Research Review No. 69

June 2008

Price: £4.50

Turnip yellows virus (syn *Beet western yellows virus*): an emerging threat to European oilseed rape production?

by

Mark Stevens, Graham McGrann and Bill Clark

Broom's Barn Research Centre, Higham, Bury St Edmunds
Suffolk IP28 6NP

This is the final report of a review lasting for four months which started in January 2008. The work was funded by a contract of £10,000 from HGCA (Project No. 3419).

Contents

Section Page no.

1. Summary

Nomenclature: The International Committee for the Taxonomy of Viruses (ICTV) approved the proposal to reclassify the European non-sugar beet infecting strains of *Beet western yellows virus* (BWYV) as an independent species within the genus *Polerovirus*, with the name *Turnip yellows virus* (TuYV) being accepted. Recent molecular evidence supported the separation of the beet-infecting and non-beet infecting isolates as two distinct viruses. European strains of *Beet western yellows virus* (BWYV) that infect oilseed rape in the UK should now be referred to as *Turnip yellows virus* (TuYV).

Turnip yellows virus (TuYV) is probably the most important, yet least understood, viral disease of oilseed rape in the United Kingdom. It is likely that TuYV is one of the principal reasons why commercial oilseed rape crops do not reach their genetic yield potential. The virus is probably present throughout the UK and at high levels in southern England. Virus symptoms, which are not readily recognisable, are usually not expressed before stem extension and can easily be confused with other stress symptoms and nutritional deficiencies.

The main virus vector is the peach-potato aphid, *Myzus persicae*. Annual sampling of *M. persicae* populations have shown that up to 72% of winged *M. persicae* carry TuYV. Studies in the 1980s and early 1990s showed that TuYV could be widely present in oilseed rape crops throughout the UK. Work at Broom's Barn Research Centre in the 90s showed that TuYV could decrease yields by up to 26%. All yield parameters including the number of primary branches, numbers of seeds per pod and percentage oil per seed were affected; the glucosinolate concentration in the oil was also significantly increased in infected plants. Recent work in Australia showed seed yield losses up to 46%. Estimates of yield loss indicate that at an individual crop level, control of TuYV could raise average yields from 3.3 t/ha to between 4.4 and 6.0 t/ha. If only half of those losses could be prevented (10-15%) by controlling TuYV, then the value of the yield improvement would be in the order of £100-150/ha (equivalent to £60-90 million per year to UK OSR growers).

Milder autumn and winter conditions favour the development of the aphid vectors and encourage virus spread. Climate change will exacerbate the situation as warmer conditions will encourage the survival and multiplication of *M. persicae* throughout the winter.

Strategies are required to decrease the impact of TuYV and its aphid vectors on the yield of oilseed rape. Cultural practices, seed treatments and foliar sprays provide an opportunity to limit the impact of TuYV in a responsible and sustainable manner. The levels of resistance to TuYV in current UK varieties are not known. The identification and exploitation of potential resistance genes provides an alternative strategy to control this important viral disease. Recent work in Germany identified a potential resistance gene that could be exploited to provide resistance to TuYV. Identification and exploitation of other sources of resistance to TuYV are required.

2. Introduction

2a. History and taxonomy of *Turnip yellows virus*

Beet western yellows virus (BWYV) was originally identified in the USA during the late 1950s as an important virus causing stunting and chlorosis in a wide range of plant species resulting in yield losses in crops such as sugar beet, spinach, lettuce and turnip (Duffus, 1977; Duffus, 1961).

Just prior to the identification of BWYV in the USA a similar virus, *Beet mild yellowing virus* (BMYV) had been characterised from sugar beet in the UK (Russell, 1958). In England a BWYV-like virus was subsequently found on hosts that had previously been reported as immune to BMYV, such as lettuce (Russell and Duffus, 1970). The BWYV-like virus was biologically and serologically similar to BWYV from the USA but clear differences were observed between the host ranges of the viruses, and European strains of BWYV did not infect sugar beet unlike its equivalent from the USA (Duffus and Russell, 1970).

This distinction between the European and USA isolates of BWYV led to considerable debate over their taxonomy, with names such as *Turnip yellows* virus (Schubert *et al*., 1998), *Brassica yellows virus* (Hauser *et al*., 2000a), and *Brassica yellowing virus* (Hauser *et* al., 2000b) being suggested for the non-beet infecting isolates. This led to the International Committee for the Taxonomy of Viruses (ICTV) approving the proposal to reclassify the non-sugar beet infecting strains of BWYV as an independent virus in the genus *Polerovirus*, family *Luteoviridae*; the name *Turnip yellows virus* (TuYV) was ratified by the committee (Mayo, 2002). Recent molecular evidence further supported the separation of the beet-infecting and non-infecting isolates as two distinct viruses (Stevens *et al*., 2005). Comparisons of the gene and protein sequences of European TuYV and beet-infecting BWYV (from the USA) highlight the differences between the two, supporting the biological data that TuYV and BWYV-USA are distinct viruses (Beuve *et al*., 2008; Hauser *et al*., 2000b). Therefore, to avoid confusion, in this review the Brassica-infecting BWYV-like virus will be referred to as TuYV in accordance with the ICTV judgement (Mayo, 2002).

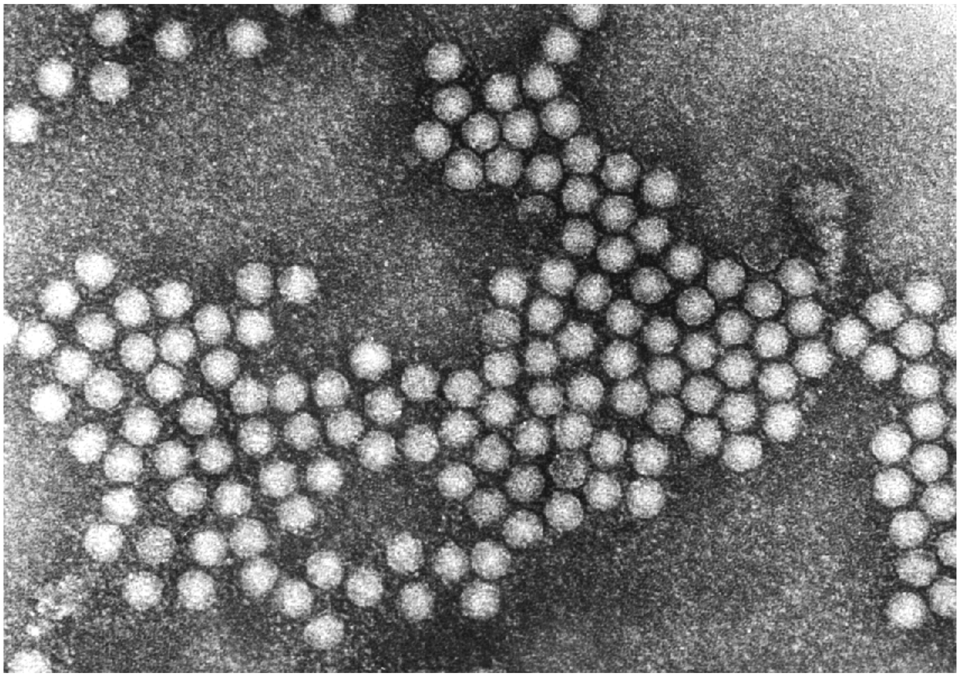

Fig. 1. Electron micrograph of virus particles of *Turnip yellows virus*.

2b. Host range of TuYV

TuYV has a wide host range and experimentally can infect species from at least 13 plant families, including many species of agronomic importance (Table 1). The virus is of particular interest as a pathogen of oilseed rape (Jay *et al*., 1999; Smith and Hinckes, 1985; Hill *et al*., 1989) but is also of economic importance in lettuce (Walkey and Payne, 1990; Walkey and Pink, 1990).

The diverse range of cultivated plants and weed species susceptible to TuYV increases the potential reservoir of hosts in which the virus can survive throughout the winter, and provides a source for future virus outbreaks (Stevens *et al*., 1994; Smith and Hinckes, 1985; Latham *et al*., 2003).

2c. Symptoms of TuYV

Oilseed rape plants infected with TuYV produce a wide range of symptoms – most of which go unnoticed as they resemble stress and nutrient-deficiency symptoms. This includes reddening of leaf margins and interveinal yellowing and reddening (Figures 2 and 3).

Table 1. Arable crop hosts of TuYV

Latin name	**Common name**	**Reference**
Brassicaceae		
Brassica juncea	Indian mustard	Graichen and Rabenstein, 1996
Brassica napus ssp. *napus*	Oilseed rape	Stevens *et al*., 1994
Brassica napus ssp. *rapifera*	Swede	Graichen and Rabenstein, 1996
Brassica oleracea var *acephala*	Kale	Stevens *et al*., 1994
Brassica oleracea var *alboglabra*	Chinese kale	Graichen and Rabenstein, 1996
Brassica oleracea var *botrytis*	Cauliflower	Stevens *et al*., 1994
Brassica oleracea var *capitata*	Cabbage	Stevens *et al*., 1994
Brassica oleracea var *capitata*	Cabbage	Stevens *et al*., 1994
Brassica oleracea var *gemmifera*	Brussels sprouts	Stevens *et al*., 1994
Brassica oleracea var *gongylodes*	Kohlrabi	Graichen and Rabenstein, 1996
Brassica oleracea var *italica*	Calabrese	Stevens *et al*., 1994
Brassica oleracea var *sabauda*	Savoy cabbage	Graichen and Rabenstein, 1996
Brassica rapa ssp. *chinensis*	Pak choi	Graichen and Rabenstein, 1996
Brassica rapa ssp. *narinosa*	Broadbeaked mustard	Graichen and Rabenstein, 1996
Brassica rapa ssp.*oleifera*	Turnip rape	Graichen and Rabenstein, 1996
Brassica rapa ssp. *parachinensis*	False pak choi	Graichen and Rabenstein, 1996
Brassica rapa ssp. *pekinensis*	Chinese cabbage	Graichen and Rabenstein, 1996
Brassica rapa ssp. *perviridis*	Spinach mustard	Graichen and Rabenstein, 1996
Brassica rapa ssp. *rapifera*	Turnip	Graichen and Rabenstein, 1996
Lepidium sativum	Garden cress	Graichen and Rabenstein, 1996
Raphanus sativus var. *niger*	Winter radish	Graichen and Rabenstein, 1996
Raphanus sativus var. *oleiformis*	Fodder radish	Graichen and Rabenstein, 1996
Raphanus sativus var. *sativus*	Cultivated radish	Graichen and Rabenstein, 1996
Sinapis alba	White mustard	Graichen and Rabenstein, 1996
Chenopodiaceae		
Spinacea oleracea	Spinach	Graichen and Rabenstein, 1996
Compositae		
Lactuca sativa	Lettuce	Walkey and Pink, 1990
Fabaceae		
Cicer arietinum	Chickpea	Graichen and Rabenstein, 1996
Lupinus albus	White lupin	Graichen and Rabenstein, 1996
Pisum sativum	Pea	Graichen and Rabenstein, 1996
Vicia faba	Broad bean	Graichen and Rabenstein, 1996

The wide host range of TuYV also includes many alternative hosts of which a large number are common arable weed species (Table 2).

Table 2. Alternative host species for TuYV (after Stevens *et al*. (1994), Graichen and Rabenstein (1996), Pallett *et al*. (2002), Thurston *et al*. (2001), Coutts *et al*. (2006)).

Latin name	**Common name**	**Latin name**	**Common name**
Asteraceae		**Fumarianceae**	
Conzya spp.	Fleabane	*Fumaria officinalis*	Common Fumitory
Matricaria perforata	Scentless mayweed	**Hydrophyllaceae**	
Brassicacae		*Phacelia tanacetifolia*	Scorpion weed
Arabidopsis thaliana	Thale cress	**Lamiaceae**	
Brassica carinata	Abyssinian cabbage	*Lamium amplexicaule*	Henbit deadnettle
Brassica nigra	Black mustard	*Lamium purpureum*	Purple deadnettle
Brassica rapa ssp. *rapa*	Field mustard	**Papaveraceae**	
Brassica rapa ssp. *sylvestris*	Wild turnip	*Papaver rhoeas*	Corn poppy
Capsella bursa-pastoris	Shepherd’s purse	**Polemoniaceae**	
Camelina sativa	Gold-of-pleasure	*Navarretia squarrosa*	Stinkweed
Lepidium campestre	Field pepperweed	**Portulaceae**	
Lepidium ruderale	Roadside pepperweed	*Montia perfoliata*	Miner's lettuce
Raphanus raphanistrum	Wild radish	**Primulaceae**	
Raphanus sativus var. *albus*		*Anagallis arvensis*	Scarlet pimpernel
Raphanus sativus var. *violaceus*	White radish	**Scrophulariaceae**	
Sinapis arvensis	Wild mustard	*Veronica arvensis*	Corn speedwell
Thlaspi arvense	Fanweed	*Veronica persica*	Common field speedwell
Caryophyllaceae		**Solanacecae**	
Stellaria media	Common chickweed	*Nicotiana benthamiana*	
Spergula arvensis	Corn spurry	*Nicotiana clevelandii*	Cleveland's tobacco
Compositae		*Nicotiana occidentalis*	
Chrysanthemum segetum	Corn marigold	*Physalis floridiana*	
Senecio vulgaris	Groundsel	*Physalis pubescens*	Hairy nightshade
Taraxacum officinale	Common dandelion	*Solanum nigrum*	Blackberry nightshade
Zinnia peruviana	Peruvian zinnia	**Urticaceae**	
Cucurbitaceae		*Urtica urens*	Annual nettle
Citrullus lanatus	Afghan (wild)	**Valerianaceae**	

	melon		
Fabaceae		*Valerianella locusta*	Lewiston cornsalad
Lupinus luteus	Yellow lupin	**Violaceae**	
Ornithopus sativus	Pink serradella	*Viola arvensis*	Field pansy
Trifolium resupinatum	Persian clover		

Fig. 2. Range of symptoms caused by natural infection with TuYV on leaves of oilseed rape collected from commercial crops during May.

Fig. 3. Leaf symptoms of TuYV in oilseed rape, April 2008. Plants were artificially inoculated with viruliferous aphids on 31 October 2007.

Many host plants infected with TuYV can show symptoms such as interveinal yellowing or reddening which may also be accompanied by dwarfing. Some weed species develop these distinctive symptoms when infected, for example, older leaves of shepherd’s purse (*Capsella bursa-pastoris*) become yellow, curled and brittle when infected with poleroviruses (Fig. 4), whilst *Montia perfoliata* (Miner’s lettuce) turn red (Fig. 5). Spinach leaves develop mild yellowing discolouration in interveinal areas and at the leaf tip whilst lettuce shows chlorotic blotching which later develops into severe interveinal yellowing. The leaves may also be thicker and brittle and the plants may be stunted.

Fig. 4. Typical symptoms on *Capsella bursa-pastoris* 10 weeks post-inoculation.

Fig. 5 Leaf reddening symptoms in *Montia perfoliata*.

Many plant species can remain symptomless when infected and this can be the case with oilseed rape. However, infected plants tend to show red or purple discolouration initially on older leaves, but symptoms can extend to all leaves by early summer (Fig. 2 and 3). Many weed species can also remain symptomless, with the result that infections may remain unnoticed, which has implications for the control and epidemiology of the disease. Also, symptoms typical of TuYV can be confused with nutrient deficiency, water stress, frost damage or even natural senescence. In England, between 1968 and 1970, many lettuce crops were thought to be suffering from magnesium deficiency, when actually they were infected with TuYV (Tomlinson, 1987).

Diagnostic methods available for TuYV

With any asymptomatic infection the need for and exploitation of diagnostic methods to confirm the presence and titre of the viral infection is crucial, both in host plants and individual aphids. A range of antibodies have been developed against the beet poleroviruses (including TuYV) for use in ELISA (D'Arcy et al., 1989; Smith et al., 1996), and molecular techniques such as riboprobes and RT-PCR are also available (Lemaire *et al*., 1995; Jones *et al*., 1991; Hauser *et al*., 2002).

3. Epidemiology of TuYV

3a. TuYV transmission

TuYV is vectored by a wide range of aphid species (Table 3) in a persistent (circulative, non-propagative) manner. Therefore, once acquired, aphids retain the ability to transmit the virus, even after moulting, although the virus does not pass through to their progeny (Schliephake *et al.*, 2000; Stevens *et al.*, 2006). TuYV is not thought to be mechanically or seed transmissible as TuYV is confined to the vascular tissue and virus particles are unable to enter the ovule as this has no vascular connectivity with the parent plant. Jay (1999) used ELISA to detect TuYV in the pod components of oilseed rape seeds and was able to find virus in the pod wall, septum and seed coat, but in only two out of 78 embryos. In further studies at Broom's Barn Research Centre where seed from infected plants was sown in aphid-proof growth chambers, no virus could be detected in plants up to 12 weeks after germination.

Although many aphid species have been shown to transmit TuYV, most of these do not usually exploit oilseed rape as a host under natural conditions and therefore are not likely to be important vectors. *Myzus persicae*, the peach-potato or green-peach aphid, is considered and regarded as the main vector of TuYV (Fig. 6) (Stevens *et al.*, 1995).

As TuYV is phloem limited, aphids need to ingest infected sap to acquire the virus particles. For successful transmission to occur the TuYV virus particles have to pass through two barriers within the aphid: the gut wall and the accessory salivary gland membrane. Virus particles first enter the aphid's body via ingestion of infected sap, then are transported across the gut wall to the haemocoel, and finally accumulate in the accessory salivary gland. The particles are then injected into the plant during penetration of the aphid's stylet during feeding.

The virus acquisition access period is determined by the time taken for the aphid's stylet to reach the phloem of the infected source plant and this can be as short as 15 minutes. However, acquisition of virus particles and subsequent transmission of both *Potato leaf roll virus* (PLRV) and *Barley yellow dwarf virus* (BYDV) increases with time. These principles apply to TuYV as both these viruses are closely related to the brassica-infecting virus. The latent period (i.e. the time taken between an aphid acquiring the virus before being able to transmit the particles to a new host) is usually at least 24 hours and can be as long as four days. However, once an aphid is able to transmit the virus to a new host the inoculation access period can be as short as 10-30 minutes for most poleroviruses, but again the efficiency of transmission increases the longer the aphid feeds on the plant as highlighted above. All these factors affect the development and exploitation of control strategies for limiting both primary infection and secondary spread of TuYV in oilseed rape.

Table 3. Aphid species demonstrated to vector TuYV (based on data of Schliephake *et al.*, 2000)

Latin name	**Common name**
Acyrthosiphon pisum (green race)	Green pea aphid
Aphis gossypii	Cotton aphid
Aulacorthum circumflexum	Lily aphid
Aulacorthum solani	Foxglove aphid
Brachycorynella asparagi	Asparagus aphid
Brevicoryne brassicae	Cabbage aphid
Cavariella aegopodii	Carrot aphid
Macrosiphoniella sanborni	Chrysanthemum aphid
Macrosiphum albifrons	Lupin aphid
Macrosiphum euphorbiae	Potato aphid
Myzus nicotianae	Tobacco aphid
Myzus persicae	Peach-potato aphid
Nasonovia ribisnigri	Currant lettuce aphid
Pentatrichopus fragaefolii	Strawberry aphid
Rhopalosiphum maidis	Corn aphid
Rhopalosiphum padi	Oat aphid
Sitobion avenae	Grain aphid

The transmission rate of TuYV can be influenced by many factors such as the aphid species, clone or biotype and/or virus strain. Abiotic factors such as temperature and humidity may also influence the ability of aphids to transmit the viruses they carry. For example, changes in temperature affect aphid behaviour during virus acquisition and/or inoculation and also influence the host plant as a source or its suitability to become a new virus host.

Fig. 6. *Myzus persicae*, the peach-potato aphid, the most important vector of TuYV.

Myzus persicae is a highly efficient vector of TuYV with transmission rates of over 90% having been reported experimentally (Schliephake *et al*., 2000). Sampling of *M. persicae* populations in either the network of suction traps operated by the Rothamsted Insect Survey (Fig. 7) or yellow water traps positioned in sugar beet fields, between May and August, as part of the aphid warning scheme for virus yellows control (Fig. 8), have shown that up to 72% of winged *M. persicae* carry TuYV (Stevens *et al*., 2008; Stevens *et al*., 1995). The number of viruliferous *M. persicae* varies from year to year and from region to region depending on weather conditions which influence the survival and fecundity of aphid populations; the weather will also impact on the number of available sources of the virus.

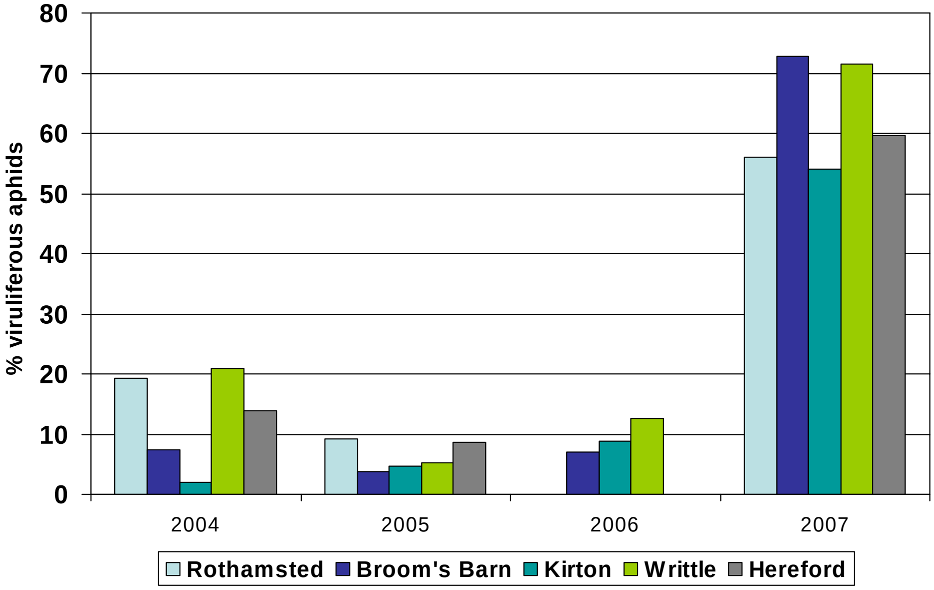

Fig. 7. Percentage TuYV content of *M. persicae* caught in five suction traps between 2004 and 2007.

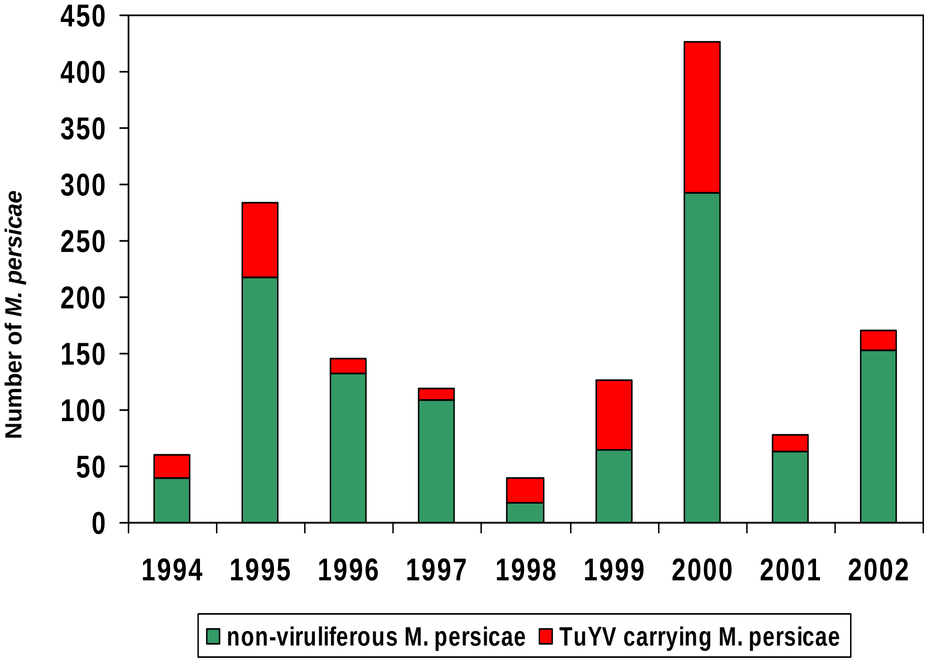

Fig. 8. Number of *M. persicae* caught in yellow water pans in sugar beet crops at Broom's Barn Research centre between 1994 and 2002 and the number carrying TuYV.

It is important to stress that when up to 72% of winged *M. persicae* carry (and potentially transmit) TuYV it becomes extremely difficult to prevent widespread primary infection of host crops with virus such as oilseed rape even with the extensive use of seed treatments and/or aphicide spays. Aphid risk maps, to highlight those areas of production at greatest risk from potential virus infection, are used by the sugar beet industry. A summary of these data for 2007 between April and July are shown in Fig. 9.

Other aphid species that play an important but more limited role in the transmission of TuYV are *Macrosiphum euphorbiae* and *Brevicoryne brassicae*. However, these species have much lower rates of virus transmission (Schliephake *et al*., 2000), fewer individuals tend to carry the virus (Stevens *et al*., 1995) and work by Herrbach (1994) has shown that French clones of *B. brassicae* were unable to transmit the virus. Not only are these aphid species a problem as vectors of TuYV but, along with *M. persicae,* are also able to cause some damage to oilseed rape crops by direct feeding action (Jones *et al*., 2007), although this may be specific to a Mediterranean environment.

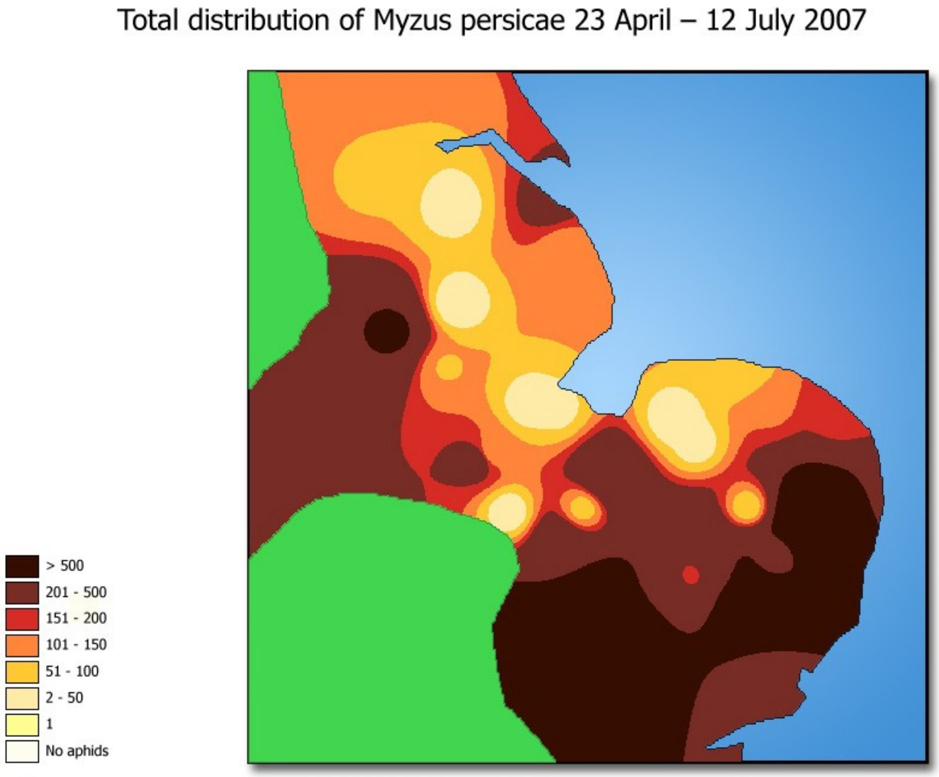

Fig. 9. *M. persicae* distribution map in sugar beet crops for 2007.

3b. Incidence of TuYV infection in oilseed rape crops

The incidence of TuYV in an oilseed rape crop depends on aphid flight during that season whilst spread of the virus will be determined by the abundance and movement of its aphid vectors (both winged and wingless) within the crop (Walsh and Tomlinson, 1985). In Germany, high flight activity of aphids during the autumn of 1995 was followed by high levels of virus infection in the winter oilseed rape crop during 1995/1996 (Graichen and Schliephake, 1999; Graichen *et al*., 1997).

Smith and Hinckes (1985) showed that by mid-October 33% of the plants in an oilseed rape crop were already infested with *M*. *persicae* and 5% of the plants had TuYV. The highest number of plants infested with *M*. *persicae* was found in December, but the aphid population then became undetectable until March. However, during this period the incidence of TuYV increased to approximately 50% infection by the end of April.

Previously, levels of TuYV incidence in oilseed rape crops appear to have been variable ranging from less than 10% to up to 85% infection (Hill *et al*., 1989; Walsh *et al*., 1989; Njuguna *et al*., 1986; Nagarajan *et al*., 1987; Jay *et al*., 1999). Hardwick *et al*. (1994) reported that average plant infection rates of TuYV ranged between 49-73% in the UK during the early 1990s. More recently, in a series of national field trials conducted by Bayer CropScience in 2006-7, the incidence of TuYV in untreated crops ranged from 30 to 100%.

In further studies supported by the British Beet Research Organisation (BBRO) aphid numbers were monitored in oilseed rape crops at up to seven locations within East Anglia between 2004 and 2007. These data were collected to determine the number of aphids surviving through the winter that then may migrate and pose a risk to the following sugar beet crop in the spring; the levels of TuYV infection in the oilseed rape crops were also assessed (Fig 10). These data show that large numbers of *M. persicae* overwintered in oilseed rape crops during the winter of 2006/07 leading to extensive spread of TuYV by the following spring. Again high numbers of *M. persicae* (over one per plant) are known to have survived on crops during the 2007/08 winter.

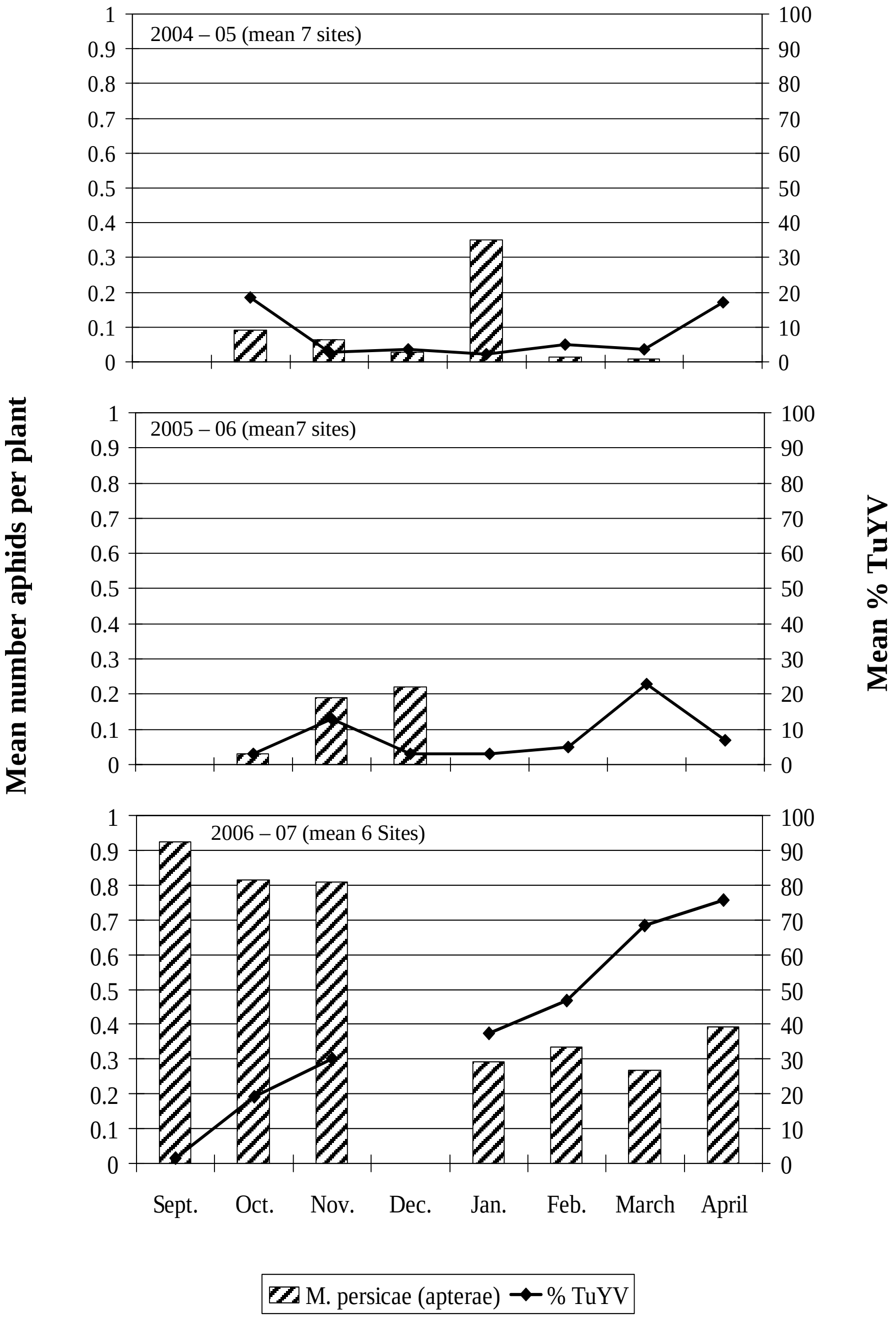

Fig. 10. Mean abundance of *M. persicae* and levels of TuYV infection in oilseed rape crops in East Anglia between 2004 and 2007.

In general the incidence of TuYV within an oilseed rape crop tends to increase from the initial autumn infection to a maximum level in the following March or April.

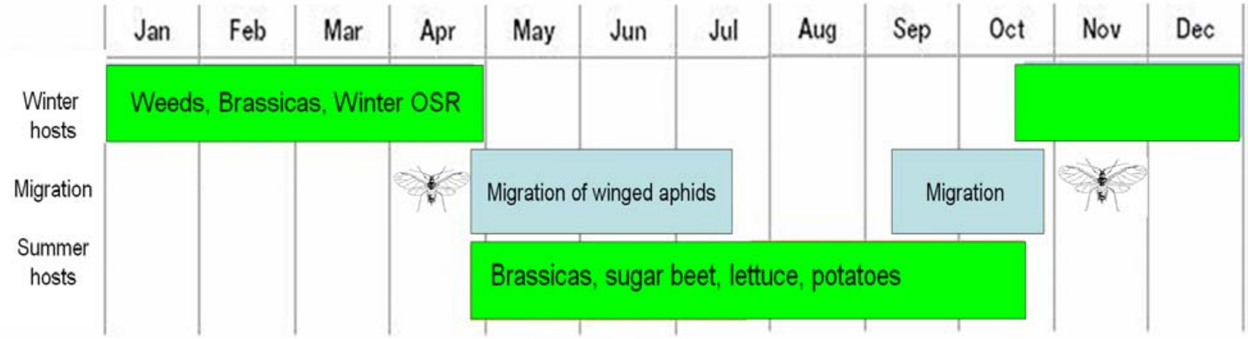

Fig 11. Typical pattern of survival and spread of *M. persicae* in the UK

As with most insect-borne viruses of the northern hemisphere the severity of winter plays an important role in virus distribution and incidence. Mild winters tend to encourage the multiplication and spread of the aphid vectors of TuYV such that the following year's crop is likely to exhibit a higher incidence of virus (Hill *et al*., 1989; Stevens *et al*., 2008). Typical August or September sowings of oilseed rape result in an emerged crop that coincides with the autumn aphid migrations (Fig. 11), providing a suitable overwintering habitat for the vectors of TuYV (Walsh and Tomlinson, 1985). The effect of sowing date on the incidence of TuYV appears to vary by year as in some years, late-sown oilseed rape crops have lower virus incidence (Njuguna *et al*., 1986) whilst in other years there was no difference in virus incidence regardless of sowing date (Nagarajan *et al*., 1987).

TuYV appears to be distributed quite widely throughout the UK with infected crops having been identified from the north, west, east, south and central parts of England as well as from Scotland and Wales (Hill *et al*., 1989; Stevens *et al*., 2008; Smith and Hinckes, 1985). The survey data from Hill *et al*. (1989) also highlighted a general trend of higher incidence of TuYV in crops from the south and south west of the UK over the four years they investigated. However, following a mild winter period the levels of virus incidence appeared to be higher throughout the UK. With its national distribution TuYV can be considered one of the most common viruses of oilseed rape in the UK (Hardwick *et al*., 1994).

4. Worldwide distribution of TuYV

Turnip yellows virus (or BWYV in most cases) has been reported as infecting oilseed rape crops in the UK (Gilligan *et al*., 1980; Smith and Hinckes, 1985), Germany (Schröder, 1994), France (Kerlan, 1991), Czech Republic (Polak and Majkowa, 1992), Austria (Graichen *et al*., 2000), Serbia (Jasnic and Bagi, 2007), Iran (Shahraeen *et al*., 2003), Australia (Coutts *et al*., 2006) and the USA (Duffus and Russell, 1970; 1972). To clarify the issue of specifically which virus was present in Europe, host range investigations were done both in the UK and Germany.

Stevens *et al*. (1994) showed that <u>none of the TuYV strains</u> from within the UK from different brassica crops including oilseed rape, cabbage, Brussels sprouts, Kale, calabrese, cauliflower or red cabbage <u>was able to infect sugar beet</u>. Graichen and Rabenstein (1996) examined collections of strains from Germany, France, UK and New Zealand that had been isolated from oilseed rape, cabbage, Brussels sprout and spinach. Their research also showed that not one of these virus isolates from *Brassica* species was able to infect sugar beet whilst all were transmitted to oilseed rape; a result that led them to conclude that the European luteoviruses of the genus *Brassica* represent isolates of TuYV (Graichen and Rabenstein, 1996).

It is most likely that the virus infecting oilseed rape in Europe is TuYV but this may not necessarily but the case in other continents. Recently the genetic sequence of an Australian isolate of BWYV has been shown to more closely resemble that of the European TuYV than BWYV-USA (Coutts *et al*., 2006). Until more isolates have been compared the possibility that both TuYV and BWYV are present in Australia cannot be ruled out (Jones *et al*., 2007). TuYV in oilseed rape has not been confirmed in the USA and it is unclear what brassica-infecting poleroviruses are in the US. Until multiple isolates of brassica-infecting poleroviruses from countries where BWYV has been reported, such as Spain (Moreno *et al*., 2004), China (Xiang *et al*., 2007 <u>http://www.bspp.org.uk/NDR/july2007/2007-16.asp</u>) and Iran (Shahrareen *et al*., 2003) have been sequenced then the full extent of the worldwide distribution of TuYV cannot be confirmed.

5. Impact of TuYV on oilseed rape yield

TuYV infects the leaves and stems of the oilseed rape plant as well as the components of the pods including the pod wall, the septum and the seed coat (Jay *et al*., 1999; Walsh and Tomlinson, 1985). Plants infected with TuYV can be slightly stunted, have reduced leaf area per plant and produce fewer primary branches (Jay *et al*., 1999). TuYV infected plants also tend to produce fewer seeds per pod which are heavier and larger than the seeds of uninfected plants (Jay *et al*., 1999).

The effects of TuYV on the yield of oilseed rape vary between reports and appear to be dependent on both the variety examined (Walsh *et al*., 1989) and the incidence of virus infection in the trial (Jay *et al*., 1999). Smith and Hinckes (1985) demonstrated that experimental plots of oilseed rape with 100% TuYV infection yielded approximately 10% less seed and 13.4% less oil than plots with 18% virus infection. These losses equated to an approximate 0.3 t ha^{-1} loss in seed yield. Seed production in single plants of oilseed rape genotypes could be reduced by between 40-50% despite the relatively mild symptoms caused by virus infection (Schröder, 1994); whilst Walsh *et al*. (1989) reported no significant differences between seed yields of infected and uninfected plants. Jay *et al*. (1999) showed that losses in seed yield caused by TuYV infection ranged from 11-26% and that in some years, infection could significantly lower the oil content by more than 2% and increase the glucosinolate levels by almost 14%. Oilseed rape losses can be further compounded by mixed infections between TuYV and viruses such as *Cauliflower mosaic virus* (Walsh and Tomlinson, 1985).

In Germany, commercial oilseed rape varieties with 90-100% TuYV infection have been reported to yield between 12% and 34% fewer seeds than plants in trials that were almost virus free (Graichen and Schliephake, 1999). Whilst in Australia sites that had 97-98% TuYV incidence in their crops suffered yield losses of up to 46% (Jones *et al*., 2007). The yield losses in Australia could be attributed to fewer seeds being produced by infected plants with up to 3% diminished oil content and an increase in erucic acid, two factors which reduce the seed quality of oilseed rape. Furthermore, this research was able to suggest that a 1% increase in TuYV incidence in the crop could result in a seed yield loss of 6-12 kg ha^{-1} (Jones *et al*., 2007).

The research to date clearly indicates that TuYV decreases the yield of oilseed rape and the effects of this virus may be one the main reasons why oilseed rape in the UK has not reached its full genetic yield potential (Fig. 12). Experiments in the UK have shown that TuYV can cause up to 26% yield loss. Recent Australian research has demonstrated much higher losses, up to 45%. In seasons with mild winters and aphid activity in the autumn, similar losses may be occurring in the UK. These estimates of loss indicate that at an individual crop level, control of TuYV could raise average yields from 3.3 t/ha to between 4.4 and 6.0 t/ha. If we take a cautious approach and assume less than half of those losses could be prevented (10-15%) by controlling TuYV, then the value of the yield improvement would be in the order of £100-150/ha (equivalent to £60-90 million per year to UK OSR growers). Even if mild winter conditions suitable for TuYV spread only occurred 50% of the time, then the value of yield improvements would be in the order of £30-40 million per year.

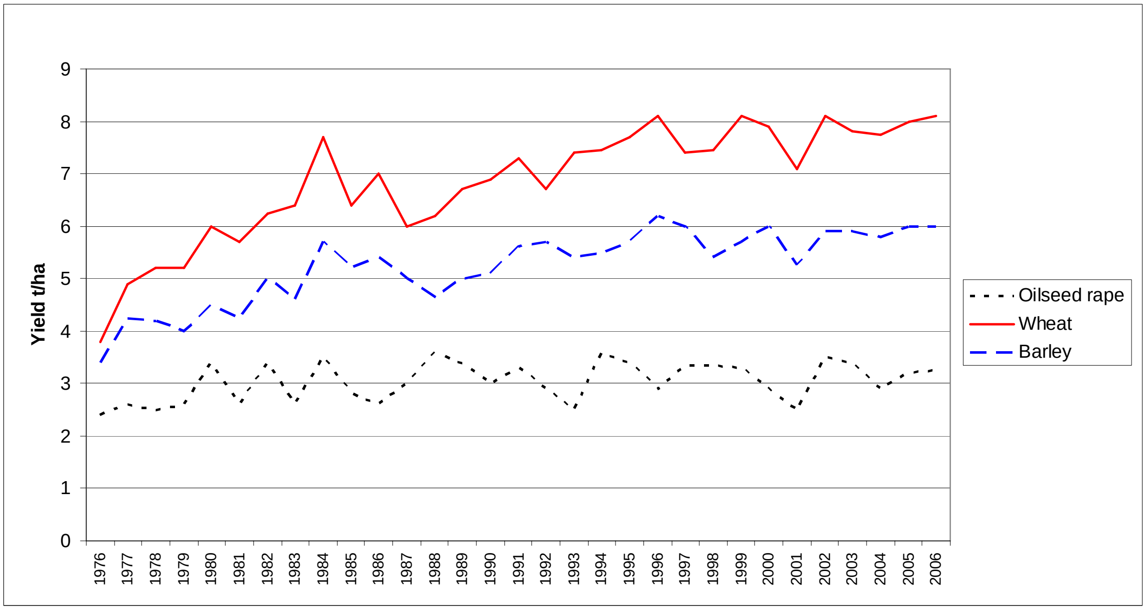

Fig. 12. UK Crop yields of wheat, barley and oilseed rape 1976-2006 (Defra statistics)

6. Impact of TuYV on other brassica crops

Although oilseed rape is the most important host of TuYV, the wide host range of this virus means it has the potential to be economically damaging for a number of other brassica crops. TuYV causes only very mild vein clearing symptoms on leaves of white cabbage (*Brassica oleracae* var *capitata*) but is still able to cause significant reductions (15-20%) to the mean trimmed head weights of commercial varieties (Hunter *et al*., 2002). However, when TuYV co-infection with other viruses such as *Cauliflower mosaic virus* or *Turnip mosaic virus* occurs these losses can be more than doubled and TuYV has now been associated with ‘tipburn’, a storage disorder of white cabbage (Hunter *et al*., 2002).

TuYV is also a potential threat to lettuce production and became prevalent in the UK during the 1970s and 1980s through severe outbreaks in the east and south-east of England (Tomlinson, 1972; Tomlinson and Walkey, 1973). The virus usually manifests itself in the crop a few weeks before cutting with the outer leaves showing strong inter-veinal yellowing that can lead to the plant becoming entirely yellow or even white in colour with dark brown necrosis of the margins of the older leaves (Tomlinson, 1972). Yield losses in some lettuce varieties have been reported to be as high as 63% whilst maturation can be reduced by up to 38% (Walkey and Pink, 1990). These losses can be further amplified during mixed viral infections between TuYV and viruses such as *Lettuce mosaic virus* or *Cucumber mosaic virus* (Walkey and Payne, 1990). The development of TuYV symptoms could be controlled by spraying crops with carbendazim (Tomlinson *et al*., 1976) but this practice was never adopted commercially. Early attempts to identify sources of genetic resistance in lettuce against TuYV had mixed results (Walkey and Pink, 1990; Watts, 1975). Although field resistance against the virus was found, the levels of control were not high. However, two related wild *Lactuca* species, *L. perennis* and *L. muralis*, were shown to have extreme resistance or possible immunity against TuYV, but sterility barriers between these species and commercial breeding lines meant that these sources of resistance were not exploited further (Walkey and Pink, 1990).

7. Strategies for control of TuYV in oilseed rape crops

7a. Chemical control of the vectors

Chemical control of the insect vectors is a common approach used worldwide and a key strategy to limit the impact of TuYV on yield. Targeted autumn sprays would be required for aphid control in oilseed rape and aphids would need to be controlled as soon as they were identified in the crop, possibly with the use of a second spray if necessary. Preliminary work during the 1980s demonstrated the potential of insecticides for the control of TuYV in oilseed rape. In field trials between 1985 and 1988 Read and Hewson (1988) showed that treatment of commercial winter oilseed rape crops with 6.25 g a.i ha^{-1} deltamethrin reduced virus incidence. Hill *et al*. (1989) demonstrated that complete control of aphids could be achieved by either weekly applications of pirimicarb or a single application of deltamethrin at the two true-leaf stage. This work also highlighted the effect of insecticide treatments on yield, indicating that positive significant yield effects could be obtained with a spray regime of deltamethrin. Further studies by Walsh *et al*. (1989) showed that the granular carbamate insecticide carbofuran did not control TuYV, whilst foliar sprays of the pyrethroid, lambda-cyhalothrin were able to reduce the incidence of TuYV by 86% and 65% in a trials conducted in 1986 and 1987. These levels of control were similar to those reported by others (Smith and Hinckes, 1985; Njuguna *et al*., 1986; Nagarajan *et al*., 1987).

Use of insecticide seed treatments, such as imidacloprid, clothianidin and thiamethoxam have revolutionised aphid control in sugar beet (Dewar and Cooke, 2006) decreasing the impact of the yellowing viruses which can decrease beet yields by up to 28%.

Recently, experiments have been conducted with a clothianidin-based seed treatment for oilseed rape (Adam and Hopkinson, 2008; Stevens et al., 2008).

Fig. 13. Trial plots of oilseed rape (left) protected by the clothianidin and beta-cyfluthrin based seed treatment and (right) untreated.

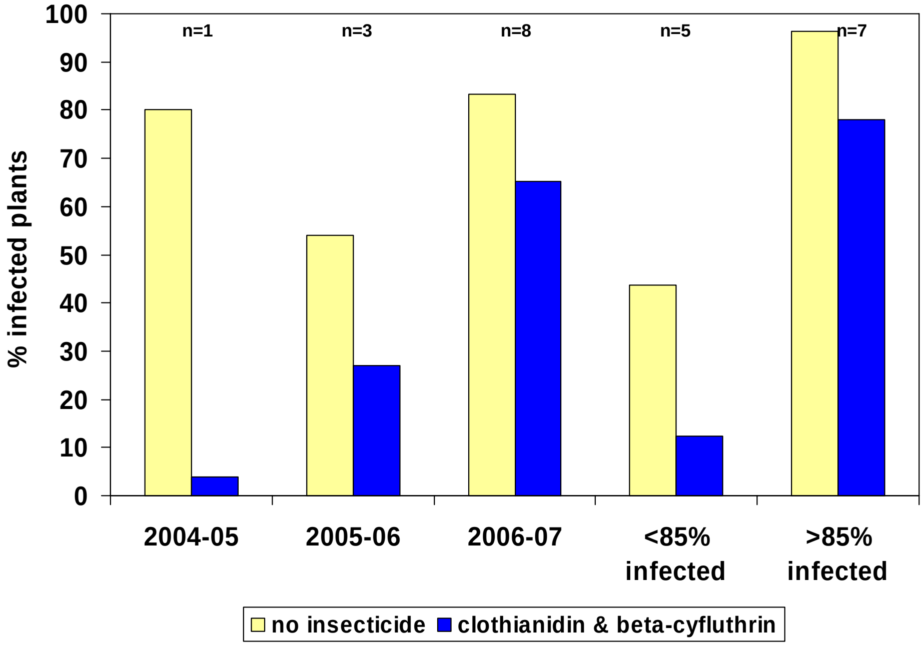

Fig. 14. The impact of a clothianidin-based seed treatment on the incidence of TuYV (measured in the spring of harvest each year) in trials between 2004 and 2007.

The new neonicotinoid seed treatment (based on clothianidin) is designed to protect the crop against aphids and other invertebrate pests and may be available in the UK in 2008. The only other insecticide seed treatment available to UK growers is based on imidacloprid and beta-cyfluthrin (Chinook) which is primarily aimed at flea beetle control at the cotyledon growth stage; the rate of imidacloprid in this product is 200g a.i./100kg of seed. The clothianidin-based seed treatment offers the potential of protecting the crop against virus-carrying aphids during the autumn months before colder weather appears. However, it will not provide protection against re-infection throughout winter and early spring following mild winters. In recent experiments the levels of the virus in untreated crops were generally high as three of the four trials were conducted in years where the winter was mild. Yield increases ranged from 2% where virus incidence in untreated crops was relatively low, to 30% when virus incidence was high (Stevens *et al*., 2008; Figs 13, 14 and 15).

Seed treatments of imidacloprid have been used in Australia to control TuYV infections. Applications of imidacloprid at a rate of 525g a.i./100 kg of seed was shown to dramatically increase seed yield by between 84-88%, further demonstrating the usefulness of such treatments to protect crops from TuYV by targeting the vector (Jones *et al*., 2007). Thus, the rate of imidacloprid used for aphid control in Australia is more than double the rate used in the UK in Chinook, which is not registered for aphid control in the UK, but used specifically for flea-beetle (see above). The use of seed treatments appears to be a promising measure for use in current integrated control strategies against pests and diseases of oilseed rape (Kazda *et al*., 2005).

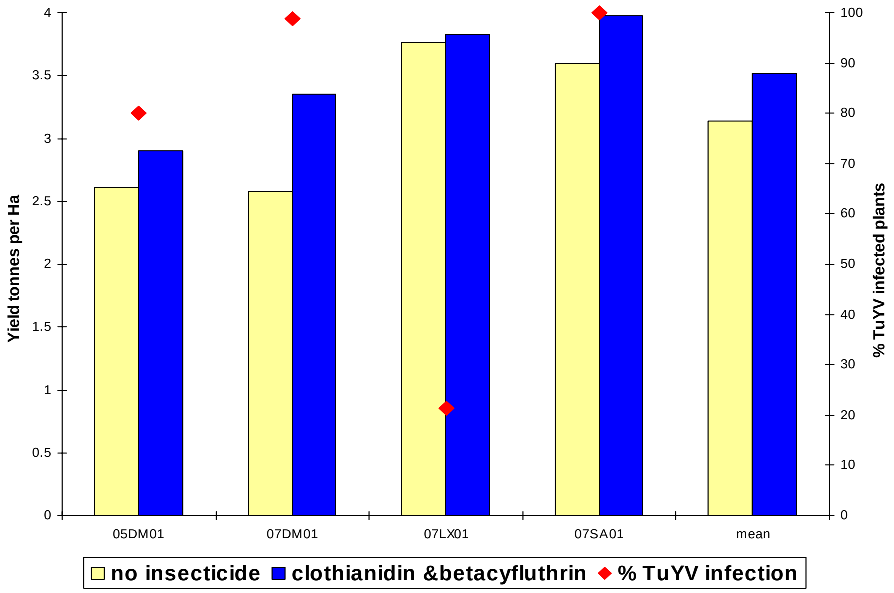

Fig. 15. Seed treatment effect on oilseed rape yield and percentage TuYV infection at four trial sites in 2006/07.

7b. Insecticide resistance

Myzus persicae has three well-defined insecticide resistance mechanisms which impact on the efficacy of insecticide treatments applied to control them. These mechanisms are esterase resistance, modified acetylcholinesterase (MACE) and knockdown resistance (kdr). The overproduction of the insecticide-detoxifying carboxylesterases (esterase resistance) via modification of genes encoding theses enzymes provides broad-spectrum resistance to organophosphates (OPs) and low resistance to carbamates and pyrethroids (Foster *et al*., 2007). Changes in site-specific insecticide targets have generated both modified acetyl cholinesterase (MACE) and knockdown resistance (kdr). These alterations have conferred strong resistance to the di-methyl carbamates, pirimicarb and pyrethroids respectively. The incidence and abundance of these resistance mechanisms in UK populations of *M. persicae* are monitored annually via the suction traps operated by the Rothamsted Insect Survey and the data can be used to warn growers of current risks leading to changes in insecticide recommendations. Year to year fluctuations in resistance can occur as seen with the rise in MACE and kdr resistance over the last five years. This has important implications for the future control of TuYV in oilseed rape.

As highlighted in the previous section, the neonicotinoid insecticides offer great potential for oilseed rape growers. These compounds act as antagonists at the nicotinic acetylcholine receptor in the post-synaptic membrane of insects and mimic the mode of action of nicotine. This group of insecticides which includes imidacloprid, thiacloprid, thiamethoxam and clothianidin have become extremely popular for controlling aphid pests in the northern hemisphere and are now registered for use on a wide range of arable and horticultural crops. However, there are already examples of resistance to the neonicotinoids around the world including whiteflies, leafhoppers and the Colorado beetle. Further work at Rothamsted Research has shown that *M. persicae* remains susceptible to the neonicotinoid treatments (either as seed treatments or foliar sprays) applied in the UK. Although there is some variation in the response of *M. persicae* populations to this chemical group, there is no field resistance observed as yet.

The use of insecticide seed treatments does offer real potential for controlling TuYV in oilseed rape crops. However, the widespread use of some pesticides may become increasingly restricted following current negotiations between EU countries on the proposed replacement of EU regulations 91/414 which could lead to some chemicals being withdrawn from use (Jones *et al*., 2007). New restrictions on insecticide use coupled with increasing risks of insecticide resistance in aphid populations (Foster *et al*., 2007) means that alternative control strategies need to be investigated.

Currently, the only approved aphicides for use in oilseed rape are pirimicarb and the pyrethroids deltamethrin, lambda-cyhalothrin, tau-fluvalinate, and lambda-cyhalothrin plus pirimicarb. In light of the high levels of MACE and kdr resistance in *M. persicae* clones, these chemicals will not control up to 80% of current aphid populations. Alternative products (or evaluation of existing chemistry not registered for oilseed rape) are required that could be exploited to protect autumn-sown oilseed rape crops from virus infection.

7c. Plant breeding for TuYV resistance in oilseed rape

In Germany, field and glasshouse experiments were conducted to investigate potential sources of resistance to TuYV from over 650 genotypes of summer and winter oilseed rape varieties, breeding lines and resynthesized rape forms (Graichen and Peterka, 1999). All of the oilseed rape genotypes were susceptible to TuYV infection except a single resynthesized rape form, called R54. The R54 line was crossed with three different TuYV susceptible varieties and subsequent progeny plants were tested for their reaction to the virus by field inoculation with viruliferous aphids. The results of these trials showed that 15 progeny populations were completely free of virus whilst other populations showed much reduced levels, highlighting that the TuYV resistance of R54 was heritable (Graichen and Peterka, 1999). A major quantitative trait locus involved in TuYV resistance from the R54 source was identified and molecular markers that could be used to assist TuYV resistance breeding have been described (Dreyer *et al*., 2001).

The successful transfer of TuYV resistance to susceptible oilseed rape varieties shows that there is potential for the use of genetic resistance in the control of this virus. However, further screening of worldwide oilseed rape germplasm is required to exploit different sources of resistance to TuYV to maintain a diverse gene pool for plant breeding purposes.

The search for natural sources of resistance to TuYV in Brassica germplasm is clearly an important goal for oilseed rape breeders. However, with current attitudes to plant genetic engineering appearing to change, the use of transgenics may be an alternative approach to controlling this virus. Exploitation of pathogen-derived resistance to the *Luteoviridae* is an area that has been investigated widely and there are excellent examples of immunity achieved by this approach, for example BYDV (Wang *et al*., 2000). Current research at Broom's Barn Research Centre is investigating pathogen-derived resistance to BMYV using a range of different virus sequences. As BMYV and TuYV are closely-linked, the findings of these studies, particularly as they are being undertaken in *Arabidopsis* (a common host for both these viruses), could be of benefit for oilseed rape breeders in the future.

8. Conclusions

Turnip yellows virus (TuYV), formerly known as *Beet western yellows virus* (BWYV), is probably the most important yet least understood viral disease of oilseed rape in the United Kingdom and may be one of the principal reasons why commercial oilseed rape crops do not reach their genetic yield potential. Studies in the 1980s and early 1990s showed that TuYV can be widely present in oilseed rape crops in the UK. However, virus symptoms, which are not readily recognisable, are rarely expressed before stem extension and can be confused with other stress-related symptoms and nutritional deficiencies. This partly explains why the disease is not widely recognised by farmers and advisers.

The main virus vector is the peach–potato aphid, *Myzus persicae*.

Experiments conducted in the UK have shown that TuYV can decrease final yield by up to 26%. All yield parameters including the number of primary branches, numbers of seeds per pod and percentage oil per seed were affected; the glucosinolate concentration in the oil was also significantly increased in infected plants. Recent Australian research has demonstrated much higher losses, up to 45%. In seasons with mild winters and aphid activity in the autumn, similar losses may be occurring in the UK. These estimates of yield loss indicate that at an individual crop level, control of TuYV could raise average yields from 3.3 t/ha to between 4.4 and 6.0 t/ha. If only half of those losses could be prevented (10-15%) by controlling TuYV, then the value of the yield improvement would be in the order of £100-150/ha (equivalent to £60-90 million per year to UK OSR growers).

Warmer autumn and winter conditions favour the migration and development of the aphid vectors and encourage virus spread. Climate change will exacerbate the situation as warmer conditions will encourage the survival and multiplication of *M. persicae* throughout the winter, similar to the seasons of 06/07 and 07/08.

The current status of resistance to TuYV in UK oilseed rape varieties is unknown.

Insecticide resistance is widespread, particularly resistance to the di-methyl carbamates, pirimicarb and pyrethroids. The incidence and abundance of these resistance mechanisms in UK populations of *M. persicae* have important implications for future control of TuYV in oilseed rape. The neonicotinoid insecticides (e.g. imidacloprid, thiacloprid, thiamethoxam and clothianidin) offer great potential for oilseed rape growers. There are variations in the response of *M. persicae* populations to the neonicotinoids but currently no field resistance has been observed.

Integrated strategies are likely to be required to decrease the significance of TuYV and its aphid vectors on the yield of oilseed rape in the UK. Cultural practices, resistant varieties, seed treatments and foliar sprays provide an opportunity to limit the impact of this disease in a responsible and sustainable approach. Control of this disease should raise yields of oilseed rape significantly and increase the profitability of the crop markedly.

9. Recommendations

There are many unanswered questions regarding the epidemiology of TuYV in oilseed rape, its impact on the yield of current and future varieties in the UK and the ability of the industry to prevent initial infection and subsequent spread of TuYV by aphid vectors.

To raise current yields it is essential that the effects of TuYV are limited by cultural practices, insecticides and/or resistant varieties. It is likely that an integrated approach to controlling this disease will be necessary for the future success of oilseed rape production in the UK.

The current resistance status of UK oilseed rape varieties to TuYV is unknown. A primary goal should be to test the reaction of oilseed rape varieties currently grown in the UK to TuYV infection and investigate the potential of any of these genotypes for resistance to the virus. If there is no identifiable resistance in the current gene pool then efforts should be made to identify novel sources that can be prospectively bred into elite oilseed rape varieties.

Research is required to gather data to indicate the actual distribution of the virus in the UK and equate this to the ‘in field’ losses caused by TuYV infection.

The potential for chemical control of the aphid vector is clearly an area of research that could be very productive. The use of insecticide seed treatments in sugar beet has provided sustainable control of the virus yellows complex of viruses and it would be a good model for designing chemical control strategies against TuYV in oilseed rape.

A useful tool in the investigation of TuYV-brassica interactions would be the model plant *Arabidopsis thaliana*. This weed species has had its entire genome sequenced and numerous mutants (e.g. alterations within specific plant defence pathways) have been well characterised. This model system has the potential to successfully aid the genetic dissection of the TuYV-oilseed rape interactions as it has been used previously for the TuMV-brassica interactions (Rusholme *et al.*, 2007).

In order to build upon existing knowledge and research findings the following points are highlighted as potential areas of research that would be required to ensure that appropriate steps can be taken to manage TuYV .

- Survey the distribution and abundance of TuYV in UK oilseed rape crops.
- Determine whether strains of TuYV vary across UK and if there are more aggressive strains.

- Determine the impact of TuYV on yield parameters of current varieties.
- Determine whether any current varieties are resistant to TuYV.
- Monitor the autumn migration of aphids (principally *M. persicae*) and determine percentage carrying TuYV.
- Exploit seed treatments for TuYV control.
- Develop aphid thresholds for the application of autumn aphicides.
- Compare current aphicides and adjuvants for control of TuYV in oilseed rape (and other brassica crops).
- Identify resistance genes from brassica germplasm collections. Use existing molecular markers to assess current breeding material.
- Exploit *Arabidopsis* to identify ‘conventional’ resistance to TuYV.
- Pathogen derived resistance to TuYV (in *Arabidopsis*).
- Assess the impact of TuYV on the physiology, growth, resource capture of oilseed rape.
- Assess the impact of TuYV on other brassica crops such as cabbage, broccoli, cauliflower and lettuce.

10. References

Adam, N. M. & Hopkinson, D. 2008. Crop establishment improvements and foliar pest control in winter oilseed rape using a new clothianidin based seed treatment. In *Proceedings Crop Protection in Northern Britain 2008*, pp. 151-156.

Beuve M, Stevens M, Liu H Y, Wintermantel W M, Hauser S, Lemaire O. 2008. Biological and molecular characterization of an American sugar beet-infecting Beet western yellows virus isolate. *Plant Disease*, **92**:51-60.

Coutts B A, Hawkes J R, Jones R A C. 2006. Occurrence of Beet western yellows virus and its aphid vectors in over-summering broad-leafed weeds and volunteer crop plants in the grainbelt region of south-western Australia. *Australian Journal of Agricultural Research*, **57**:975-982.

D'Arcy, C. J., Torrance, L. & Martin, R. R. 1989. Discrimination among luteoviruses and their strains by monoclonal antibodies and identification of common epitopes. *Phytopathology* **79**, 869-873.

Dewar, A. M. & Cooke, D. 2006. Pests. In *The Sugar Beet Book* (ed. A. P. Draycott), pp. 316-358. Oxford: Blackwells Publishing.

Dreyer F, Graichen K, Jung C. 2001. A major quantitative trait locus for resistance to *Turnip yellows virus* (TuYV, syn. *Beet western yellows virus*, BWYV) in rapeseed. *Plant Breeding*, **120**:457-462.

Duffus J E. 1961. Economic significance of *Beet western yellows* (*Radish yellows*) on sugar beet. *Phytopathology*, **51**:605-607.

Duffus, J. E. & Russell, G. E. 1970. Serological and Host Range Evidence for the Occurrence of Beet Western Yellows Virus in Europe. *Phytopathology* **60**, 1199-1202.

Duffus J E, Russell G E. 1972. Serological relationship between Beet western yellows and Turnip yellows viruses. *Phytopathology*, **62**:1274-1277.

Duffus J E. 1977. Aphids, viruses, and the yellow plague. In *Aphids as virus vectors*, pp. 361-383.

Foster, S. P., Devine, G. & Devonshire, A. L. 2007. Insecticide resistance. In *Aphids as Crop Pests* (ed. H. F. van Emden & R. Harrington), pp. 261-286: CAB International UK.

Gilligan C A, Pechan P M, Day R, Hill S A. 1980. *Beet western yellows virus* on oilseed rape (*Brassica-napus* L). *Plant Pathology*, **29**:53-53.

Graichen K, Rabenstein F. 1996. European isolates of *Beet western yellows virus* (BWYV) from oilseed rape (*Brassica napus* L ssp *napus*) are non-pathogenic on sugar beet (*Beta vulgaris* L var *altissima*) but represent isolates of *Turnip yellows* *virus* (TuYV). *Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz-Journal of Plant Diseases and Protection*, **103**:233-245.

Graichen K, Peterka H. 1999. Generation of oilseed rape with resistance to turnip yellows luteovirus. In *10th International Rapeseed Congress - New Horizons from an Old Crop*, pp. 148.

Graichen K, Schliephake E. 1999. Infestation of winter oilseed rape by *Turnip yellows luteovirus* and its effect on yield in Germany. In *10th International Rapeseed Congress - New Horizons from an Old Crop*, pp. 131.

Graichen K, Rabenstein F, Kurtze E. **2000.** The occurrence of *Turnip yellows virus* in winter oilseed rape in Austria. *Pflanzenschutzberichte*, **59**:35-46.

Hampton R O, Keller K E, Baggett J R. **1998.** *Beet western yellows luteovirus* in western Oregon: Pathosystem relationships in a vegetable-sugar beet seed production region. *Plant Disease*, **82**:140-148.

Hardwick, N. V., Davies, J. M. L. & Wright, D. M. 1994. The incidence of three diseases of winter oilseed rape in England and Wales in the 1991/92 and 1992/93 growing season. *Plant Pathology* **43**, 1045-1049.

Hauser S, Stevens M, Mougel C, Smith H G, Fritsch C, Herrbach E, Lemaire O. **2000.** Biological, serological, and molecular variability suggest three distinct polerovirus species infecting beet or rape. *Phytopathology*, **90**:460-466.

Hauser S, Weber C, Vetter G, Stevens M, Beuve M, Lemaire O. **2000.** Improved detection and differentiation of poleroviruses infecting beet or rape by multiplex RT-PCR. *Journal of Virological Methods*, **89**:11-21.

Hauser, S., Stevens, M., Beuve, M. & Lemaire, O. 2002. Biological properties and molecular characterization of beet chlorosis virus (BChV). *Archives of Virology* **147**, 745-762.

Herrbach, E. 1994. Serological detection of beet western yellows luteovirus in the non-vector *Brevicoryne brassicae (Homoptera, Aphididae). Journal of Phytopathology-Phytopathologische Zeitschrift* **142**, 43-50.

Hill S A, Lane A, Hardwick N V. **1989.** The incidence and importance of *Beet western yellows virus* in oilseed rape. *Aspects of Applied Biology*, **23**:311-318.

Hunter P J, Jones J E, Walsh J A. **2002.** Involvement of *Beet western yellows virus*, *Cauliflower mosaic virus*, and *Turnip mosaic virus* in internal disorders of stored white cabbage. *Phytopathology*, **92**:816-826.

Jasnic S, Bagi F. **2007.** Viral diseases of oilseed rape. *Biljni Lekar (Plant Doctor)*, **35**:458-461.

Jay C N, Rossall S, Smith H G. 1999. Effects of *Beet western yellows virus* on growth and yield of oilseed rape (*Brassica napus*). *Journal of Agricultural Science*, **133**:131-139.

Jones, T. D., Buck, K. W. & Plumb, R. T. 1991. The detection of beet western yellows virus and beet mild yellowing virus in crop plants using the polymerase chain reaction. *Journal of Virological Methods* **35**.

Jones R A C, Coutts B A, Hawkes J. 2007. Yield-limiting potential of *Beet western yellows virus* in *Brassica napus*. *Australian Journal of Agricultural Research*, **58**:788-801.

Kazda J, Baranyk P, Nerad D. 2005. The implication of seed treatment of winter oilseed rape. *Plant, Soil and Environment*, **51**:403-409.

Kerlan C. 1991. Les viroses, une étude de longue haleine. *Le dossier, Oleoscop n°5, septembre*, **91**:6-7.

Latham L J, Smith L J, Jones R A C. 2003. Incidence of three viruses in vegetable brassica plantings and associated wild radish weeds in south-west Australia. *Australasian Plant Pathology*, **32**:387-391.

Lemaire, O., Herrbach, E., Stevens, M., Bouchery, Y. & Smith, H. G. 1995. Detection of sugar-beet-infecting beet mild yellowing virus isolates with a specific RMA probe. *Phytopathology* **85**, 1513-1518.

Mayo, M. A. 2002. ICTV at the Paris ICV: results of the plenary session and the binomial ballot. *Archives of Virology* **147**, 2254-2260.

Moreno A, de Blas C, Biurrun R, Nebreda M, Palacios I, Duque M, Fereres A. 2004. The incidence and distribution of viruses infecting lettuce, cultivated Brassica and associated natural vegetation in Spain. *Annals of Applied Biology*, **144**:339-346.

Njuguna J G, Govier D A, Cockbain A J. 1986. Viruses of oilseed rape. *Report of Rothamsted Experimental for 1985*:123.

Nagarajan K, Govier D A, Cockbain A J. 1987. Diseases of break crops. Oilseed rape. Viruses. *Report of Rothamsted Experimental for 1986*:115.

Pallett D W, Thurston M I, Cortina-Borja M, Edwards M L, Alexander M, Mitchell E, Raybould A F, Cooper J I. 2002. The incidence of viruses in wild *Brassica rapa* ssp *sylvestris* in southern England. *Annals of Applied Biology*, **141**:163-170.

Polak J and Majkowa L. 1992. Winter oilseed rape as a likely source and reservoir of *Beet western virus*. *Ochrana Rostlin*, **28**:191-196.

Read M A, Hewson R T. 1988. Prevention of *Beet western yellows virus* (BWYV) in winter oilseed rape by control of aphid vectors with deltamethrin. In *Brighton Crop Protection Conference - Pest and Diseases*, pp. 989-996.

Rusholme R L, Higgins E E, Walsh J A, Lydiate D J 2007. Genetic control of broad-spectrum resistance to turnip mosaic virus in Brassica rapa (Chinese cabbage). Journal of General Virology **88**, 3177-3186.

Russell G E. **1958.** Sugar beet yellows: a preliminary study of the distribution and interrelationships of viruses and virus strains found in East Anglia, 1955-1957. *Annals of Applied Biology*, **46**:393-398.

Russell G E, Duffus J E. **1970.** An aphid-transmitted yellowing virus disease of lettuce in England. *Plant Pathology*, **19**:148-149.

Schliephake E, Graichen K, Rabenstein F. **2000.** Investigations on the vector transmission of the *Beet mild yellowing virus* (BMYV) and the *Turnip yellows virus* (TuYV). *Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz-Journal of Plant Diseases and Protection*, **107**:81-87.

Schroder M. **1994.** Investigations on the susceptibility of oilseed rape (*Brassica-napus* L, ssp *napus*) to different virus diseases. *Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz-Journal of Plant Diseases and Protection*, **101**:576-589.

Schubert J, Rabenstein F, Graichen K, Richter K. **1998.** Comparison of the 5'-end nucleotide sequences of luteoviruses from oilseed rape and sugar beet. *Archives of Phytopathology and Plant Protection*, **31**:519-530.

Shahraeen N, Farzadfar S, Lesemann D E. **2003.** Incidence of viruses infecting winter oilseed rape (*Brassica napus* ssp *oleifera*) in Iran. *Journal of Phytopathology*, **151**:614-616.

Smith H G, Hinckes J A. **1985.** Studies on *Beet western yellows virus* in oilseed rape (*Brassica-napus* ssp *oleifera*) and sugar beet (*Beta vulgaris*). *Annals of Applied Biology*, **107**:473-484.

Smith, H. G., Barker, G. L., Stevens, M. & Hallsworth, P. B. 1996. Production of monoclonal antibodies for the detection of beet mild yellowing virus and related strains. *European Journal of Plant Pathology* **102**, 163-169.

Stevens M, Smith H G, Hallsworth P B. **1994.** The host-range of Beet yellowing viruses among common arable weed species. *Plant Pathology*, **43**:579-588.

Stevens M, Smith H G, Hallsworth P B. **1995.** Detection of the luteoviruses, *Beet mild yellowing virus* and *Beet western yellows virus*, in aphids caught in sugar-beet and oilseed rape crops, 1990-1993. *Annals of Applied Biology*, **127**:309-320.

Stevens M, Freeman B, Liu H Y, Herrbach E, Lemaire O. **2005.** Beet poleroviruses: close friends or distant relatives? *Molecular Plant Pathology*, **6**:1-9.

Stevens M, Liu H-Y, Lemaire O. 2006. Virus Diseases. In *Sugar Beet*, pp. 256-285.

Stevens, M., M., A. N. & Harrington, R. 2008. *Turnip yellows virus* (TuYV) in winter oilseed rape: the importance of autumn migrations of the peach-potato aphid, *Myzus persicae*, and the role of insecticide seed treatments in reducing impact of the disease. In *Proceedings Crop Protection in Northern Britain 2008*, pp. 175-180.

Thurston M I, Pallett D W, Cortina-Borja M, Edwards M L, Raybould A F, Cooper J I. **2001.** The incidence of viruses in wild *Brassica nigra* in Dorset (UK). *Annals of Applied Biology*, **139**:277-284.

Timmerman E L, Darcy C J, Splittstoesser W E. **1985.** *Beet western yellows virus* in Illinois vegetable crops and weeds. *Plant Disease*, **69**:933-936.

Tomlinson J A. **1972.** Beet western yellows disease of lettuce. *Grower*, **78**:347.

Tomlinson J A, Walker V M. **1973.** *Beet western yellows viru*s in lettuce. *Report of the National Vegetable Research Station for 1972.*:pp. 97-99.

Tomlinson J A, Faithfull E M, Ward C M. **1976.** Chemical suppression of symptoms of two virus diseases. *Annals of Applied Biology*, **84**:31-41.

Walkey D G A, Payne C J. **1990.** The reaction of two lettuce cultivars to mixed infection by *Beet western yellows virus, Lettuce mosaic virus* and *Cucumber mosaic virus*. *Plant Pathology*, **39**:156-160.

Walkey D G A, Pink D A C. **1990.** Studies on resistance to *Beet western yellows virus* in lettuce (*Lactuca sativa*) and the occurrence of field sources of the virus. *Plant Pathology*, **39**:141-155.

Walsh J A, Tomlinson J A. **1985.** Viruses infecting winter oilseed rape (*Brassica-napus* ssp *oleifera*). *Annals of Applied Biology*, **107**:485-495.

Walsh J A, Perrin R M, Miller A, Laycock D S. **1989.** Studies of *Beet western yellows virus* in winter oilseed rape (*Brassica-napus* ssp *oleifera*) and the effect of insecticidal treatment on its spread. *Crop Protection*, **8**:137-143.

Wang M-B, Abbott D. and Waterhouse, P M. 2000. A single copy of a virus derived transgene encoding hairpin RNA gives immunity to barley yellow dwarf virus. Molecular Plant Pathology **1**, 401-10.

Watts L E. **1975.** Response of various breeding lines of lettuce to *Beet western yellows virus*. *Annals of Applied Biology*, **81**:393-397.