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# INFECTIOUS DISEASES OF HORSERADISH (*COCHLEARIA ARMORACIA* L.) IN POLAND

## ABSTRACT

Poland is an important horseradish grower in Europe, therefore the problems concerning its cultivation and diseases are considered vital. Horseradish diseases can be classified as noninfectious and fungal, bacterial or virus origin. The most important fungal disease is white-rust caused by *Albugo candida*, which can be responsible for up to 50% loss of yield, in the case of heavy infection. Bacterial diseases are not very important now. Turnip mosaic virus (TuMV), arabis mosaic virus (ArMV) and tomato black ring virus (TBRV) were found so far on horseradish in Poland. The most important is TuMV, which was found in almost 100% collected horseradish samples and caused yield loss about 40%. The virus TuMV belongs to the *Potyvirus* genus (VC57.0.1) from the *Potyviridae* family (VC57), which is the largest family of plant viruses. It infects many economically important plants. Attempts to eradicate TuMV by means of thermotherapy proved ineffective. At the Research Institute of Vegetable Crops, methods of obtaining TuMV-free horseradish plants (together with methods of its fast multiplication) have been developed in *in vitro* cultures.

*Key words:* *Cochlearia armoracia*, diseases: fungal and viral, horseradish

## INTRODUCTION

Horseradish (*Cochlearia armoracia* L., *Armoracia rusticana* (Gaerth)) has not been a popular subject of scientific reports, and its diseases have enjoyed even less attention. It is one of the most important spicy vegetables (Courter and Rhodes 1969). Edible part of horseradish is the root, which has a characteristic pungent taste. The taste is produced by allyl isothiocyanate ($C_2H_5CNS$) and butyl thiocyanate ($C_4H_9CNS$) released on crushing the root.

In Poland horseradish is an economically important plant, grown in particular in the region of Sieradz, where the area of its cultivation makes up about 50% of the total area occupied by this species in this country and which produces about 50% of the country's yield. Other large areas of its cultivation are near Warszawa, Piotrków and Szczecin.

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In total, horseradish is grown on 2000 - 3000 ha. On a small- or amateur-scale it is commonly grown in the whole country. It has been an economically important plant and recently a demand for it has grown again after a few years of decline. Horseradish growers are also encouraged by the fact that some food processing plants had to buy this vegetable from abroad, e. g. Hungary.

On a commercial scale horseradish is reproduced only vegetatively from root cuttings, which are prepared from thick (0.8-1.4 cm in diameter) lateral roots, 30-35 cm long. To produce commercial root at least 25 cm long of first grade quality, the minimum length of the cutting should be 30 cm. Unfortunately, the cuttings are often a source of disease, which can spread over the whole plantation.

In general horseradish is resistant to pests and diseases, however, along with intensification of production and because of permanent vegetative reproduction, diseases have become a limiting factor.

Horseradish diseases can be classified as noninfectious, fungal and virus borne.

## THE FUNGAL DISEASES OF HORSERADISH

The most important fungal disease in horseradish is white-rust caused by *Albugo candida* (Fiedorow 1971, Kurzawińska and Poniedziałek 1987, Macias 1996, 1997 - Table 1), which can be responsible for as much as 50% loss in yield in case of heavy infection.

Table 1

**Pathogens found on horseradish in Poland**

| Causative agent | Source of literature |
|---|---|
| Fungi | |
| *Albugo candida* | Fiedorow 1971, Kurzawińska and Wiech 1982 |
| *Cercospora armoraciae* | Macias 1996, 1997 |
| *Ramularia armoraciae* | Fiedorow 1971, Kurzawińska and Wiech 1982 |
| *Verticillium* | Fiedorow 1971, Macias 1997 |
| Viruses | |
| *Turnip mosaic virus (TuMV)* | Twardowicz-Jakusz *et al.* 1977, Macias 1997, Kochman and Stachyra 1960, |
| *Arabis mosaic virus (ArMV)* | Twardowicz-Jakusz *et al.* 1977, Macias 1997 |
| *Tomato black ring virus (TBRV)* | Twardowicz-Jakusz *et al.*1977 |

*A. candida* attacks 100 species of cultivated plants but is most dangerous for horseradish. In Poland, besides horseradish from among cultivated species it attacks also rape and gold of Pleasure. In the initial stadium *A. candida* causes chlorotic spots on the upper surface of the leaves. Later the spots become prominent and turn reddish or violet in colour. On the bottom surface of leaf blades, in the places corre-

sponding to the spots, white bubbles appear. At first they are single, small and their diameter is only 1-2 mm. Later they grow bigger, new bubbles appear and form crusty rings. In severe cases they can cover the whole surface of the leaf. Such projections can be found also on upper surfaces of the leaves and on leaf petiole, floral shoots and flower stems (Macias 1996). The white structures contain conidial spores, which when released in favourable conditions form zoospores and spread the infection. *Albugo candidia* is a good example of a disease, which causal agent requires high relative humidity of air for its development, and rather a drop of water. Infection spreads always at higher humidity and particularly rainy wheather. The optimum temperature for germination of conidia is 10-15°C (a possible germination temperature range from 0-28°C). As the conidia are endowed with flagella they can move in water and invade plants through stomata. The whole process takes from 3 to 30 hours, depending on environmental conditions. The spots on the upper leaf surface become visible after 6-8 days and the white projections 2-3 days later. This fungus also attacks the roots. Infection of the roots is manifested as discolored thickening (reddish or brownish), cracks along the root and root rotting during long-term storage. Symptoms of infection are difficult to notice at an early stage of infection and therefore, the roots are the main source of spreading the disease. The fungus can also survive winter on infected leaves and the dormant bodies can initiate a new infection in the spring (Fiedorow 1971). Under favourable conditions of temperature and humidity oospore forms on short hyphae a bubble – like floating spores which are a resorvoir of infections bodies for the next growing season. However, according to Macias (1996), this source of infection is not important. Frequently besides *Albugo candida* in the place of leaf infection develops another species of fungus, *Peronospora cochleariae*. This fungus only seldom is found on its own, but it adds to the leaftissue deterioration caused by white rust.

For the control of the disease it is vital to observe 3-4 year crop rotation on the same field. Remnants of the plants should be removed from the field and distroyed at the time of the root harvest. Most important is the use of pesticides. Horseradish cuttings should be dressed before planting out. The cuttings could be treated by immersion in suspension of Ridomil MZ 72 WP 0.3% or with fungicides containing mancozeb for 30 min. Instead they can be carefully sprayed or sprinkled if they are kept in furrows. The treatment is simple and effective so it should become a common practice (Macias 1997). In the vegetation period the plants should be sprayed first of all with mancozeb preparations (i. e. Dithiane 75 WG, Dithane 455 S.C., Nemispor 80 WP, Penncozeb 75 WP). First treatment is performed at the time when first symptoms are visible and then it is repeated every 7 – 10 days, depending on wheather conditions.

In late summer other fungal diseases can appear on horseradish leaves, for example *Cercospora armoraciae* (Fiedorow 1971, Kurzawińska and Wiech 1982). It caused dark brown spots on both sides of the leaves, which turn pale and sometimes are surrounded with brown slightly protruding rings. The spots, in particular those on the upper surfaces of the leaf blades, in their central parts are covered with conidial spores. Heavily infected leaves eventually wither and die.

The symptoms caused by the fungus *Ramularia armoraciae* (Fiedorow 1971) are similar, although the spots are lighter and smaller. At the centre the spots become crusty, so after some time tiny holes appear. The control of the disease requires similar procedures as in the case of *Albugo candida*.

From the practical point of view the disease known as root blackening is very important (Błaszczak *et al.* 1977). This condition can be caused by five factors (Macias 1997). Taking into regard the frequency of appearance, intensity and yield loss, the most important form of horseradish blackening in Poland, is the non-infectious root blackening. Similar symptoms of root blackening can be caused by the fungus *Verticillium*. The signs of infection with this fungus are discoloration of fibre-vascular bundles in the main root. At first they are yellow, then gradually turn brown and finally almost black. The discolorations can be found along the entire length of the root and are most pronounced at the apical end. In other parts of the root they are hardly visible with a naked eye. The lateral roots do not reveal the symptoms. Older leaves wilt, especially on the plants in flowering. The leaves turn yellow, fast wither and their conductive bundles become black. Verticillosis is spread by infected cuttings or infected soil. This disease can be controlled by planting the cuttings into *Verticillium*-free soil, use of healthy cuttings and crop rotation by every 4-5 years, so that the dormant forms could die out.

In mid nineties a new horseradish disease was stated in Poland (Macias and Ślusarski 1998). Its sympoms are dry, brownish spots on the roots. For this reason the disease was named dry brown spot of horseradish roots. The surface of the spots becomes hardy and corky eventually turning brown. The corky tissue peals off. In the ruptures of the outer skin and pealing tissue pycnidia of the fungus visible with naked eyes are appearing. Around the spots adventitious roots emerge, which, however deteriorate gradually. The infected plant becomes stunted, the oldest leaves wilt, turn yellow and die. Sometimes chlorotic spots are present on the leaves. At the end of the growing season, on the leaf petioles sometimes slightly concave elongated spots are present with visible aggregates of pycnidia. The attacked plant can be easily pulled off from the ground.

Causing agent of this disease is fungus *Phoma lingam*. It is anamorph of the fungus *Leptosphaeria maculans*, which so far was not found on horseradish. This is the first report of horseradish infected by *Phoma*

*lingam* in Poland (Macias and Ślusarski 1998). There was also no evidence available in literature on presence of this disease on horeseradish in other countries. It was believed that horseradish is a species very resistant to *Phoma lingam*, which is a very common pathogen on plants belonging to family *Cruciferae*. Reasons for occurrence and rapid spread of this disease on horseradish are unknown. One can suppose that this could be caused by avoiding crop rotation, which often is true on small specialized farms, where crop rotation is practically impossible to apply. The disease was found in the main region of horseradish cultivation located around Sieradz. In 1997 an average infestation was about 50-60% but on some farms it reached almost 100%.

Dry rot of horseradish roots is a new disease so official recommendation of its chemical control have not been reported yet. So at present the most important is observing principles of proper soil management, after all:

1. using only healthy planting material avoiding material from own farm,
2. performing proper crop rotation so horseradish would be cultivated on the same field after at least 4 years,
3. accurate collection and destroying of plant remnants,
4. weeds control which are hosts of this pathogen,
5. avoidance of horseradish cultivation on wet and cold soils with poor air – water properties.

## VIRAL DISEASES OF HORSERADISH

Only three viruses: turnip mosaic virus (TuMV), arabis mosaic virus (ArMV), and tomato black ring virus (TBRV) have been found so far on horseradish grown in Poland (Twardowicz-Jakusz *et al.* 1977, Kochman and Stachyra 1960). Of these three the most important is TuMV. According to Macias (1997) it is being found in almost 100% of collected horseradish samples. As reported by Paludan (1973) TuMV caused a 37% yield loss. A similar result, a 40% loss was reported by Macias and Górecka (1989). Experiments carried out at the Research Institute of Vegetable Crops have proved (Macias 1997) that early infection, instantly after leaf sprouting from a cutting, has more damaging effect on the yield than planting virus-infected cuttings. Infection of cuttings later in the growing season did not cause a marked drop in the yield, however, it increased the incidence of internal darkening of roots. Arabis mosaic virus (ArMV) causes less acute symptoms. It can be responsible for a reduction of the plant growth even when the symptoms are obscure or unnoticed. This virus occurrence is bound to a given area and it is spread by the saprophytic nematodes from the genus *Longidorus*. The virus TBRV is seldom found in Poland.

**Turnip mosaic virus (TuMV)**

Turnip mosaic virus (TuMV) is a member of the *Potyvirus* genus (VC57.0.1.), classified in the *Potyviridae* family (VC57.), the largest family of plant viruses. The family is composed of at least 200 distinct species. The *Potyviridae* group causes significant losses in agricultural, pasture, horticultural and ornamental crops (Ward and Shukla 1991). Turnip mosaic virus has a very wide experimental host range, infecting 318 species in 156 genera of 43 families (Edwardson and Christie 1991). It occurs world-wide in temperate and tropical regions. In an extensive survey of economically important field vegetable viruses occurring in 28 countries, turnip mosaic virus was found to be one of the most important (Tomlinson 1987). It naturally infects many of economically important *Brassica* crops: oilseed rape, cauliflower, Chinese cabbage, calabrese, Brussels sprout, kale, and other nonbrassica crops and ornamentals: peas, lettuce, chicory, rhubarb, stocks and wallflowers. These infections cause significant losses (Shattuck 1992). Turnip mosaic virus has been found on horseradish in many countries also (Dana and McWhorter 1932, Pound 1948, Chenulu and Thornberry 1965, Thornberry 1960, Shukla and Schmelzer 1972). The complete nucleotide sequence of one Canadian and one Japanese isolates have been reported (Nicolas and Laliberte 1992, Ohshima *et al.* 1996). Several partial nucleotide sequences of different TuMV isolates were also available (Kong *et al.* 1990, Nakashima *et al,* 1991, Sano *et al.* 1992, Nakashima *et al.* 1993, Petrzik and Lehmann 1996, Lehmann *et al.* 1997). The TuMV genome is 9830-9833 nt long, excluding a 3' terminal poly (A) tail. A viral protein (VPg) is covalently attached to the 5' end of genomic RNA. The 3'-untranslated region of TuMV is 209 nucleotides long (Petrzik and Lehmann 1996), and can be predicted to fold into a stable secondary structure (Turpen 1989). The 3'-untranslated sequences of TuMV isolates are highly conservative. An AUG triplet at position 130-132 was assigned as the initiation codon for the translation of the genome size viral polyprotein which would consist of 3163-3164 amino acid residues. This polyprotein is proteolytically processed by three virus-encoded proteinases into the following mature proteins: protease (P1), helper component-protease (HC-Pro), third protein (P3), peptide ($6K_1$), cylindrical inclusion protein (CI), peptide ($6K_2$), small nuclear inclusion protein (NIa), large nuclear inclusion protein (NIb) and coat protein (CP). (Fig. 1).

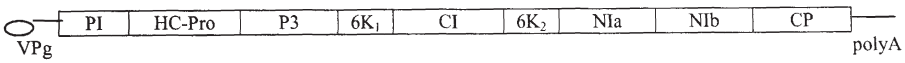


Fig. 1 Map of the turnip mosaic virus genome.

P1 – protease, HC-Pro – helper component-protease, P3 –third protein, $6K_1$ – peptide, CI – cylindrical inclusion protein, $6K_2$ – peptide, NIa – small nuclear inclusion protein, NIb – large nuclear inclusion protein and CP- coat protein, VPg – viral protein genome linked. Open reading frame encoding the polypeptide is represented as an open bar, horizontal solid lines stand for non-coding regions.

Turnip mosaic virus (TuMV) causes chlorotic spots on the leaf blades, sometimes the first symptoms are the leaf-veins turning pale and smaller leaf-blades. Chlorotic rings can be present and then become necrotic, as necrosis of veins may sometimes happen. In summer time, during intensive growth of plants, these symptoms are less visible or sometimes they are absent, but in fall they again become acute. If horseradish plantation suffers because of shortage of mineral nutrients, nitrogen in particular, the plants which are infected by the virus demonstrate severe chlorosis, whereas plants supplied with enough of nitrogen or vigorous plants, e. g. obtained by *in vitro* method, can look healthy. Some strains of the virus evoke strips on thick leaf veins and leaf petioles. These strips can sometimes reach the root. TuMV is transmitted with the propagating material, i. e. by cuttings. During the growing season the disease is spread by aphids (on stiletto in non-permanent way).

**Arabis mosaic virus (ArMV)**

Arabis mosaic virus (ArMV) is a member of the *Nepovirus* genus (VC18.03.), belonging to the *Comoviridae* family (VC18.). *Nepoviruses* are characterized by isometric particles and by their nematode transmission. Their genome consists of two separately encapsidated, single-stranded RNAs of positive polarity. Each RNA is bound to a genome-linked protein (VPg) at its 5' end and is polyadenylated at its 3' end. Arabis mosaic virus is readily sap-transmissible, has a wide host range, infects the seed of many host plants, and it is transmitted by the nematode *Xiphinema diversicandatum*. The ArMV spreads in the African region, the Eastern Asian region, the Eurasian region, the North American and the Pacific regions. Arabis mosaic virus occurs naturally in many species of wild and cultivated monocotyledonous and dicotyledonous plants. It infected 93 species in 28 dicotyledonous families when the inoculation was done mechanically (Shukla and Schmelzer 1972). The ArMV occurs in many plants including raspberry, strawberry, cucumber, lettuce, celery, sugar beet, hop, rose, white clover, grapevine and horseradish (Hickman and Varma 1968, Novak and Lanzova 1982). It was found on horseradish in Poland, too (Twardowicz-Jakusz *et al.* 1977, Macias 1997).

Arabis mosaic virus has a bipartite single-stranded RNA genome of approximate $M_r$ values of $2.4 \times 10^6$ and $1.4 \times 10^6$ respectively (Harrison and Murant 1977). It has a single coat protein of approximately 55K, and isohedral particles of 28 nm in diameter, composed of 60 coat protein monomers in a T=1 presumably pseudo T=3 structure (Takemoto *et al.* 1985). Translation of the two RNAs gives two polyproteins: RNA-1 encodes the protease which cleaves the two polyproteins into functional units, whereas RNA-2 encodes the coat protein (CP), the gene for which has been shown to be 3'-terminal for grapevine fanleaf virus (GVFLV) (Serghini *et al.*1990).

**Tomato black ring virus (TBRV)**

Tomato black ring virus (TBRV) is a member of the *Nepovirus* genus (VC18.03.), belonging to the *Comoviridae* family (VC18). Host range of TBRV is wide: infects naturally many species of wild and cultivated monocotyledonous and dicotyledonous plants. An isolate from *Robinia pseudoacacia* infected 76 species in 29 dicotyledonous families after mechanical inoculation. The tomato black ring virus occurs in many plants including: tomato, bean, sugar beet, lettuce, raspberry, strawberry, celery, potato, peach, leek, onion, cabbage, grapevine, and lucerne. The TBRV was found on horseradish also (Shukla and Schmelzer 1972, Thomas 1973) and occurs on horseradish in Poland too (Twardowicz-Jakusz *et al.* 1977).

Tomato black ring virus has a genome consisting of two ssRNA species of $M_r$ $2.69 \times 10^6$ (RNA-1) and $1.56 \times 10^6$ (RNA-2) (Murant *et al.* 1981). Both species are 3'-polyadenylated (Mayo *et al.* 1979) and contain a 5'-terminal genome-linked protein (VPg) (Koenig and Fritsch 1982). It has bipartite genomes, although the larger RNA is able to replicate independently of the second RNA in inoculated protoplasts (Goldbach *et al.* 1980; Robinson *et al.* 1980). The smaller RNA of TBRV (RNA-2) codes for the coat protein (Franssen *et al.* 1982). When translated *in vitro* each virus RNA induces the synthesis of polyproteins corresponding to their entire coding capacity (Fritsch *et al.* 1980). Cleavage of the TBRV polyprotein yielded no functional protein except coat protein, that could be detected *in vivo* and *in vitro*. The nucleotide sequence of TBRV RNA-1 and RNA-2 have been determined (Meyer *et al.* 1986, Greif *et al*. 1988).

**Attempts to obtain TuMV-free horseradish**

An attempt was made to eradicate TuMV from horseradish plants by thermotherapy. Unfortunately it appeared to be not very successful; the infection was eliminated only in one of three experiments (Holmes 1965). Paludan (1973) reported the inactivation of TuMV in horseradish cuttings by keeping them at 38°C for 60 days. The treatment proved effective – 83% virus-free cuttings were obtained. The description of this experiment was concise but without much detail. Błaszczak *et al.* (1977) reported that in two experiments conducted at 37°C for 30 or 35 days, many root cuttings died and rotted. Moreover, the cuttings which survived were not TuMV-free. In view of this result, attempts were made to eliminate TuMV from horseradish roots by the tissue culture method. Another reason for using of the *in vitro* method was a high rate of horseradish multiplication desired for breeding purposes. As mentioned above, despite abundant flowering horseradish did not produce seeds, which require special labour consuming plant treatment and, therefore the multiplication rate achieved is only 2-4. At the Research Institute of Vegetable Crops, methods for obtaining TuMV-free horseradish plants and ensuring a high rate of their multiplication were developed using *in*

*vitro* cultures (Górecka 1992). TuMV-free plants were obtained from meristem cultures (Górecka *et al.* 1989). About 40% of produced plants were virus-free. The healthy plants were subsequently used as a source of explants for fast propagation of healthy horseradish plants by tissue cultures. The initial explants were triangular leaf-fragments of about 1 cm in size. The multiplication rate was about 40 in one passage and we considered it rather high. The plantlets (rosettes) rooted easily apart from glass – in over 90% (Górecka 1992). The plants grew very well and gave a yield markedly higher than the controls of the first year in field (Macias and Górecka 1989). Taking into account the fact that TuMV has so many host species, including weeds, it is understandable that reinfections take place easily and in the first year 60% plants were reinfected. This finding and successful experiments on obtaining TuMV-resistant rape plants drew our attention towards horseradish transformation as a means to obtain TuMV-free plants.

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