SESVANDERHAVE

TECHNICAL SHEET

Yellowing

By Niels Wynant, Project Manager Biotische Stress at SESVanderHave

WHAT IS THE GENETIC CONTRIBUTION?

In May 2018, the European Commission decided on a ban on the outdoor use of neonicotinoids (NNI), effective since 19 December 2018. How can the genetic contribution from SESVanderHave help breeders limit the impact of this disease?

WHAT WAS THE ROLE OF THE NNI?

Niels WYNANT: More than 90% of European surfaces were coated with a seed coating containing neonicotinoids, allowing for almost total elimination of viral yellows, beet flies, flea beetles, and damage from many below-ground pests (spotted snake millipede, crane fly, wireworm, pygmy mangold beetle). Despite this large-scale use, the vector aphids did not disappear and yellowing continues to pose a threat.

WHAT ARE THE CONSEQUENCES OF THIS RETREAT?

Niels WYNANT: To date, TEPPEKI® (flonicamid) is an approved insecticide against aphids. A specific aphicide, it is efficacious on all types of aphids, and safe to all beneficial insects. It can be applied from the 6-leaf stage and has a persistence of up to 3 weeks. The return to repeated pyrethroid-based foliar treatments is also possible, but these treatments are more difficult to position in time and therefore less certain in terms of effectiveness. Moreover, there are also aphids that are resistant to these molecules and foliar treatments are harmful to the auxiliary fauna. As a result, there will be yield losses depending on the frequency, occurrence (in the field) and severity (on the plant) of the disease.

IS THERE A GENETIC SOLUTION?

Niels WYNANT: In addition to the agronomic solutions being implemented by the sector, plant breeders will have to find tolerances to the various viruses responsible for yellowing and/or to the aphids that are vectors of the disease. Such tolerances are not yet available at present. The diversity of viruses existing in Europe further complicates the task of plant breeders, because tolerance to all of these viruses is needed. Also the level of tolerance needed to deal with the disease will have to be evaluated. In any case, this represents a big challenge for breeders, and breeding efforts have been stepped up in recent years.

DATA SHEET OF YELLOWING

The symptoms

They appear in circles in the fields, sometimes starting in June, in the form of lightening and then yellowing of the lamina between the veins of leaves. The leaves thicken and become brittle.

SEVERAL VIRUSES INVOLVED

It should be noted that there are various types of yellowing:

First of all there is severe Yellowing caused by the BYV (Beet Yellows Virus) belonging to the genus Closterovirus, characterised by a lemon-yellow colouration, which can subsequently cause small reddish necroses.

Then there are three viral species belonging to the genus Polerovirus which are responsible for mild yellow symptoms: the Beet Mild Yellowing Virus (BMYV), the Beet Western Yellows Virus (BWYV) and the Beet Chlorosis Virus (BChV). Then there is a milder form (BMYV= Beet Mild Yellowing Virus), characterised by a more orange colouration, often followed by cryptogamic infestation (e.g. Alternaria) and premature leaf necrosis.

THE VECTOR: MYZUS PERSICAE OR GREEN PEACH APHID

The main vector aphids of yellowing are the green peach aphid (Myzus persicae) and the black bean aphid (Aphis fabae), but there are several other aphids of lesser importance that are also considered vectors (e.g. Myzus ascalonicus). Potential virus reservoirs include weeds (goosefoot, chickweed, veronica, etc.), spinach or ensiled or unharvested beet. The severity of attacks depends on the populations of vector aphids and the presence of virus reservoirs close to beet fields. The risks of early, widespread attacks are higher when climate conditions are favourable to aphid outbreaks (mild autumn and winter) permitting a large initial population in spring. A dry, warm spring then promotes rapid development of the colonies.

Virus acquisition and transmission by the Myzus Persicae aphid

The mechanisms of virus acquisition and transmission by vector insects are extremely important in the transmission of virus diseases. However, they differ according to the type of virus.

1. VIRAL ACQUISITION

Since the virus is not transmitted to the aphid's offspring, the latter must necessarily go through a viral acquisition phase.

For the Poleoviruses, with persistent transmission mode, by extracting sap from an infected plant, the aphid ingests viral particles that will circulate in the digestive tract. Some particles degrade during digestion or are excreted with the honeydew, others pass through the gut wall and accumulate in the aphid's body.

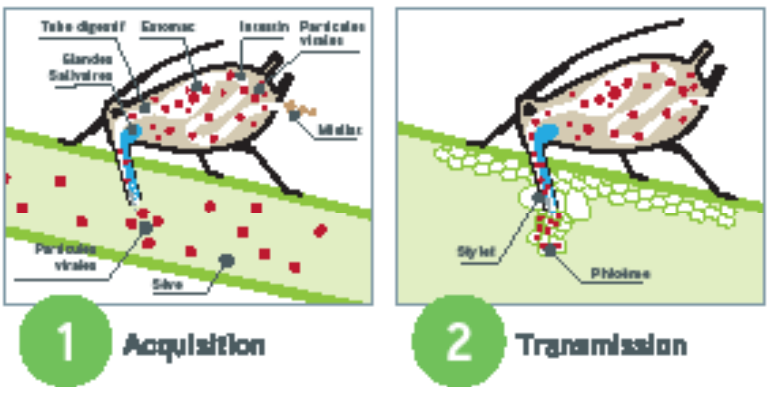
The aphid then becomes viruliferous until it dies.

For the Closterioviruses, with semi-persistent transmission mode, the viral acquisition phase lasts about 24 hours. The virus remains viable for about 48 hours on the insect's mouthparts. It may be retransmitted upon acquisition but is lost when the aphid moults.

2. TRANSMISSION TO HEALTHY PLANTS

When an aphid is vector of viral particles, it can transmit the virus to the plants on which it feeds. Its stylet allows the aphid to access the various tissues of the plant. The virus may pass from the saliva to the sap, i.e. from its vector to the plant (this is the host inoculation phase). It should be noted that poleroviruses infect only the phloem vessels within the leaves, whereas closteroviruses may infect the entire leaf.

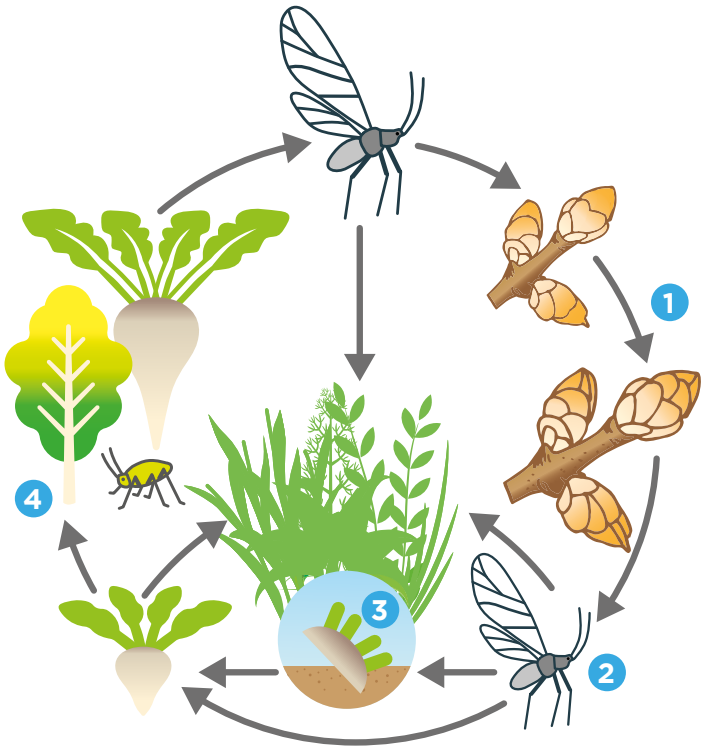
Once the plant has been inoculated, it is too late and the infection spreads to the entire plant.



Life cycle of Myzus Persicae

1. Eggs are laid in late autumn on buds of peach trees and various Prunus, the primary host plant.

2. In April, these eggs hatch into the fundatrices, each producing about forty larvae. These then migrate to their secondary host: crucifers and various other plants including economically important crops such as spinach, cane sugar, tomatoes, potatoes, maize, cereals and sugar beets.



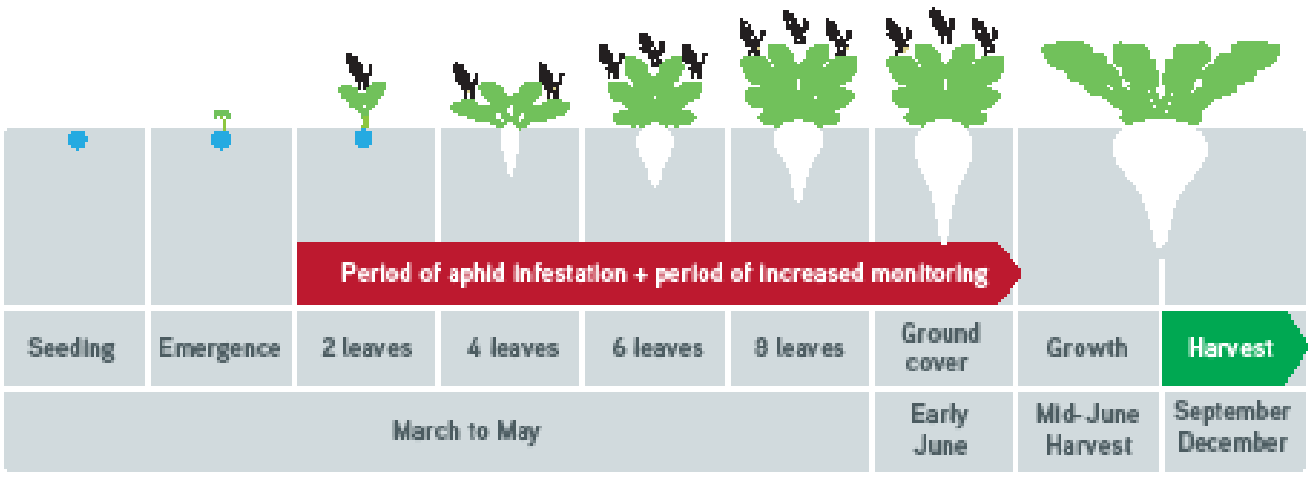
RISK PERIOD

The risk period depends on the duration of flight, crossed with the level of susceptibility of the beet that can be affected by aphids. It starts as soon as the first aphids appear in the plot, i.e. from the 2-leaf stage until ground cover.

3. In temperate climates like France, the adults and the larvae can survive in winter on their secondary hosts, in fodder beet and sugar beet silos, on garden beets stored along with their regrowth, or on beet regrowth abandoned at the time of harvesting.

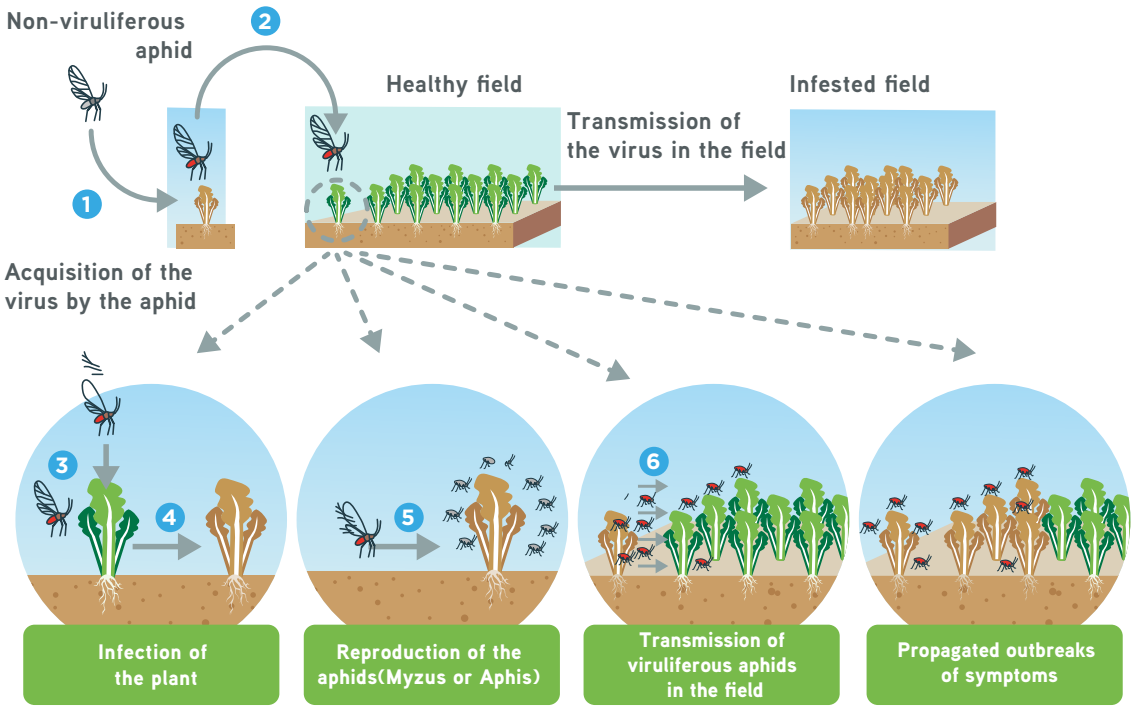
4. There are three to four generations, either winged or wingless, each year. From mid-May they can be found on the beets.

5. Winged adults, both male and female, reappear from early September. The winged males can then be observed on the beets.



Steps of infection in the field

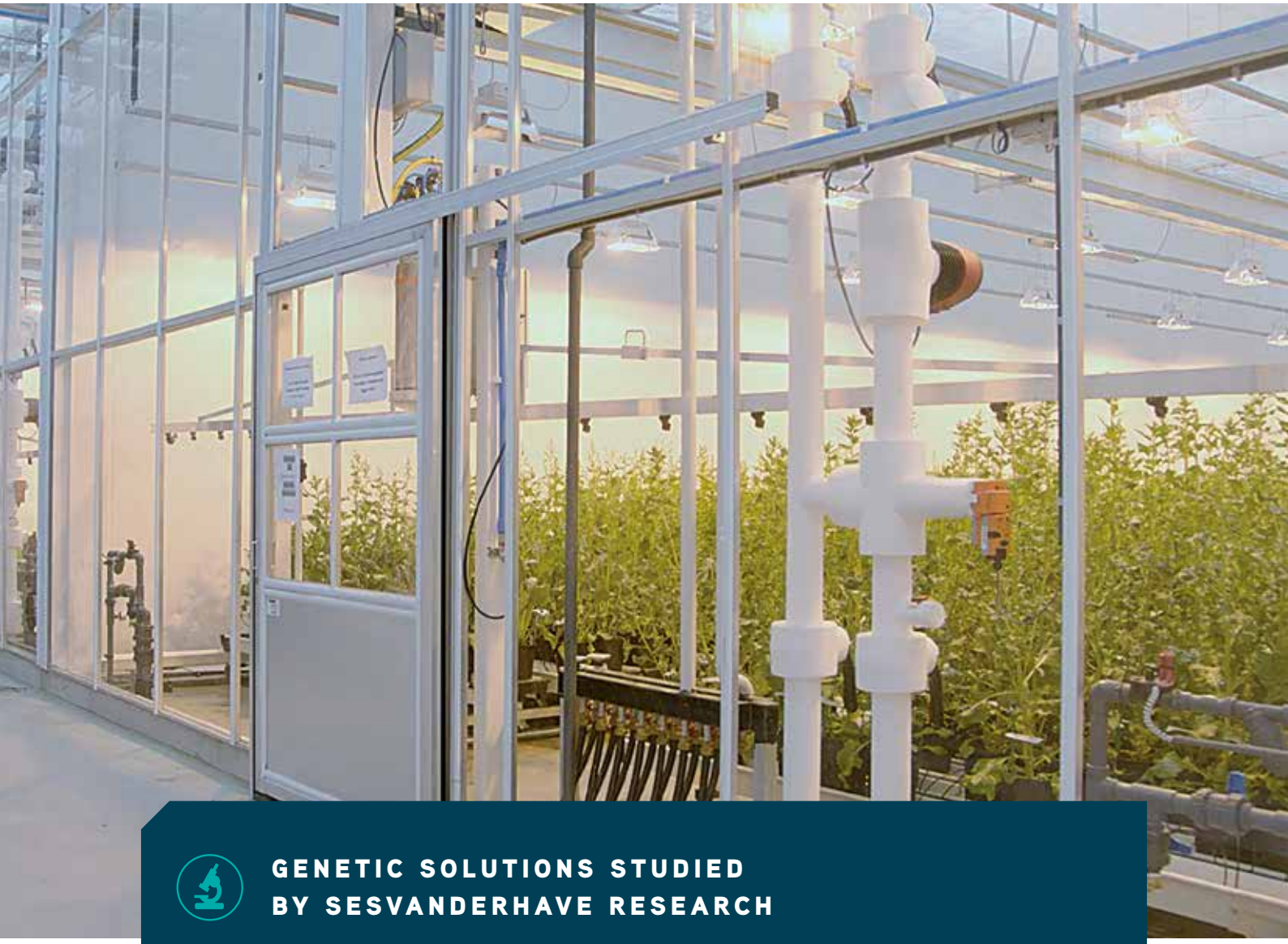
1. Flight of Myzus Persicae aphids on beet field from the 2-leaf stage.
2. Primary infection: arrival of winged aphids, some of which are viruliferous (in red) and will contaminate the plants.
3. Secondary dissemination: by wingless aphids (Aphis fabae) in the form of patches around the contaminated areas. Young wingless adults, of which there are too many, will colonise neighbouring plants thereby transmitting the virus if they are viruliferous.



The different viruses are studied in the laboratory.

Genetic analyses are performed.

Multiple greenhouse trials are conducted.



GENETIC SOLUTIONS STUDIED BY SESVANDERHAVE RESEARCH

Resistance to aphids

Prevention of aphid colonisation or multiplication in the plant (architecture of the plant, composition of the leaves, etc.)

Resistance to viruses

Disturbed dynamics of the viral cycle

Tolerance to viruse

Virus multiplication in the plant without alteration of the plant's physiology and therefore no yield loss

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More info?

Check our social media channels and website www.sesvanderhave.com