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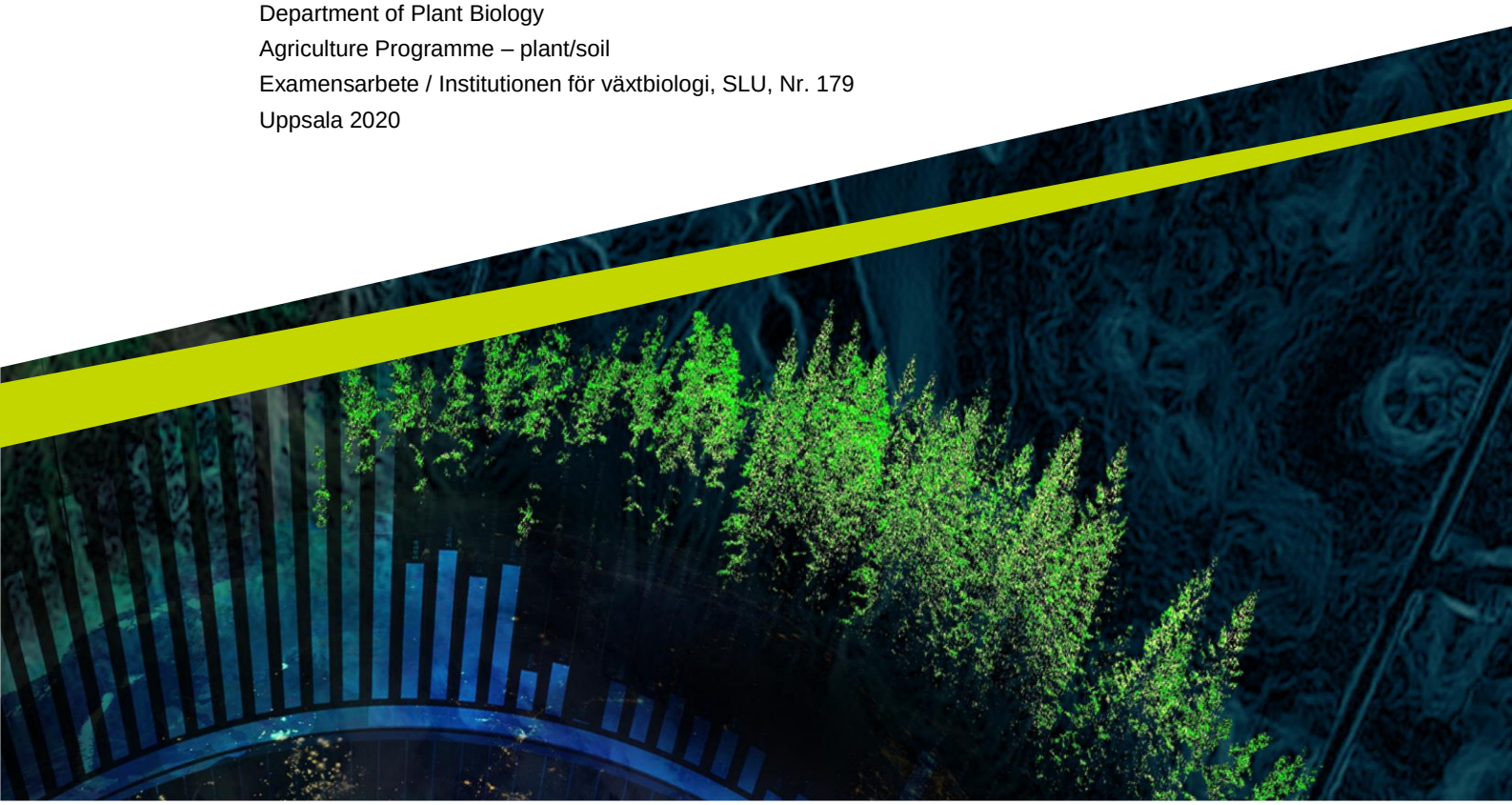

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Sammanfattning

Denna uppsats beskriver virusgulsot i sockerbetor och omfattar både äldre och nyare kunskap om virus, vektorer och värdväxter, samt kontrollmetoderna mot sjukdomen. Syftet är att utforska nya potentiella kontrollmetoder och hur sjukdomen kan komma att påverka jordbruket i framtiden.

Sockerbetor kan bli drabbade av ett flertal sjukdomar och virusgulsoten är en av de viktigaste. Virusgulsot hos sockerbetor orsakas av beet yellows virus (BYV), beet mild yellowing virus (BMYV), beet chlorosis virus (BChV) och beet western yellows virus (BWYV). Infektioner orsakar visuella symptom på bladen som kloros och nekros och även en minskad tillväxt hos grödan. Infektioner leder till att avkastningen på socker minskar i samband med att biomassan och sackaroskoncentrationen reduceras samt att blåtalet blir förhöjt. De virus som orsakar virusgulsot hos sockerbetor har olika stora värdkretsar men infekterar främst växter från familjen Amaranthaceae. Mottagliga ogräs och grödor kan vara infektionskällor och bör tas i beaktning om sjukdomen förekommer i sockerbetsfält.

Persikbladlusen (*Myzus persicae*) och betbladlusen (*Aphis fabae*) är de mest effektiva vektorerna för sjukdomen. Virusgulsot hos sockerbetor sprids genom att virussmittade bladlöss suger på bladen. Storleken på bladlössens populationer påverkas av förekomsten av deras primära värdväxter, naturliga fiender och vädret. Varmt och torrt väder resulterar oftast i fler migrerande bladlöss som kan fångas i luftströmmar och driva längre sträckor. Hög förekomst av virusgulsot hos sockerbetor i Sverige har främst orsakats av att virussmittade bladlöss migrerat från söder. Temperaturen påverkar även mängden övervintrande bladlöss i stukor eller på värdväxter. Tidigare har de låga temperaturerna i Sverige motverkat övervintring av aktiva bladlöss, men det är en stor risk för att övervintring blir mer vanligt förekommande i framtiden som resultat av klimatförändringarna.

Sedan tidigt 1900-tal har virusgulsot hos sockerbetor orsakat bekymmer för både odlare och sockerproducenter, vilket har lett till internationella sammarbeten och omfattande forskning inom området. Under det senaste århundradet har förekomsten av virusgulsoten hos sockerbetor varierat mellan låga och höga nivåer. De senaste årtiondena har förekomsten av virusgulsoten varit låg på grund av utvecklingen av effektiva insekticider och prognosmetoder. Kontrollmetoder mot virusgulsot består av att så tidigt, minska lagringen i stukor och övervintrande sockerbetor samt att använda insekticider. Nya kontrollmetoder har för närvarandet blivit aktuellt med tanke på att neonikotinoider blev förbjudna i EU 2018, vilket var den mest förekommande kontrollmetoden mot virusgulsot i sockerbetor. Bristen på effektiva kemiska bekämpningsmedel mot den högt insekticidresistenta *M. persicae* tyder på att virusgulsoten hos sockerbetor kan bli mer förekommande i framtiden. Utveckling eller tillåtelse av nya insekticider i sockerbetsodling, utveckling av resistenta sorter och användandet av biologisk kontroll är kontrollmetoder som borde bli utforskade i framtiden.

Nyckelord: virusgulsot, sockerbetor, BYV, BMYV, BWYV, BChV, Myzus persicae, persikbladlus, Aphis fabae, betbladlus, neonikotinoider

Abstract

This thesis describes virus yellows in sugar beets and converse both older and new knowledge about the viruses, vector and host relations and the control measures against the disease. The aim is to explore new potential control measures and to predict how the disease may affect agriculture in the future.

Sugar beets are affected by various diseases, with virus yellows being one of the most important ones. Virus yellows in sugar beets is caused by beet yellows virus (BYV), beet mild yellowing virus (BMYV), beet chlorosis virus (BChV) and beet western yellows virus (BWYV). Infections cause visual symptoms like leaf chlorosis, brittleness, necrotic spots and decreased growth. Infections result in sugar yield loss by the reduction in biomass and sucrose concentration as well as by increasing juice impurity levels. The host ranges of the viruses causing virus yellows are narrow to large and mainly include species in the family Amaranthaceae. Weeds and crops, which are susceptible to virus yellows can act as infection sources and should be taken into consideration if the disease is occurring in sugar beet crops.

The green peach aphid (*Myzus persicae*) and the black bean aphid (*Aphis fabae*) are the most important vectors for the disease. Virus yellows is spread by viruliferous aphids via feeding. Aphid populations are influenced by the occurrence of their primary host plants, natural enemies and the weather. Warm and dry weather usually results in more migrating aphids, which can get caught in the wind and drift over longer distances. When the incidence of virus yellows has been high in Sweden, the cause has been that viruliferous aphids have migrated from the south. The temperature does also influence the number of overwintering aphids in clamps or on host plants. In the past, the temperatures in Sweden have disfavoured overwintering of active aphids. However, there is a great risk that overwintering aphids become more common in the future due to climate change.

Since the early 1900's, virus yellows has caused concern to both farmers and sugar manufacturers, which has led to international corporations and extensive research on the issue. Throughout the 1900's, the incidence of virus yellows has fluctuated between low and high levels. During the last decades, the incidence of virus yellows has been low because of the development of effective insecticides and virus yellows forecasting. Control measures against virus yellows consist of early sowing, limiting clamps and overwintering sugar beets as well as of insecticide usage. New control measures are now of importance since the ban of neonicotinoids in the EU in 2018, which limits the most used control measure against vectors of virus yellows. The lack of effective chemical control measures against the highly insecticide-resistant *M. persicae* suggests that virus yellows might become more frequently occurring in sugar beets in the future. Development or introduction of new insecticides for sugar beet production, development of resistant cultivars and using biological control are control measures that should be explored in the future.

Keywords: virus yellows, BYV, BMYV, BChV, BWYV, sugar beet, Myzus persicae, green peach aphid, Aphis fabae, black bean aphid, neonicotinoids.

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Abbreviations

BChV	Beet chlorosis virus
BMYV	Beet mild yellowing virus
BWYV	Beet western yellows virus
BYV	Beet yellows virus
EU	European union

1. Introduction

Being one of the most important diseases in sugar beet, virus yellows has historically affected the agricultural industry and research significantly. Virus yellows had the largest impact between 1930-1995 and a lot of research about the disease has been carried out through these years (Dunning 1988; Qi *et al*. 2004). During the last decades, virus yellows have had a low incidence predominantly because of the development of effective insecticides, which eliminate vectors, and virus yellows forecasting (Qi *et al*. 2004; Hauer *et al.* 2017). Neonicotinoids, such as imidacloprid, clothianidin and thiamethoxam have been used as a seed coating in almost all conventionally grown sugar beet crops but they were banned in the European union (EU) in 2018 (Hauer *et al.* 2017; European Commission 2020). Neonicotinoid resistance has also started developing in the main vector, which is the green peach aphid (*Myzus persicae*) (Srigiriraju *et al.* 2010; Slater *et al.* 2012; Umina *et al.* 2014; Panini *et al.* 2014; Voudouris *et al.* 2017). New control measures are now of importance since the main control measure has been limited.

The low incidence of virus yellows during the last decades has resulted in a lack of recent research and review articles in the subject. Research on virus yellows seems to gaining more interest again because of the prevailing situation and overview articles that collect both recent and older information about virus yellows would prove useful. This thesis describes the sugar beet production today and revisits the history of virus yellows to understand the impact it has had on sugar beet production. The viruses, symptoms, vector, and host relations are also described and discussed for a better understanding of the spread of virus yellows. The already existing and potential control measures for virus yellows are also explored. The aim was to collect information on the factors that contribute to the spread of virus yellows and to get a general overview of the disease. As well as, to predict how virus yellows could affect the agriculture in the future.

2. Sugar beet

Sucrose is a common sugar that is consumed widely by humans. It is obtained from two crops, sugar cane and sugar beet, with sugar beet providing 20% of the world demand for sugar (Draycott 2008; Finkenstadt 2013). The sugar beet stores sucrose and nutrients in the hypocotyl, which is the part between the root and stem and used for sugar production (Anderberg & Anderberg 2005). The sucrose concentration is between 13-22% in sugar beets, depending on cultivar (Cattanach *et al.* 1991).

Beets were first grown as a garden vegetable, about 2000 years ago (Draycott 2008). It was later used for fodder and from the 1700's and onwards, mainly cultivated for sucrose (Biancardi *et al.* 2012). The origin of sugar beet is the wild beet, *Beta vulgaris* ssp. *maritima,* which grows around shores. Different wild beets in the Mediterranean were probably first to be selected and later used for breeding (Draycott 2008). From the Middle Ages in Europe until today, sugar has been widely used for sweetening purposes (Draycott 2008). Sugar beet is primarily grown for human consumption, but also for bioenergy, bioplastics and animal feed (Cattanach *et al.* 1991; Finkenstadt 2013). The residues from the industrial sucrose production, such as the sugar beet pulp and molasses, can be used as a feed supplement for livestock (Cattanach *et al.* 1991). The molasses are also used for alcohol production, pharmaceuticals and for yeast in the baking industry (Cattanach *et al.* 1991).

Apart from sugar beet (*B. vulgaris* ssp. *vulgaris* var. *altissima*), there are three more groups of beets which are cultivated: the mangelwurzel, or field-beet/fodder beet (*B. vulgaris* var. *macrorhiza*), which has a lower concentration of sucrose and is used for livestock (Anderberg & Anderberg 2005), as well as chard (*B. vulgaris* ssp. *vulgaris*, Cicla-Group) and beetroot (*B. vulgaris* ssp. *vulgaris* Conditiva-Group), which are used for culinary purposes (Anderberg & Anderberg 2005).

Sugar beet is grown in about 50 countries in Mediterranean, temperate and continental climates (Qi *et al.* 2004; Draycott 2008). The production is mainly located between 30 º and 60 º N, with Europe accounting for more than half of the sugar beet production (Draycott 2008). Beets can be grown on soils with relatively high amounts of sodium because it is originally a shore species.

In 2019, sugar beets were grown over an area of 27 300 hectares in Sweden, by 1300 companies (Jordbruksverket 2019). The total arable land usage in Sweden was 2 551 500 hectares, which means that sugar beets account for 1.07 % of the arable

land usage (Jordbruksverket 2019). Sugar beet farming has been reduced by 28 200 hectares since the year 2000 (Jordbruksverket 2019). In 2015, there was an excess amount of sugar beets in the EU, which lead to an agreement between farmers and sugar producers to regulate the production, resulting in a decrease in sugar beet production in 2015/2016 (Jordbruksverket 2019). The production of sugar beets in Sweden is mainly conventional, only 150 hectares are farmed organically (Ekoweb 2019). This is primarily because of the lack of organic sugar beet processing facilities in Sweden. All the organic sugar beets are today grown under contract to Nordic Sugar and shipped to Denmark for processing (Ekoweb 2019).

To be able to store a great amount of sugar, the sugar beet needs resources to be able to produce a large number of roots and leaves for photosynthesis. This is achieved by cultural practises like application of fertilizers and liming as well as the use of weed and pest control. Several pests can reduce yield in sugar beets. Pests are different insects, nematodes, fungi and viruses (Viketoft *et al.* 2019). Pests can decrease the growth by damaging or deforming the root and reducing healthy foliage and sucrose concentration (Viketoft *et al.* 2019). One of the most important ones is the disease virus yellows, which can severely reduce the number of healthy leaves, increase juice impurities, reduce photosynthesis, growth, and storage of sugar (Clover *et al.* 1990; Stevens *et al.* 2004). Another disease is rhizomania, which is caused by beet necrotic yellow vein virus (BNYVV), and can severely reduce root mass and sucrose content (Viketoft *et al.* 2019). Aphids, thrips, beet fly (*Pegomya betae*), pygmy mangold beetle (*Atomaria linearis*), centipedes and the silver Y (*Autographa gamma*) are some of the most important pests that cause feeding damage on sugar beets (Viketoft *et al.* 2019). Nematodes like the beet cyst eelworm (*Heterodera schachtii*) and the yellow beet cyst nematode (*H. betae*) can also deform and damage the sugar beet root (Viketoft *et al.* 2019).

3. Virus yellows

Virus yellows is induced by a complex of viruses and the disease can be caused by one or more viruses at a time (Nilsson & Larsson 1990). Beet yellows virus (BYV) was the first virus discovered causing virus yellows (Nilsson & Larsson 1990). Later a virus inducing milder symptoms was found and established in 1958 as beet mild yellowing virus (BMYV) (Russell 1958). Throughout the years, more mild variants of BYV have been found (Nilsson & Larsson 1990), and it is not always the case that BYV causes more severe symptoms than BMYV. There is also an American species called beet western yellows virus (BWYV) that causes virus yellows much alike BMYV. The latest one to be discovered, beet chlorosis virus (BChV), does also cause virus yellows (Stevens *et al.* 2005a).

3.1. BYV

Beet yellows virus is a virus from the family *Closteroviridae* and a member of the genus *Closterovirus* (Agranovsky & Lesemann 2011). It was first described in 1936. The family *Closteroviridae* contains three genera of plant viruses with 26 species causing infections in a variety of crops such as cucumber, grapevine, beet, and cereals (Agranovsky & Lesemann 2011). Important viruses of this family are *Grapevine leafroll-associated virus 3*, *Lettuce infectious lettuce virus* and *Citrus tristeza virus* (Agranovsky & Lesemann 2011).

The virus particles of members of the family *Closteroviridae* are characterized by their helical, filamentous and flexuous structure (Agranovsky & Lesemann 2011). The length of the virus particle is between 1250-2200 nm and it has a diameter of 12 nm (Agranovsky & Lesemann 2011). The genome consists of a single molecule of positive-sense single-stranded RNA, ssRNA (+), encapsidated with two coat proteins, one major and one minor coat protein (Agranovsky & Lesemann 2011; Biswas *et al.* 2017). The 22-25 kDa major coat protein forms the main part of the virion whereas the minor coat protein encapsidates the 5´-terminal part of the genome, forming a 75-100 nm segment at this end which results in a characteristic rattlesnake structure (Agranovsky & Lesemann 2011; Biswas *et al.* 2017).

3.2. BMYV, BChV and BWYV

Beet mild yellowing virus, *Beet chlorosis virus* and *Beet western yellows virus* are viruses of the genus *Polerovirus* in the family *Luteoviridae* (Stevens *et al.* 2005a). The poleroviruses have icosahedral virus particles with a diameter of 24-26 nm (van den Heuvel *et al.* 2011). The genome consists of a single molecule of ssRNA (+) (Stevens *et al.* 2005a). Other plant viruses in the genus are *Potato leafroll virus*, *Cereal yellow dwarf virus*-RPV and *Cucurbit aphid-borne yellows virus* (van den Heuvel *et al.* 2011).

The classification of the poleroviruses have been changed throughout the years. When BMYV was described by Russel (1958) in the UK, an American version of the virus was noted not long after and was initially called Radish yellows and then re-named BWYV (Stevens *et al.* 2005a). This American species of BWYV has a wide host range in weeds, many species in the family of Amaranthaceae, which includes sugar beet and spinach, but also crops in other families, like lettuce and broccoli (Stevens *et al.* 2005a). BMYV has a narrower host range, mainly plants from Amaranthaceae, like beets and spinach, but also weeds from different families, like *Capsella bursa-pastoris* and *Senecio vulgaris* (Stevens *et al.* 2005a). However, another virus yellows variant was found later in Europe that seemed to be alike BWYV concerning the wide host range, but that did not infect beets (Stevens *et al.* 2005a). The name BMYV has since then been used to describe European isolates that are able to infect beets and the European BWYV variant that did not infect beets, but most importantly lettuce and Brassica crops, is now called turnip yellows virus, TuYV (Stevens *et al.* 2005a). There might be more strains or species of the genus *Polerovirus* established in the future. Hauser *et al.* (2002) suggested that the name *Brassica yellowing virus* should be used for BWYV isolates that did not infect beets but a large group of Brassica species. When comparing the genome, Hauser *et al.* (2002) argued that TuYV should be considered a distinct serotype of *Brassica yellowing virus*. Recent studies by Newbert (2016) have found TuYV present sugar beets, but it is not yet proven if TuYV contribute to the symptoms.

BChV was identified in 1989 and through various studies, including antibody testing, analysing symptoms, identifying host range etc, it was established as a new species of the genus *Polerovirus* in 2002 (Stevens *et al.* 2005a).

3.3. Symptoms and impact of virus yellows

Infections with BYV, BMYV, BWYV and BChV induce symptoms like chlorosis in the leaves and growth reduction, which impact the yield of the sugar beet as well as changed nutritional aspects like sugar and sodium concentration. Yield loss caused by virus yellows has been reported to be up to 30% in the UK (Qi *et al.* 2004). The decrease in sugar yield has been calculated from field trials and can be up to 29% in trials with BMYV, up to 36% with BWYV, up to 24% for BChV and 47% with BYV (Lewellen & Skoyen 1984; Smith & Hallsworth 1990; Stevens *et al.* 2004).

BMYV, BChV and BWYV induce yellowing spots in the elder leaves and these spots develop 4-6 weeks after inoculation (Olsson Nyström 2019). With time, the yellowing spreads throughout the leaves, which get thickened and crispy (Fig. 1) (Olsson Nyström 2019). BChV induces milder yellowing than BMYV and BYV and these symptoms are distinguished by interveinal chlorosis as the tissue around the midrib with lateral veins remain green in most cases (Stevens *et al.* 2004). It is possible that the beet becomes infected with more than one of the viruses at the same time, which makes it harder to visually identify the virus through symptoms (Nyström & Hansen 2019). BMYV-affected leaves are also more susceptible to infection with fungi, for example *Alternaria* ssp. (Nilsson & Larsson 1990).

Figure 1. BMYV-infected sugar beet leaves (pictures from MaribоHilleshög).

Infections early in the season causes severe yield losses while infection after the end of June does not usually have any great impact (Smith & Hallsworth 1990). A field study by Stevens *et al.* (2004) found that most damaging to sugar yield, 19-27% decrease, was if plants became infected with BMYV at the 4-6 leaf stage. However, BChV had a greater impact on older plants compared to BMYV and a more variable effect on yield, between 8-24%, depending on year, strain and inoculation time (Stevens *et al.* 2004).

The outcome of the sucrose extraction process depends on the sucrose content and the juice quality (Syngenta 2016). The juice quality is determined by how low the concentrations are of amino-nitrogen, potassium and sodium (Syngenta 2016).

Infections with BMYV and BChV will increase the sodium content in the root and this is not dependent on inoculation date (Stevens *et al.* 2004). However, the potassium content is not clearly affected according to Stevens *et al.* (2004). The amino-nitrogen content can also be increased by infection with BMYV and BChV. However, the effects of BChV infection were more variable depending on the year in field trials, and the impact of BChV infection was only noticed one year when the inoculation date was early (Stevens *et al.* 2004).

BYV mainly targets the phloem, where virus particles accumulate in intracellular inclusions (Nilsson & Larsson 1990). Closteroviruses can be distinguished by their ability to induce clusters of vesicles containing double-stranded RNA (dsRNA) (Agranovsky & Lesemann 2011).

The different strains of BYV induce symptoms of varying degree (Nilsson & Larsson 1990). The leaves get yellow spots or yellowing at the edges and the symptoms generally develop 3-4 weeks after inoculation if infected early in the season (Nilsson & Larsson 1990). With time, the yellowing spreads throughout the leaves and lateral veins. Necrotic spots can appear and join together in older infected leaves (Fig. 2) (Nilsson & Larsson 1990). However, leaves that are fully developed before infection will remain green (Nilsson & Larsson 1990).

Figure 2. BYV-infected sugar beets (Picture from MariboHilleshög).

BYV can severely damage growth of the beet root, but usually the sugar concentration is not that effected (Olsson Nyström 2019). In field trials by Clover *et al.* (1999), BYV-infected sugar beets had a significantly increased concentration of potassium, amino nitrogen, and sodium. The net photosynthesis was also reduced, with the period right after infection having the highest decrease in net photosynthesis (Clover *et al.* 1999).

There is a difference in symptoms and incubation time if infection takes place later in the season, between August and September when the plant is more developed (Nilsson & Larsson 1990). The incubation time increases then to 8-9 weeks and the yellowing is more limited to certain leaf areas (Nilsson & Larsson 1990). Usually, sugar production beets are harvested in late September-October before the frost arrives (Cattanach *et al.* 1991), and late infection does not affect the yield as much because the plant has mostly developed by this time.

4. History and importance of virus yellows in Sweden and Europe

In Europe, virus yellows has been observed in sugar beet fields since the early 1900's, but the causal agent was not identified as a virus until the 1930's in the Netherlands (Björling 1948; Björling 1949). The origin of virus yellows is the coast of southeastern England where wild beet had been sighted with the disease before the outbreak in cultivated sugar beet (Schlösser 1953). Viruliferous aphids were probably caught in the coastal wind and flew to the Netherlands and France where they could continue the spread of virus yellows (Schlösser 1953).

An international collaboration between European nations began in 1930, but it was postponed due to World War II and it was resumed around the year of 1950. In both the UK and Sweden, the disease has been surveyed since 1946, which has helped establishing control measures and forecasts (Björling 1956; Qi *et al.* 2004).

In Sweden, about 10-15% of the sugar beet fields in western Skåne were affected by virus yellows between the years 1946-1948 and 1% of the fields in the southeastern part of Skåne called Österlen (Björling 1956). With time, the disease started to spread more widely within Sweden, probably due to the increase in planting of peach trees and cultivation of rape crops, and in 1954, 15% of the beets were infected in Skåne and 10% in Österlen (Björling 1956). In 1953-1954, systemic insecticides started to be used in sugar beet seed crops, which reduced the infections to 1.5% in Sweden, with exception of the epidemic in 1959 (Nilsson & Larsson 1990). The year of 1959 brought the highest infection rate to date in Sweden, almost all beet fields in Skåne were affected in October and the disease rate was as high as 57% (Nilsson & Larsson 1990). Even regions in Sweden that until this date had not been affected were also infested by viruliferous *M. persicae* (Nilsson & Larsson 1990). The warm weather in Europe this year contributed to high numbers of aphids that migrated with the wind over the Baltic Sea and Öresund (Nilsson & Larsson 1990). Since then, infections have been low, only with some yearly fluctuations, but in the year of 1974 sugar beets in the UK were severely affected by virus yellows due to the mild winters reducing the sugar yield by 40% and losses were estimated to be 14 million GBP (Russell 1978; Qi *et al.* 2004).

In the beginning of the survey of the disease in Sweden, BYV was the predominant virus, but in the 1950's a shift began and BMYV occurred more frequently onwards (Björling & Möllerström 1974). For only one year, 1959, BYV

was more dominant and the reason for this was probably the wind-spread aphids that were transferred from continental Europe to Sweden (Björling & Möllerström 1974). For some years, regional differences were observed, where BYV was more dominant, but generally, BMYV was predominant in the surveys which continued until 1973 (Björling & Möllerström 1974). This is the case in the UK as well, where BMYV has been predominant between 1980-2004 (Qi *et al.* 2004). Generally, BMYV is more common in the northern and western parts of Europe, whereas BYV is more common in the Mediterranean regions (Stevens *et al.* 2005b).

The spread of virus yellows in Sweden has probably been relatively small because the weather conditions do not allow overwintering of adult aphids and because beet seed production discontinued around the late 1960's - early 1970's (Björling & Möllerström 1974; Wiktelius 1977). Wind and warm weather are two conditions that induce greater spread of aphids and symptoms of virus yellows (Björling & Möllerström 1974). In Sweden, aphids may not migrate early in the season because of the cold weather, but only later when the crop is no longer as susceptible, which reduces the impact of virus yellows.

5. Aphids and virus transmission

The viruses causing virus yellows are spread by different aphid vectors. The green peach aphid (*Myzus persicae*) is the most effective one being able to transmit all viruses and *Aphis fabae* is a vector for BYV. The transmission efficiency for BYV with apterous *A. fabae* was calculated by Limburg *et al.* (1997) as 34%, compared to 60% for apterous *M. persicae*. Through experiments by Schliephake *et. al.* (2000) a transmission rate for BMYV of 1.1% for *A. fabae* has been established, something that is so low that it probably is of no importance.

Aphids of up to 22 species including also *Macrosiphum euphorbiae, Myzus ascalonicus* and *Myzus certus*, have been reported to spread virus yellows, but not all with the same efficiency (Heathcote 1988a). However, *M. euphorbiae* had only a transmission rate of 1.8% for BMYV, compared to a transmission rate of 28.6% with *M. persicae* (Schliephake *et al.* 2000).

The poleroviruses causing virus yellows are persistent, meaning that they are non-propagative and circulative (Gray & Gildow 2003; Stevens *et al.* 2005a). This means that BMYV, BChV and BWYV are not replicating inside the aphid, but circulating inside the aphids and are transported through the cells via endocytosis/exocytosis (Gray & Gildow 2003). In aphids, the virus is transported through the gut tissue and into the hemocoel and exits through the salivary glands, which enables the virus to be transferred to the phloem of plants via feeding (Gray & Gildow 2003; Brault *et al.* 2007).

To become viruliferous, the aphids need to feed on an infected plant for a certain amount of time. The type of virus that is present influences the time. For a viruliferous aphid to spread infection, it does also need a certain feeding time on healthy plants (Watson & Russell 1940).

The acquisition time for BYV is 7 minutes and inoculation in a healthy plant takes 30 minutes (Watson & Russell 1940). BYV has a retention time in *M. persicae* of 72 hours and in *A. fabae* of between 24-48 hours. The virus is not present after moulting and this is because the virus is semi-persistent and not circular (Russell 1962; Nilsson & Larsson 1990; Limburg *et al.* 1997).

The acquisition time for BMYV is longer compared to BYV since it is a persistent virus (Nilsson & Larsson 1990). BMYV has a latency period of 24 hours after acquisition (Nilsson & Larsson 1990). Russel (1962) concluded through experiments that a minimum of 24 hours of feeding on infected plants is necessary

for efficient transmission by *M. persicae* to healthy plants, with 48-72 hours of feeding on infected plants being the most effective for inoculation. Because of the circulative nature of BMYV, the virus can be present in the aphid even after moulting (Russell 1962). This indicates that BMYV can be present in *M. persicae* for its whole life and that viruliferous *M. persicae* can spread BMYV to many sugar beet plants during its lifetime (Smith 1988).

5.1. The green peach aphid, *Myzus persicae*

The green peach aphid is widespread and for a long time it has been recognized as the main vector for virus yellows (Heathcote 1988a). The lifecycle of *M. persicae* is complex, some aphids are holocyclic, i.e., sexual forms that develop under a short photoperiod and reproduce by laying eggs on primary host plants (Blackman 1972). There are also anholocyclic aphids which reproduce parthenogenetically on secondary host plants (Blackman 1972). Intermediate aphid populations that produce both sexual and asexual forms can also be found (Blackman 1972).

Under the growing season, the aphid can produce up to 20 generations in mild climates and the development of a generation takes about 10-12 days (Capinera 2001). There is a difference in appearance of the aphid depending on its developmental stage (Björling *et al.* 1955). During summer, there are wingless (apterous), yellowish green ones and winged (alatae), shiny black-spotted ones (Fig. 3) (Björling *et al.* 1955). The precursor form to the alatae is red. The summer aphids are all females called *fundatrigeniae* and can give non-fertilized birth during the season (Björling *et al.* 1955). The host plants are a wide range of weeds, garden plants and herbaceous crops (Björling *et al.* 1955).

Figure 3. Different development stages of M. persicae, alatae (left) and apterous (right) (Pictures from MariboHilleshög).

In autumn, a new generation of aphids is born as female alatae and males that search winter shelter on their primary host plants (Björling *et al.* 1955). The most important primary host plant is the peach tree, but also other *Prunus* species like the apricot tree and wild black cherry, *Prunus serotine* (Björling *et al.* 1955). The

aphids can lay their eggs on other plants as well, but the non-primary host plants are often not as suitable as a feeding source for the progeny and can result in death for the new-borns (Björling *et al.* 1955).

On the primary host trees, the winged female aphids give unfertilized birth to apterous sexual females (oviparae), which are alike in the appearance to the apterous summer aphids, but a bit redder in colour (Björling *et al.* 1955). The oviparae reproduce with the males and produce eggs which are laid on the cortex adjacent to the buds and overwinter (Capinera 2001).

In spring, the eggs hatch with apterous *fundatrices*, also named stem mothers, which start feeding on flower buds and young foliage (Capinera 2001). The stem mothers reproduce asexually, giving birth to living progeny (*fundatrigenia*), which in turn reproduce in the same manner as the stem mothers (Björling *et al.* 1955). When the population is dense enough, a generation of alatae are born that fly away from the primary host plant in May - June and attack herbaceous crops, weeds and gardens (Björling *et al.* 1955; Heie & Petersen 1961). During the summer, many generations of *fundatrigenia* and alatae are produced (Björling *et al.* 1955).

Aphids are generally poor flyers and cannot control the direction or speed if the wind speed exceeds 1 metre per second (Wiktelius 1977). Therefore, winged aphids can drift by the wind and be transported in the air for long distances (Wiktelius 1977). However, the migration of aphids is disadvantaged by rain and cold weather which can limit the migration to overwintering host plants during autumn, particularly in Scandinavia (Heie & Petersen 1961).

The *fundatrigenia* and alatae can survive in their active form during the winter if plants are available and the temperature is high, between 4-10℃ (Capinera 2001). *M. persicae* can also survive being inactive during severe frost for a shorter period and the eggs are also rarely affected (Heie & Petersen 1961; Heathcote 1988b). This means that the aphids can continue to reproduce asexually throughout the autumn and winter if the environment allows it (Björling *et al.* 1955). Glasshouses can have suitable conditions for overwintering, but the number of aphids is usually limited and restricted to a few plants (Heie & Petersen 1961). Storage clamps can be of importance, because aphids can seek shelter there and feed on the beets (Nilsson & Larsson 1990). Studies by Heie and Petersen (1961) showed that *M. persicae* cannot survive in clamps if the temperature is below 4℃ for three months in succession. Actions have been taken in Sweden because of this, for example by limiting the presence of outdoor clamps and shortening the storage time to the first of April (Björling & Möllerström 1974).

There is a possibility that overwintering of adult aphids becomes more common in the future in otherwise colder climates. In Sweden, data from SMHI shows an average temperature of 4-5℃ in the south of Sweden between December 2019 and March 2020 (SMHI 2020). The mean minimum temperatures all exceeded 0℃ (SMHI 2020), and these are suitable conditions for overwintering of *M. persicae*.

The mean temperatures between 1961-1990 were below 0°C during the winter months which concludes a drastic temperature rise during the latest decades (SMHI 2020) favouring overwintering for a large number of different insects.

5.2. The black bean aphid, *Aphis fabae*

The body of the black bean aphid (*Aphis fabae*) is dark green to black with dark antenna and cornicles (Fig. 4 & Fig. 5). It has an apterous developmental stage and alatae can occur if colonies become crowded or stressed (Godfrey & Trumble 2008). Usually, it forms colonies under the foliage (Godfrey & Trumble 2008), which reduces the efficiency of sprays, for example with pyrethroids.

Figure 4. Apterous A. fabae (Kohlmann 2013).

Figure 5. Alatae of A. fabae (Dupont 2014).

The black bean aphid is widespread, having over 200 different host plants globally and mainly causing yield losses in crops such as sugar beet and broad bean (Saruhan 2018). In sugar beet crops, *A. fabae* is the aphid that causes most feeding damage, to both young and older plants (Heathcote 1988a).

Winter hosts for *A. fabae* are mainly European spindle (*Eunymus europaeus*) and Guelder rose (*Viburnum opulus*) (Heie & Petersen 1961). If populations are dense on their primary winter host *E. europaeus*, there is a higher probability for reduced impact of natural enemies and populations often increase (Way & Banks 1968). As for other aphids, the occurrence and frequency of *A. fabae* are influenced by the weather, where warm and dry years usually result in higher aphid populations (Heie & Petersen 1961). Variation in population size of *A. fabae* can also be related to fluctuations in the presence of natural enemies, like parasitic fungi (Heie & Petersen 1961).

Large *A. fabae* populations might not always result in considerable spread of virus yellows. The number of *A. fabae* in sugar beet plants did not seem to correlate with the occurrence of virus yellows in field trials made by Heie & Petersen (1961). However, they did find a possible connection between the number of *M. persicae* and the occurrence of virus yellows. Possible explanations might be that *A. fabae* sometimes migrate to fields of sugar beet only late in the summer, which can give

M. persicae an advantage and probably account for the spread of virus yellows (Björling & Möllerström 1974). *A. fabae* also moves less frequently between plants compared to *M. persicae*, which limits spread (Heathcote 1988a).

6. Control measures

During the last 100 years, agricultural practises have advanced a lot, and this is a contributing factor to the low virus incidence in crops today. The main principles for limiting yield reduction by virus yellows are either to prevent virus transmission by vectors or to reduce infection symptoms via resistance or tolerance. Today, controlling spread and impact of virus yellows consists of using insecticides, limiting wintering clamps and early sowing. Because the aphids prefer to feed on young leaves, damage can to some extent be prevented by sowing early (Olsson Nyström 2019). An older plant will also have higher resistance to both the virus and foliar damage caused by aphids (Nilsson & Larsson 1990).

6.1. Insecticides

To limit virus yellows, managing the presence of vectors is the key control measure. Over the years, this has been done using different insecticides. Systemic insecticides were introduced in the 1950's and helped controlling aphid populations onwards (Nilsson & Larsson 1990). These insecticides were mainly organophosphates and carbamates, some containing compounds with high toxicity that are banned in most parts of the world today (Rapini & Marrazza 2016; Silberman & Taylor 2020). A few organophosphates and carbamates are still used in some European countries (Hauer *et al.* 2017), but the development of resistance in insects and new better alternatives have reduced the usage of these insecticide classes (Tomizawa & Casida 2005).

Today, neonicotinoids are the main insecticide class used for seed treatments of beets (Hauer *et al.* 2017). Imidacloprid was the first type of neonicotinoid introduced commercially in 1991 (Elbert *et al.* 1990). Around this time, several different types of neonicotinoids were discovered and patented. This includes the heterocyclic neonicotinoids: nithiazine, imidacloprid, thiacloprid, thiamethoxam as well as the the acyclic neonicotinoids: nitenpyram, acetamiprid, clothianidin and dinotefuran (Tomizawa & Casida 2005). Since then, no new major class of insecticides has been developed and put into market (Tomizawa & Casida 2005). Neonicotinoids are mainly used for seed treatments and are not efficient as a contact insecticide (Tomizawa & Casida 2005).

The neonicotinoids imidacloprid, thiamethoxam and clothianidin act systemically and protect the whole plant from sucking and piercing arthropods (Tomizawa & Casida 2005; Hauer *et al.* 2017). When used as a seed treatment, the

insecticide protects the plant for up to 12 weeks, which is the most critical growing stage (Dewar *et al.* 1996; Tomizawa & Casida 2005). In a field study by Dewar *et al.* (1996) the importance of imidacloprid was studied and it was concluded that the average yields of the four study sites were 19% higher than for the untreated control plots.

Imidacloprid is a worldwide-used neonicotinoid and surveys from the last years have shown imidacloprid resistance developing throughout the world in *M. persicae* (Srigiriraju *et al.* 2010; Umina *et al.* 2014; Voudouris *et al.* 2017).

Myzus persicae is one of the most resistant species to date and has developed a broad resistance to up to 75 different types of compounds used in insecticides (Sparks & Nauen 2015). The resistance to organophosphates, carbamates and pyrethroids is widespread in *M. persicae* (Nauen & Elbert 2003). Several studies have demonstrated that a broad resistance also to imidacloprid is starting to occur in *M. persicae*. Umina *et al.* (2014) carried out a survey in Australia, where they found high levels of resistance in *M. persicae* to both carbamates and synthetic pyrethroids, moderate levels of resistance to organophosphates and indication of resistance to neonicotinoids (Umina *et al.* 2014). Even more recent field data from tobacco crops in Greece shows that imidacloprid resistance is developing in *M. persicae* (Voudouris *et al.* 2017). Imidacloprid resistance in *M. persicae* has also been detected at peach farms in Italy (Panini *et al.* 2014) and in southern France and Spain (Slater *et al.* 2012). Resistance has also been found in eastern USA, where surveys in 2010 found moderate resistance to imidacloprid in *M. persicae* (Srigiriraju *et al.* 2010).

The EU have put severe restrictions on usage of the neonicotinoids clothianidin, imidacloprid and thiamethoxan since 2013 in outdoor farming in flowering crops and in 2018 a total ban was established (European Commission 2020). Extensive data suggests that the components in neonicotinoids are contributing to the death of pollinators (European Commission 2020). Sugar beet was originally not affected by this ban, because sugar beet that is cultivated for sugar production does not flower (Hauer *et al.* 2017). However, the ban of neonicotinoids in 2018 came to affect the sugar beet production as well (European Commission 2020).

Throughout the years, a few countries in Europe have got different exemptions which has led to some conflicts, for example, the exemptions for spring rape crops given to Finland but not to Sweden (Växtskyddsrådet 2016). The approval of clothianidin and thiamethoxan expired in 2019 and imidacloprid has an expire date of 31 July 2022 (European Commission 2020).

The ban on neonicotinoids might increase the usage of pesticides like pyrethroids, which Hauer *et al.* (2017) argue might pose a greater risk to pollinators and non-target organisms than neonicotinoids currently do. However, pyrethroids are not usually used in beet production (Hauer *et al.* 2017). Non-systemic sprays and foliar insecticides need careful monitoring of thresholds, timing is of great

importance and the virus infections need to be high for the measure to be economically sustainable (Dewar *et al.* 1996). As mentioned before, *M. persicae* is resistant to pyrethroids, but the usage of pyrethroids might increase for controlling other pests.

If no pest management would be able to control aphids, epidemics of virus yellows would probably be more frequent. Qi *et al.* (2004) estimated how high the percentage of infected fields in the UK would have been in the past without any pest management. Without any pest management, the UK would probably have had 11 potentially severe epidemics in the eastern region and 8 epidemics in the northern region since the worst epidemic in 1974 (Qi *et al.* 2004).

Since resistance in *M. persicae* has been noted, the efficiency and usage of neonicotinoids as a control measure against this particular pest might have been reduced in the future regardless. Having that in mind, the ban of neonicotinoids in the EU probably does not matter in the case of virus yellows. However, one thing is certain, new insecticides, which do not harm non-target organisms, are needed. Currently, there are few options for controlling aphids, and insects in general, and the risk for resistance is therefore higher (Hauer *et al.* 2017). There are currently some chemicals that might be good substitutions for neonicotinoids, but most of them are not available for use in beets at this moment (Hauer *et al.* 2017).

6.2. Reducing clamps and overwintering

The virus cannot persist in harvested beets in clamps or beet seeds, however, it is possible that the viruliferous aphids overwinter in clamps (Heie & Petersen 1961). To limit overwintering of aphids in clamps, it is important to limit the aphids on the beets by careful topping while harvesting, and by late clamping and covering up (Heie & Petersen 1961). Minimizing the time that beets are stored in clamps over all is also an important factor for reducing spread of virus yellows (Björling & Möllerström 1974).

It is also important to minimize overwintering beet plants and groundkeepers, because if infected they can spread disease the following year, and possibly to neighbouring fields (Björling & Möllerström 1974). A great way to minimize this is tillage after harvest.

Overwintering sugar beets and groundkeepers are usually not a problem in Sweden due to the cold weather (Björling & Möllerström 1974), but in warmer climates and when the weather has been warmer, this is for sure an important factor and might become a greater problem due to climate change. In beet seed crops, the crop is grown for two years, and this makes it possible for infected plants to overwinter and continue to spread the virus the second year. This might be problematic if neighbouring fields also are sown with beets or other susceptible

plants, like spinach, and weeds, like *Capsella bursa-pastoris* and *Chenopodium foliosum*, which might continue the spread (Björling & Möllerström 1974).

6.3. Host plants

The host range for BYV, BMYV, BWYV and BChV differs but they do share some host plants. BWYV has the largest host range, calculated to over 150 species in 23 families, although some variants and strains of BWYV differs in host range (Yoshida & Tamada 2019). BMYV has a more limited host range of 23 species in 8 families (Appendix Table 1) (Duffus & Russel 1970). BYV has a host range of over 120 species in 15 families of dicot plants (Appendix Table 1) (Duffus 1973; Agranovsky & Lesemann 2011). Most research has been carried out with BYV because it was the first one of the yellowing viruses of sugar beet that was discovered (Duffus 1973), and it is therefore possible that there are additional hosts of BMYV that have not been discovered yet.

The host plants are infection sources for virus yellows and can to some degree influence the amount of viruliferous aphids. Susceptible plants like autumn-sown spinach can influence the spread of virus yellows (Björling & Möllerström 1974). Infected overwintering weeds can be infection sources as well, particularly for BMYV (Jadot 1973). Perennial weeds can harbour viruses causing virus yellows for years in succession and are therefore of importance, *Chenopodium bonus-henricus* is an example (Björling 1958). Weeds and crops susceptible to these viruses should be taken into consideration if affected fields of sugar beets are occurring, because these alternative hosts can become infection sources. Careful crop rotation, where susceptible plants are not close to each other, and weed control might limit local spread.

6.4. Biological pest control

To use and promote natural enemies as pest control is a strategy preformed in IPM (UC IPM 2020). This is exercised in organic farming, but it is also a way to reduce the usage of chemical pesticides in conventional cropping systems. Natural enemies to aphids are parasitic fungi and different predatorial insects (Capinera 2001).

There are many natural enemies for *M. persicae* and *A. fabae*. The most common ones are lady beetles, flower flies, lacewings, parasitic wasps and entomopathogenic fungi (Capinera 2001). Promoting these insects by providing habitat and overwintering sites, as well as by limiting insecticide usage, can increase the efficiency of biological pest control (Thornhill 1988; Godfrey & Trumble 2008).

Pathogenic fungi have been used in biological pest control for decades where entomopathogenic fungi are important pathogens to Hemiptera and can regulate aphid populations through epizootics (Eilenberg *et al.* 2009). Fungi of the phylum *Entomophthoromycota* are naturally occurring in temperate regions and some species have the ability to regulate *M. persicae* (Ben Fekih *et al.* 2013). There have been incidents where entomopathogenic fungi have crashed aphid populations naturally, which indicates that they could be a possible microbial insecticide (Elkassabany *et al.* 1992). The problem with naturally occurring epizootics is that they often occur too late in the season, when damage already has been caused by aphids (Shah *et al.* 2004).

There have been experiments with the efficiency of the pathogenic fungi of the phylum *Entomophthoromycota* against aphids and they seem to have a potential (Eilenberg *et al.* 2009). However, experiments with the release of *Entomophthoromycota* and if it would have a long-lasting effect have not been conducted yet (Eilenberg *et al.* 2009). A commonly studied fungus species is *Pandora neoaphidis* which can infect a broad range of aphids in temperate regions (Shah *et al.* 2004). In an experiment by Shah *et al.* (2004), *A. fabae* and *M. persicae* were found to be moderately susceptible to infection of *P. neoaphidis*.

In an experiment by Saruhan (2018), six isolates of two entomopathogenic fungi, *Lecanicillium muscarium* (five isolates) and *Simplicillium lamellicola* (one isolate), were used against *A. fabae* with three different suspensions of conidia, 1×10^4 ,1×10^5 and 1×10^6 conidia ml^{-1}. For all fungal isolates and concentrations, treatments resulted in a high cumulative mortality rate (between 67 and 100%), with treatments using *L. muscarium* showing the highest cumulative mortality rate. For all fungal isoles, the highest concentration resulted in the highest cumulative mortality rate, peaking at six days after inoculation (Saruhan 2018). *Lecanicillium lecanii* is also an interesting entomopathogenic fungus for control of *M. persicae*. The mortality of *M. persicae* nymphs after treatment with different isolates of *L. lecanii* was calculated in an experiment by Diaz *et al.* (2009) revealing a mortality

ranging between 57 and 95% when using a dose of 1×10^9 conidia ml^{-1}. At daytime, the temperature was 23℃ and at night, it was 18℃.

Similar results have occurred in experiments by Vu *et al.* (2007) where the most potent strain 41185 of *L. lecanii* induced 100% mortality after 4 days of treatment with conidia at 25℃ and 90% relative humidity. The same strain had a high virulence at 45% relative humidity with a broader temperature span, 25-30℃. The temperature influenced the conidial germination and the strain 41185 had a low growth at temperatures below 20℃ (Vu *et al.* 2007).

If the most potent strain of *L. lecanii* or *L. muscarium* would serve as a biopest control, the usage would probably be limited to warm greenhouses or temperate and warm climates considering the temperature and relative humidity restrictions. Sweden is probably too cold in the spring for *L. lecanii* or *L. muscarium*, but more southern parts of Europe might fulfil the criteria of the fungus. One practical problem is also the usage of fungicides in sugar beet crops, if fungicides would be used in the crop, the entomopathogenic fungi would die as well. Fungi like *Aphanomyces cochlioides* and *Pythium ultimum*, which cause seedling damping-off disease, *Erysiphe betae* which causes powdery mildew, and *Ramularia beticola* are important pests in sugar beet crops that are controlled with fungicides (Luterbacher *et al.* 2000; Thach *et al.* 2013). However, the use of natural enemies as pest control seems promising, and it would prove a good resource for controlling insecticide-resistant aphid species.

6.5. Resistant sugar beets

Developing resistant beet cultivars has been a goal for plant breeders since the 1930's when virus yellows became widespread (Björling & Möllerström 1974; Koch & Lowe 1988). Resistant beets would both reduce the costs of insecticides for farmers and have a positive environmental impact (Koch & Lowe 1988). With the effectiveness of insecticides and the low occurrence of virus yellows in crops today, resistance to virus yellows has not been a priority for plant breeders during the last decades. As discussed earlier, due to the ban of neonicotinoids in the EU, virus yellows could potentially become more frequent in the future and new control measures are therefore of importance (Hauer *et al.* 2017). Resistance or tolerance should be part of a broad control strategy because resistance can often be broken by the pathogen if it only is managed by a single control mechanism (Vale *et al.* 2001). This is the case with insecticides as well, prolonged exposure to the same type of insecticide class results in resistance developing faster in pests. Durable resistance is preferred in breeding (Vale *et al.* 2001), but even if the resistance is broken within a relatively short time, it would serve as a useful component in

disease management. A combination of control measures would be most sustainable.

Lines of sugar beet, which are tolerant/partially resistant to virus yellows have been developed at USDA-Salinas in California and they were released in 2002 (Lewellen 2004; Stevens *et al*. 2005a). These lines are multigerm, self-fertile diploid that segregate for male sterility (Lewellen 2004). They have shown tolerance to rhizomania, BYV, BChV and BWYV with a generally high sugar yield and low bolting (Lewellen 2004).

Today, there are no commercial cultivars of sugar beets with resistance to virus yellows in the EU (SLU 2019). However, there is a possible market for it which recently has been noted by different plant breeders.

In Sweden, the Danish/Swedish company MariboHilleshög in cooperation with SLU Grogrund attempts to develop resistant varieties to virus yellows (SLU 2019). The BMYV-resistant genotype shows no visual symptoms and has a considerable difference in foliage compared to susceptible genotypes (Fig. 6).

Figure 6. Comparison of a BMYV-resistant sugar beet genotype (left) and a susceptible genotype (right) in a field trial by MariboHilleshög (Pictures from MariboHilleshög).

In the past it has proven difficult to develop cultivars with resistance to virus yellows without compromising traits important for production like yield and bolting (Cleij 1964; Koch & Lowe 1988). Trials with sugar beet resistant to BYV and BMYV have been taking place in the EU several times with promising results (Cleij 1964; Russel *et al.*1972; Koch & Lowe 1988). Some of these genotypes were not monogerm, which is otherwise normal in today's agriculture (Koch & Lowe 1988).

Six possible types of resistance to BMYV and BYV have been proposed by Björling (1966b) and Russel (1978). Today, there is more precise information about the molecular mechanisms controlling resistance, but the explanations from Russel (1978), Russel *et al.* (1972) and Björling (1966) are nevertheless great basic knowledge which is focused on virus yellows in sugar beets.

The first one is complete immunity, where the virus is not multiplying in the plant and there is no effect on the plant (Russell 1978). This is the case in all non-susceptible crops and weeds. Immunity should not be confused with extreme resistance, because immunity is absolute and plants with extreme resistance become infected even though the effect of infection is low (Russell 1978).

The second mechanism that was proposed by Russel (1978) is resistance to virus infections, when a susceptible plant escapes the infection even if it has been exposed to viruliferous aphids. This type of resistance is often preferred for breeding and successful trials of sugar beet genotypes with this trait have been carried out (Russel 1978). Breeding for resistance to both BYV and BMYV with this trait have proven difficult in the past (Russel 1978), but is a reality today (Lewellen 2004).

The third mechanism of resistance is to minimize the spread of the virus in the plant. The virus is not spread further from the point of inoculation, which limits the risk of widespread symptoms in the plant and aphids becoming viruliferous (Russel 1978). The most common resistance mechanism in this category is hypersensitivity, where cells die prematurely in response to a virus infection (Russell 1978). Many signals are involved in hypersensitivity, and the cell death is likely a signal to the plant to initiate its direct defence mechanisms (Heath 2000). Leaves usually develop necrotic spots, which are called local lesions, where inoculation has taken place. Typically, the virus stays in the local lesions or adjacent cells, but sometimes they re-localize to healthy tissues (Russel 1978). Factors like temperature and light can make it possible for the virus to re-localize and continue spreading (Russell 1978).

To the plant breeder, hypersensitivity has been one of the easiest and most effective resistance methods (Russell 1978). The downside of hypersensitivity is that it often is strain specific and loses effect if a virus strain would break the resistance (Russell 1978). This phenomenon is more frequently occurring for some plant families and crops compared to others, and plants of the family Solanaceae are often affected (Russell 1978).

Antiviral properties of the host are also a factor when it comes to limiting the spread of virus yellows in the plant. If the plant has a strong antiviral response when inoculated, the spread will be limited (Russell 1978).

Resistance to virus multiplication is the fourth mechanism. The concentration of the virus decreases and as a result the uptake of virus by aphids can be lower.

The spread to neighbouring plants can in this way be reduced or delayed (Russell 1978).

The term virus tolerance is used when a susceptible plant has reduced or barely any symptoms at all when infected (Russell 1978). True tolerance, where no symptoms are induced after infection, is the type of tolerance that is preferred in plant breeding programs. However, tolerance can be expressed in different ways and is not always absolute.

For instance, there is a term called symptomless carrier, which is when the plant does not display any visual symptoms, but the infection still damages the host (Russel 1978). This can be expressed in infected sugar beets, where there are no symptoms like leaf yellowing, but root mass and sugar concentration are still reduced (Russel 1978). In sugar beets, this is not a tolerance trait that is preferred for breeding, because yield depends on the root mass and sucrose concentration.

There is also a tolerance term called disease tolerance that is the other way around, where the sugar beet plants display visual symptoms like leaf chlorosis, but the root mass and sugar concentration are not that affected (Russel 1978). If true tolerance would be hard to achieve, this would probably be the second-best option.

The main problem with tolerance is that the yellowing viruses are still present in the host plants. Viruliferous aphids are then still able to spread the viruses to sugar beet fields with non-tolerant varieties or other host plants.

Resistance to virus yellows can also be achieved by making the plants resistant to aphids, i.e., resistance to aphid settling, aphid multiplication or tolerance to feeding damage (Russell *et al.* 1972). There have been experiments with different sugar beet genotypes resistant to both *M. persicae* and *A. fabae*. Russell *et al*. (1972) found that some variants of beets were less attractive for the aphids resulting in reduced aphid multiplication and settling. There seems to be different resistance mechanisms when it comes to controlling the vectors, because beet lines that were less attractive to *M. persicae* were more susceptible to *A. fabae* in experiments by Russell *et al.* (1972).

As mentioned, many experiments with virus yellows resistance in beets have been taking place and there are many different resistance mechanisms that can be taken into consideration. Because of the launching of resistant beets in the USA (Lewellen 2004), launching virus yellows resistant sugar beets in the EU does not seem to be impossible. If launched in the EU, virus-resistant cultivars would prove a great control measure against virus yellows.

7. Discussion

There is a great risk that the incidence of virus yellow increases in the coming years because of several factors: climate change affecting aphid populations and stresses in sugar beets, the lack of effective available insecticides and the absence of resistant variants discussed earlier. With sugar beet being a very profitable crop (Nordic Sugar 2018), this is alarming for both farmers and producers.

Before virus yellows becomes more frequent again, using already established control measures and developing new ones are of importance. The promising future control measures for virus yellows that can be explored are the usage of biological control, resistant cultivars and allowing existing insecticides in sugar beet crops (Kosh & Lowe 1988; Hauer *et al.* 2017). The most sustainable option is to have a wide range of control measures to delay the time for pests developing resistance (Hoy 1998). The problem lately has been that many pesticides are banned or stop working due to resistance in pests, but no new insecticide classes have been put into market since 1991 (Tomizawa & Casida 2005). This only aggravates the problem with pest resistance.

Recent technical progress in plant breeding, like the usage of genome editing with CRISPR/Cas9, could increase the chances for breeders to successfully develop pest-resistant sugar beet cultivars (Belhaj *et al.* 2015). However, this technology is classified as GMO in the EU, which restricts its usage (Callaway 2018). It is possible to use conventional breeding techniques to achieve virus yellows resistance in sugar beets as well, which is something that has been proven in the past. However, having a wide range of breeding techniques available would be ideal. The UK has been severely affected by virus yellows in the past and the EU laws affected their agriculture as well as other EU countries. However, since their recent withdrawal from the EU, they might have the ability to modify these laws in the future.

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Appendix

Table 1. Host range for BYV and BMYV[1].

Host plants	BYV	BMYV
Aizoaceae:		
Aizoon spp.	+	/
Mesembryanthemum crystallinum	+	/
Sesuvium portulacastrum	+	/
Tetragonia echinata	+	/
Tetragonia expansa	/	+
Amaranthaceae:		
Achyranthes aspera	+	/
Amaranthus retroflexus	+	/
A. albus	+	/
A. aureus	+	/
A. cararu	+	/
A. cruentus	+	/
A. caudatus	+	/
A. deflexus	+	/
A. tricolor	+	/
A. palmeri	+	/
A. paniculatus	+	/
A. patulus	+	/
Atriplex canescens	+	/
A. coronata	+	/
A. coulteri	+	/
A. elegans	+	/
A. expansa	+	/
A. hastata	+	/

+ = susceptible - = resistant / = unknown

[1] (Russell 1965; Björling & Nilsson 1966a; Duffus 1973; Bar-Joseph *et al.* 1979; Stevens *et al.* 1994).

Table 1 (continued)

	BYV	BMYV
Amaranthaceae:		
A. microcarpa	+	/
A. nitens	+	/
A. patula	+	/
A. rosea	+	/
A. semibaccata	+	/
A. siberica	+	/
A. spongiosa	+	/
Bassia hyssopifolia	+	/
B. scoparia	+	/
Beta atriplicifolia	+	/
B. cicla viridis	+	/
B. hybrida	+	/
B. lomatagona	+	/
B. macrocarpa	+	+
B. maritima	+	+
B. patellaris	+	/
B. patula	+	/
B. procumbens	+	/
B. trigyna	+	/
B. vulgaris	+	+
B. vulgaris var. cicla	+	+
B. webbiana	+	/
Blitum capitatum	+	+
B. nuttallianum	+	/
Celosia argentea	+	/
C. cristata	+	/
Chenopodium album	+	-
C. amaranthicolor	+	-
C. bonus-henricus	+	+
C. foliosum	+	+
C. giganteum	+	/
C. glaucum	+	/
C. hybridum	+	/
C. jicifolium	+	/
C. leptophyllum	+	/
C. murale	+	/
C. nutans	+	/

+ = susceptible - = resistant / = unknown

Table 1 (continued)

	BYV	BMYV
Amaranthaceae:		
C. opulifolium	+	/
C. polyspermum	+	+
C. quinoa	+	-
C. suecicum	+	/
C. urbicum	+	/
C. vulvaria	+	/
C. watsonii	+	/
Cycloloma atriplicifolium	+	/
Dysphania ambrosioides	+	/
D. botrys	+	/
Gomphrena globosa	+	+
Oxybasis rubra	+	/
Salsola kali	+	+
Spinacia oleracea	+	+
S. tetrandra	+	/
Suaeda fruticosa	+	/
S. nigra	+	/
S. splendens	+	/
Asteraceae:		
Glebionis segetum	-	+
Senecio vulgaris	+	+
S. macrophyllum	+	/
Sonchus oleraceus	+	/
Zinnia elegans	-	+
Borgainaceae:		
Pectocarya pusilla	+	/
Brassicaceae:		
Capsella bursa-pastoris	+	+
Sinapis alba	/	+
S. arvensis	/	-
Thlaspi arvense	+	-

+ = susceptible - = resistant / = unknown

Table 1 (continued)

	BYV	BMYV
Caryophyllaceae:		
Arenaria leptoclados	/	+
Cerastium viscosum	+	/
Dianthus deltoides	+	-
Gypsophila elegans	+	/
Silene armeria	+	/
S. coronaria	+	/
S. gallica	+	/
S. verecunda	+	/
Spergula arvensis	+	+
Stellaria media	+	+
Convolvulaceae:		
Cuscuta calijornica	+	/
C. campestris	+	/
C. gronovii	+	/
Convolvulus occidentalis	+	/
C. tricolor	+	/
Fabaceae:		
Melilotus indica	+	/
Lamiaceae:		
Lamium purpureum	+	+
Myrsinaceae:		
Anagallis arvensis	-	+
Papaveraceae:		
Papaver dubium	+	/
P. rhoeas	+	+
P. somniferum	+	/

+ = susceptible - = resistant / = unknown

Table 1 (continued)

	BYV	BMYV
Plantaginaceae:		
Plantago erecta	+	/
P. insularis	+	/
P. lanceolata	+	/
P. major	+	/
P. rumosa	+	/
Veronica spp.	-	+
Portulacaceae:		
Claytonia perfoliata	+	+
Portulaca grandiflora	+	/
P. oleracea	+	-
Resedaceae:		
Reseda odorata	+	/
Solanaceae:		
Nicotiana clevelandii	+	/
N. quadrivalvis	+	/

+ = susceptible - = resistant / = unknown

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