44), strongly resistant to *S. avenae*, has been the most intensively studied. Caillaud et al. (1994) showed that life-history traits of *S. avenae* are dramatically affected on TM44, i.e. a variable proportion (depending on the clone used) of aphids die at larval stages and adult fecundity is decreased ten-fold in comparison with a susceptible hexaploid cultivar. Resistance to *S. avenae* in TM44 is associated with repeated stylet penetrations without access to phloem, with numerous failures in starting sustained sap ingestion and with a drastic decrease in the number and length of sap ingestion periods (Caillaud et al. 1995a). Incorporation of TM44 resistance genes in hexaploid wheats is in progress (J. Jahier, personal communication).

TM44 is not resistant to C/BYDV multiplication. Hence, as aphid feeding behaviour is a key component of luteovirus transmission (Prado and Tjallingii 1994) and of TM44 resistance (Caillaud et al. 1995a), BYDV transmission to TM44 has to be evaluated.

The aim of this work was to assess whether or not TM44 decreases BYDV transmission by *S. avenae* in laboratory experiments and to what extent, as a prelude to field studies on the role of future wheat varieties with TM44 resistance genes on BYDV epidemiology. First, we compared the transmission probabilities of one BYDV–PAV isolate by *S. avenae* and two other aphid species to TM44, to a hexaploid wheat cultivar and to a barley cultivar, in order to separate the respective roles of aphid and plant species in virus transmission. Second, we compared the effects of TM44 and of a hexaploid wheat on the two major steps of BYDV–PAV transmission by

S. avenae (acquisition and inoculation), in order to assess the respective roles of those plant species (1) as virus sources and (2) as virus receptors.

Materials and methods

Aphid species and clones

Transmission experiments were conducted with three cereal aphid species (one clone of each): *R. padi* (clone Rp1, collected in Rennes, France on wheat in 1978), *M. dirhodum* (clone collected in Rennes on wheat in 1978) and *S. avenae* (clone Sa1 collected in Rennes on wheat in 1990). Clone Sa1 was chosen because at the adult stage, it was essentially susceptible to TM44 (Caillaud et al. 1995b), and the larvae survive on TM44, and moult into adults that generally die after one week without giving birth to offspring. Clone Sa1 larvae could still be used for virus transmission to and from TM44. Since their collection, all clones have been reared on winter wheat seedlings (cv. Orvantis) in a growth chamber at 20± 1°C, with a 16-h photoperiod.

Plant species

We used the barley (*Hordeum vulgare*) cv. Express and the hexaploid winter wheat (*Triticum aestivum*) cv. Arminda that are both susceptible to the three aphid species, and the *Triticum monococcum* line REB81044 (TM44) highly resistant to *S. avenae*, but susceptible to *M. dirhodum* and *R. padi*. Plants of the three species were reared individually until the two-leaf stage in a growth chamber at 15±1°C, with a 16-h photoperiod, in plastic pots filled with compost, and used thereafter as test plants.

Virus isolate

The BYDV–PAV isolate PAV4 was used in the experiments. It was collected from barley in le Rheu (France) in 1989 and causes severe symptoms on the barley cv. Express. This isolate was maintained on barley seedlings of cv. Express infested with the *R. padi* clone Rp1 in a growth chamber at 20±1°C, with a 16-h photoperiod.

Viral inoculum

For the three different plant species, batches of two leaf-stage seedlings, grown in plastic pots, were each inoculated by three third instar larvae (wingless) of *R. padi* collected on the infected barley plants described above. Each seedling was caged with a cellophane bag for a 120-h inoculation access period (IAP) and then all *R. padi* were carefully removed from the plants with a fine brush. After a 15-day period allowing virus multiplication in the plants, each plant was tested for virus content with a triple antibody sandwich–enzyme linked immunosorbent assay (TAS–ELISA, see below). Plants with similar optical densities (OD) were used as viral inoculum in the transmission experiments described below.

Transmission experiments

In the three following experiments, BYDV–PAV transmission was done in a growth chamber at 20±1°C, with a 16-h photoperiod. For each aphid species, groups of 100 virus-free third instar larvae (wingless) were removed from the stock colonies and placed on the virus source plants for the acquisition access period (AAP), before being transferred onto each plant (three aphids/test plant). After the IAP, aphids were sprayed with insecticide (Décis EC, Bayer CropScience, deltamethrin 1 ml l^{-1}) and the test plants were reared in a glasshouse at 15±5°C for 15 days. Each treatment (aphid species × plant species) consisted of 20 test plants and was repeated three times.

Experiment 1 Effectiveness of PAV4 transmission by the three aphid species to each of the three plant species (AAP=48 h/IAP=120 h) was compared. The same cultivar or line was used as the virus source plant and test (inoculated) plant.

Experiment 2 The effects of the duration of AAP of PAV4 by *S. avenae* (2, 6, 12, 24, 48, 120, and 240 h) with a fixed IAP (120 h) for three different treatments was studied: acquisition on Arminda/inoculation to Arminda; acquisition on TM44/inoculation to TM44; acquisition on TM44/inoculation to Arminda.

Experiment 3 For a constant 48-h AAP by *S. avenae*, seven different IAPs of PAV4 (2, 6, 12, 24, 48, 120,

240 h) for three different treatments were studied: acquisition on Arminda/inoculation to Arminda; acquisition on TM44/inoculation to TM44; acquisition on Arminda/inoculation to TM44.

Virus detection

Detection of virus antigen in leaves of test plants was done by TAS–ELISA as described for BYDV (Torrance et al. 1986; Leclercq-Le Quillec et al. 1995). Anti PAV-like purified IgG was produced by INRA, (former Laboratoire de Virologie, Versailles, France) and anti-PAV monoclonal antibody Mac91 was purchased from Neogen Europe® Ltd (Ayr, Scotland, UK). The OD was measured at 405 nm with a Dynatech MR5000 spectrophotometer after 1–2 h incubation at room temperature. As suggested by previous authors, samples were considered positive when OD values were greater than three times the mean OD of uninfected control leaves. Virus transmission efficiency (probability of transmission) was calculated for each repetition as the proportion of infected plants among the 20 plants inoculated. In the text, means are given with their standard errors (±SE).

Data analyses

Analyses of variance (ANOVAs) were performed using software S-Plus 6.2 (Insightful Corporation 2002). Better stabilisation of variances was obtained for data transformation by Arcsin√x. Normality of the data was checked by the Shapiro–Wilk test. Homogeneity of variances was assessed by Levene's test and significant effects were followed by comparison of means using Tukey's tests.

Results

BYDV–PAV transmission by three aphid species to cv. Express, cv. Arminda and TM44 (experiment 1)

Figure 1 shows a strong effect of the aphid species on the effectiveness of PAV4 transmission: *R. padi* transmitted most efficiently with a mean of 0.98±0.02 (SE), followed by *M. dirhodum* (0.76±0.07) and *S. avenae* (0.32±0.09). There were no clear differences between plant species in transmission efficiency

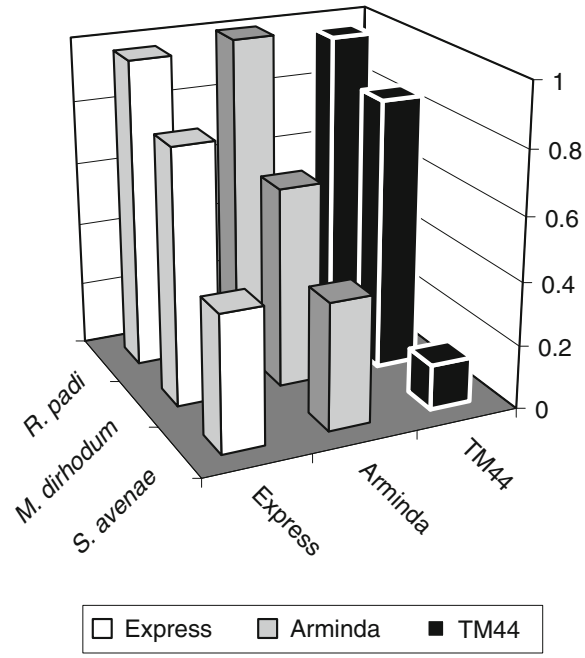

Fig. 1 Probabilities of BYDV–PAV4 transmission (experiment 1) by three aphid species to cv. Express, cv. Arminda and TM44 (AAP=48 h/IAP=120 h). Columns correspond to mean infection proportions of the test plants (three repetitions)

by *R. padi* and *M. dirhodum*. For *S. avenae*, the mean PAV4 transmission probability to TM44 was especially low (0.14±0.05), compared to that to Arminda (0.40±0.02) and Express (0.43±0.04). A first two-way ANOVA showed a highly significant effect of the aphid species on PAV4 transmission (df=2; F=89.69; P<0.0001) with significant differences between all three pairs of aphid species for transmission means (Tukey's test), no effect of the plant species (df=2; F=1.3; P=0.3) but a significant plant species × aphid species interaction (df=4; F=4.56; P=0.010). A one-way ANOVA on *S. avenae* data alone, showed a highly significant plant effect (df=2; F=14.8; P=0.005), with significant differences between TM44 and the other plant species (Tukey's test).

Effect of the virus source and the length of AAP on BYDV–PAV transmission by *S. avenae* (experiment 2)

Figure 2a shows that, for each AAP, the higher probabilities of transmission were reached when transmitting to and from Arminda. Conversely the lowest transmission probabilities were obtained when

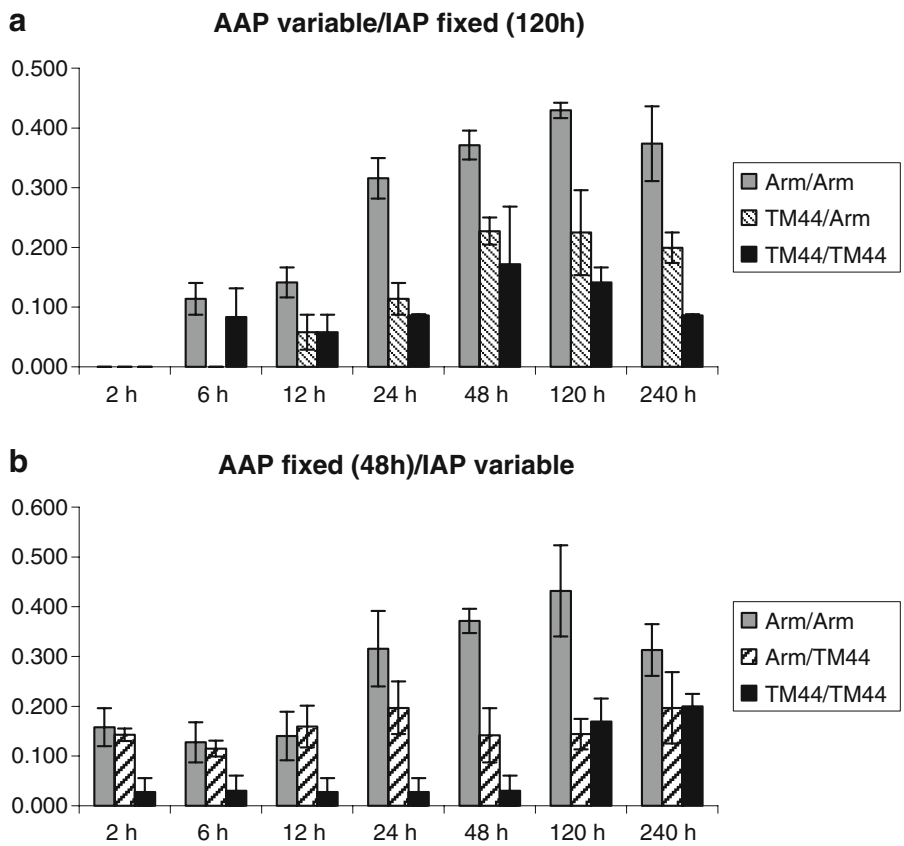

Fig. 2 Probabilities of BYDV–PAV transmission by *S. avenae*. *Arm*/*Arm* acquisition and inoculation on Arminda, *TM44*/*Arm* acquisition on TM44 and inoculation on Arminda, *Arm*/*TM44* acquisition on Arminda and inoculation on TM44, *TM44/TM44* acquisition and inoculation on TM44. **a** Effects of the virus source plant species and of AAP (experiment 2). **b** Effects of the target plant species and of IAP (experiment 3). *Columns* correspond to mean infection proportions of the test plants (three repetitions) and *bars* to standard errors

transmitting to and from TM44 (for AAP lengths of 24 h and more). Transmission probabilities were intermediate when transmitting from TM44 to Arminda.

The two-way ANOVA on transmission probabilities showed:

1. A highly significant effect of the source plant on transmission probability (df=1; F=15.187; P=0.00038): on average, the probability of PAV4 transmission to Arminda was two times lower when the source-plant was TM44 (0.117) than when the source-plant was Arminda (0.249).
2. A highly significant effect of the length of AAP on transmission probability (df=6; F=23.228; P<0.0001).
3. No interaction between the two above variables (df=6; F=1.106; P=0.299): the role of the source plant on transmission probability was the same whatever the length of AAP.

When the isolate PAV4 was acquired from and inoculated to TM44, there was no significant effect of the length of AAP on the probability of transmission (Fig. 2a), which was, in any case, low (0.0–0.17).

Effect of the target plant and the length of IAP on BYDV–PAV transmission by *S. avenae* (experiment 3)

Figure 2b shows that, for each IAP, the higher probabilities of transmission were obtained when transmitting to and from Arminda, and the lowest when transmitting to and from TM44: in this case, transmission probabilities were especially low for the shorter IAPs (below 120 h). Intermediate transmission probabilities were obtained when transmitting from Arminda to TM44.

The two-way ANOVA showed:

1. A highly significant effect of the target plant on transmission probability (df=1; F=15.312; P=0.00053): on average, probability of PAV4 transmission by *S. avenae* was 1.7 times lower when the target plant was TM44 (0.15714), that when it was Arminda (0.26571).

2. A significant effect of the length of IAP on transmission probability (df=6; F=3.335; P=0.0132).
3. A slightly significant interaction between the above two variables (df=6; F=2.566; P=0.0416) due to the fact that differences between transmission probabilities on TM44 and on Arminda as the target plant were only significant for a long IAP (24–240 h, Fig. 2b).

When the isolate PAV4 was acquired from and inoculated to TM44, there was a significant effect of IAP on the probability of transmission (df=6, F=5.91, P=0.003), which was the highest for the longest IAP (120 and 240 h; Fig. 2b).

Effects of TM44 on PAV acquisition and inoculation by *S. avenae* are partly additive

Figures 2a and b show that, when both source and target plants were TM44, transmission probabilities were, in many cases, lower than when only one of them was TM44 (except for Fig. 2a for the three shortest AAPs and Fig. 2b for the two longest IAPs), giving an indication of the probable additive effects of TM44 on both steps of acquisition and inoculation.

A two-way ANOVA analysis was done on the unique data set including all treatments (48 h AAP and 120 h IAP) and showed: (1) a significant effect of the plant species from which the virus was acquired (source; df=1; F=7.33, P=0.017) and (2) a highly significant effect of the species of plant inoculated (target) when nested in the source (df=1; F=10.1, P=0.0067). TM44 was resistant to BYDV–PAV acquisition and strongly resistant to its inoculation by *S. avenae*. This confirms that, under the conditions of the experiment (48 h AAP and 120 h IAP), both effects were partly additive.

Discussion

TM44 resistance to BYDV–PAV transmission was strong and linked to specific resistance to *S. avenae*

This paper describes, for the first time, the role of a resistance source affecting feeding behaviour of an aphid vector on the successive steps of the transmission of a persistent virus. Resistance to transmission was restricted to *S. avenae*, which is also the only cereal aphid species for which life-history traits are strongly affected by TM44 (Caillaud et al. 1994), and confirms the link between both processes.

Virus transmission by aphids is subject to many causes of variation that are difficult to control fully (including aphid age, aphid manipulation, aphid transfer from one plant species to another). In particular, it has been shown that inoculating aphid species in routine inoculum production can influence subsequent transmission efficiency (Lucio-Zavaleta et al. 2001); this could have played a minor role in increasing BYDV–PAV transmission probabilities by *R. padi* in experiment 1, but in any case, *R. padi* is recognised as an excellent vector of this virus (Plumb 1995). However, our laboratory results showed clear significant differences: the *T. monococcum* line REB81044 (TM44) was strongly resistant to BYDV–PAV4 transmission by *S. avenae* clone Sa1, cumulating in resistances to virus acquisition and virus inoculation. This resistance was maintained, however, for the duration of both periods (from 2 h to 5 days) and its level seems even higher than the similar resistance of one Chilean cultivar to BYDV–PAV transmission, as described by Givovitch and Niemeyer (1991). However, generalising these conclusions, based on one aphid clone and one virus isolate, to natural populations, needs more field work, since PAV transmission by *S. avenae* is highly variable, depending on aphid and virus genotypes and on interactions between them (Dedryver et al. 2005). More field work is also essential to quantify the demographic and epidemiological advantages of wheat material with TM44 resistance.

For persistently transmitted viruses, the most detailed study on the relationships between feeding behaviour of an aphid (*R. padi*) and virus transmission (BYDV–PAV) was carried out by Prado and Tjallingii (1994) using the electropenetrography technique or EPG (Tjallingii 1988), in which each phase of aphid feeding behaviour was associated with a characteristic electric pattern. These authors showed that duration of pattern E2 (phloem sap ingestion) was strongly correlated to the probability of virus acquisition, whereas duration of pattern E1 (salivation into sieve elements and preliminaries to the sap ingestion event) was also strongly correlated to virus inoculation. However, in this case the probability of inoculation was doubled when E1 was followed by E2, which shows that E2 has also a role in the

inoculation phase, maybe because salivation continues during the sap ingestion event.

Feeding behaviour of *S. avenae* on TM44 and on the susceptible wheat cv. Arminda was carefully studied by Caillaud et al. (1995a) by continuous 8 h EPG records. Aphids reared on TM44 took a significantly longer time to reach pattern E2 (phloem sap ingestion) and these E2 patterns had a significantly lower duration (mean and total) than on Arminda. From these results and those of Prado and Tjallingii (1994), we can hypothesise that low BYDV–PAV acquisition by *S. avenae* on TM44 was due to disturbances in sap ingestion phases. Low BYDV–PAV inoculation probabilities to TM44 are more difficult to explain because E1 patterns of *S. avenae* on TM44 were not significantly different in their numbers and in their duration, from those observed on susceptible wheat cultivars such as Arminda (Caillaud et al. 1995a). An hypothesis could be that on TM44, the scarcity and low duration of sap ingestion events (E2) following E1 did not allow the aphid to maximise the success of inoculation, also observed by Prado and Tjallingii (1994).

Resistance to virus transmission has to be approached with caution

That TM44 is not only resistant to the most noxious wheat aphid in the ancient world, but also to transmission of the main wheat virus by this species, strengthens the case for continuing the attempts of introgression of its resistance into commercial wheat varieties by conventional approaches. However, once introduced, the durability of the field resistance must be carefully evaluated before releasing any material. Several arguments support a cautionary approach:

First, the degree of field specificity of resistance genes may have important demographic and epidemiological consequences. In some cases, a resistance gene to several vector species has been successfully incorporated into a single variety: for example the Triticale Gaucho, bred for resistance to the aphid, *Schizaphis graminum* is also resistant to the acari *Aceria tulipae* (Wood et al. 1995). However, in most cases, resistances to insects are very specific, especially when due to antibiosis, as for example in the *T. monococcum* line (TM44), with strong resistance to *S. avenae* and no resistance to *M. dirhodum* and *R. padi*. Consequently, in field conditions of TM44 resistance deployment, there is a risk that the target aphid species would be very rapidly replaced by others, able to provoke the same level of direct damage and also to transmit the same viruses with the same (if not better) efficiency.

At an intra-specific level, emergence of biotypes overcoming plant resistance is also an important risk in some aphid species. During the past 50 years in North America, several greenbug (*S. graminum*) biotypes exhibiting inter- or intra-specific host plant variation, overcoming successively resistance genes of wheat or/and sorghum have developed. In the late 1980s, Puterka and Peters (1990) studied the inheritance of virulence traits in *S. graminum*, and by crossing three biotypes, produced 25 new ones, formed by the independent assortment of virulence genes during meiosis. More recently, emergence of a new biotype of the Russian wheat aphid (*Diuraphis noxia*) has been suspected in Colorado (Haley et al. 2004). Pre-existent *S. avenae* biotypes overcoming TM44 resistance do not seem to exist, but there is some intra-specific variability in the response of *S. avenae* to TM44 (Caillaud et al. 1995b): among sixty aphid clones tested, there was a marked and continuously distributed variation in performance on the resistant line, with a range in 'intrinsic rate of increase' (r_m) of 0.09–0.16, compared to 0.24–0.26 for a susceptible wheat variety. The authors pointed out the risk of selection of the less affected clones in case of field deployment of TM44 resistance genes. Moreover, preliminary crosses between *S. avenae* clones strongly and moderately susceptible to TM44 seemed to validate the hypothesis of dominance of the character 'moderately susceptible' in this aphid species, but the study of a genetic determinant for virulence has never been completed.

Finally, many plant resistances to insects are polygenic and very difficult to introduce as a whole in cultivated varieties. In most cases, a part of the resistance is lost or not expressed in the final crosses. For example, resistances to aphids from *T. monococcum* lines, until now, have been only partly introduced into wheat. Consequently we consider the most probable situation would be that future resistant varieties would be only partially resistant, because of 'dilution' of TM44 genes in successive crosses. The role of these varieties in BYDV epidemiology could be ambiguous: lowering acquisition and inoculation may be offset by increasing (1) aphid movements

from plant to plant and (2) percentage of winged morphs in the aphid population, and both should be considered in further experiments.

Acknowledgement We are grateful to M. Trottet (UMR APBV, INRA Le Rheu) for providing TM44 seeds and making useful comments on the manuscript, and to Monique Henry (Clause, Angers) for scientific advice and improving the English.

References

Caillaud, C. M., Dedryver, C.-A., Di Piétro, J.-P., Simon, J.-C., Fima, F., & Chaubet, B. (1995b). Clonal variability in the response of *Sitobion avenae* (Homoptera: Aphididae) to resistant and susceptible wheat. *Bulletin of Entomological Research*, *85*, 189–195.

Caillaud, C. M., Dedryver, C.-A., & Simon, J.-C. (1994). Development and reproductive potential of the cereal aphid *Sitobion avenae* on resistant wheat lines (*Triticum monococcum*). *The Annals of Applied Biology*, *125*, 219–232. doi:10.1111/j.1744-7348.1994.tb04964.x.

Caillaud, C. M., Pierre, J.-S., Chaubet, B., & Di Piétro, J.-P. (1995a). Analysis of wheat resistance to the cereal aphid *Sitobion avenae* using electrical penetration graphs and flow charts combined with correspondence analysis. *Entomologia Experimentalis et Applicata*, *75*, 9–18. doi:10.1007/BF02382774.

Chain, F., Riault, G., Trottet, M., & Jacquot, E. (2006). Evaluation of the durability of the *Barley yellow dwarf virus*-resistant *Zhong ZH* and *TC14* wheat lines. *European Journal of Plant Pathology*, *117*, 35–43. doi:10.1007/s10658-006-9066-8.

Chen, J., Delobel, B., Rahbé, Y., & Sauvion, N. (1996). Biological and chemical characteristics of a genetic resistance of melon to the melon aphid. *Entomologia Experimentalis et Applicata*, *80*, 250–253. doi:10.1007/BF00194768.

D'Arcy, C. J., & Mayo, M. (1997). Proposal for changes in luteovirus taxonomy and nomenclature. *Archives of Virology*, *142*, 1285–1287.

Dedryver, C. - A., Riault, G., Tanguy, S., Le Gallic, J. - F., Trottet, M., & Jacquot, E. (2005). Intra-specific variation and inheritance of BYDV–PAV transmission in the aphid *Sitobion avenae*. *European Journal of Plant Pathology*, *111*, 341–354. doi:10.1007/s10658-004-4890-1.

Di Piétro, J.-P., Caillaud, C. M., Chaubet, B., Pierre, J.-S., & Trottet, M. (1998). Variation in resistance to the grain aphid, *Sitobion avenae* (Sternorhyncha: Aphididae), among diploid wheat genotypes: multivariate analysis of agronomic data. *Plant Breeding*, *117*, 407–412. doi:10.1111/j.1439-0523.1998.tb01964.x.

Givovitch, A., & Niemeyer, H. M. (1991). Hydroxamic acids affecting barley yellow dwarf transmission by the aphid *Rhopalosiphum padi*. *Entomologia Experimentalis et Applicata*, *59*, 79–85. doi:10.1007/BF00187969.

Haley, S. D., Peairs, F. B., Walker, C. B., Rudolph, J. B., & Randolph, T. L. (2004). Occurrence of a new Russian wheat aphid biotype in Colorado. *Crop Science*, *44*, 1589–1592.

Insightful Corporation (2002). S-Plus 6.2. Seattle, WA, USA.

Jones, A. T. (1987). Control of virus infection in crop plants through vector resistance: a review of achievements, prospects and problems. *The Annals of Applied Biology*, *111*, 745–772. doi:10.1111/j.1744-7348.1987.tb02033.x.

Laurence, J., & Banks, P. (2003). Virus-resistant wheats worth the wait. *Partners in Research for Development*, *15*, 37–40.

Leclercq-Le Quillec, F., Tanguy, S., & Dedryver, C.-A. (1995). Aerial flow of barley yellow dwarf viruses and of their vectors in western France. *The Annals of Applied Biology*, *126*, 75–90. doi:10.1111/j.1744-7348.1995.tb05004.x.

Lecoq, H., Moury, B., Desbiez, C., Palloix, P., & Pitrat, M. (2004). Durable virus resistance in plants through conventional approaches: a challenge. *Virus Research*, *100*, 31–39. doi:10.1016/j.virusres.2003.12.012.

Lecoq, H., & Pitrat, M. (1982). Effect on *Cucumber mosaic virus* incidence of the cultivation of partially resistant muskmelon cultivars. *Acta Horticulturae*, *127*, 137–145.

Lister, R. M., & Ranieri, R. (1997). Distribution and economic importance of Barley Yellow Dwarf. In C. J. D'Arcy, & P. Burnett (Eds.), *Barley yellow dwarf: 40 years of progress* (pp. 29–53). St Paul, USA: APS Press.

Lucio-Zavaleta, E., Smith, D. M., & Gray, S. M. (2001). Variation in transmission efficiency among *Barley yellow dwarf virus*–RMV isolates and clones of the normally inefficient aphid vector, *Rhopalosiphum padi*. *Phytopathology*, *91*, 792–796. doi:10.1094/PHYTO.2001.91.8.792.

Martin, B., Rahbé, Y., & Ferreres, A. (2003). Blockage of stylet tips as the mechanism of resistance to virus transmission by *Aphis gossypii* in melon lines bearing the Vat gene. *The Annals of Applied Biology*, *142*, 245–250. doi:10.1111/j.1744-7348.2003.tb00247.x.

Niemeyer, H. M. (1991). Secondary plant compounds in aphid-host interactions. In R. K. Campbell, & R. D. Eikenbary (Eds.), *Aphid–plant genotype interactions* (pp. 101–114). New York, USA: Elsevier.

Palloix, A., Daubèze, A. M., Lefebvre, V., Caranta, C., Moury, B., Pflieger, S., et al. (1997). Construction de systèmes de résistance aux maladies adaptés aux conditions de culture chez le piment. *Comptes-Rendus de l'Académie d'Agriculture de France*, *83*, 87–98.

Plumb, R. T. (1995). Epidemiology of barley yellow dwarf in Europe. In C. J. D'Arcy, & P. A. Burnett (Eds.), *Barley yellow dwarf 40 years of progress* (pp. 107–127). St Paul, MN, USA: APS Press.

Prado, E., & Tjallingii, W. F. (1994). Aphid activities during sieve element punctures. *Entomologia Experimentalis et Applicata*, *72*, 157–165. doi:10.1007/BF02383550.

Puterka, G. J., & Peters, D. C. (1990). Sexual reproduction and the inheritance of virulence in the greenbug, *Schizaphis graminum* (Rondani). In R. K. Campbell & R. D. Eikenbary (Ed.), *Aphid-plant genotype interactions* (pp. 289–318). New York, USA: Elsevier.

Rasmusson, D. C., & Schaller, C. W. (1959). The inheritance of resistance in barley to the yellow-dwarf virus. *Agronomy Journal*, *51*, 661–664.

Shukle, R. H., Lampe, D. J., Lister, R. M., & Foster, J. E. (1987). Aphid feeding behavior: Relationship to *Barley yellow dwarf virus* resistance in *Agropyron* species. *Phytopathology*, *77*, 725–729. doi:10.1094/Phyto-77-725.

Smith, C. M. (1989). *Plant resistance to insects: A fundamental approach*. New York, USA: Wiley.

Tatchell, G. M. (1989). An estimate of the potential economic losses to some crops due to aphids in Britain. *Crop Protection* (*Guildford, Surrey*), *8*, 25–29. doi:10.1016/0261-2194(89)90095-1.

Tjallingii, W. F. (1988). Electrical recording of stylet penetration activities by aphids. In A. K. Minks, & P. Harrewijn (Eds.), *Aphids their biology, natural enemies and control* (vol. 2B, (pp. 95–108)). Amsterdam, The Netherlands: Elsevier.

Torrance, L., Pead, M. T., Larkins, A. P., & Butcher, G. W. (1986). Characterization of monoclonal antibodies to a U. K. isolate of *Barley yellow dwarf virus*. *The Journal of General Virology*, *67*, 549–556. doi:10.1099/0022-1317-67-3-549.

Webster, J. A. (1991). Developing aphid-resistant cultivars. In R. K. Campbell, & R. D. Eikenbary (Eds.), *Aphid–plant genotype interactions* (pp. 87–100). New York, USA: Elsevier.

Wood, E. A., Sebest, E. E., Webster, J. A., & Porter, D. R. (1995). Resistance to wheat curl mite (Acari: Eriophydidae) in greenbug-resistant 'Gaucho' triticale and 'Gaucho × wheat crosses. *Journal of Economic Entomology*, *88*, 1032–1036.