https://doi.org/10.15407/microbiolj84.03.092

H.V. TSEKHMISTER*, A.S. KYSLYNSKA

Laboratory of Plant-Microbial Interactions,
Institute of Agricultural Microbiology and Agroindustrial Manufacture, NAAS of Ukraine,
97 Shevchenka Str., Chernihiv, 14027, Ukraine

*Author for correspondence; e-mail: anna.tceh@gmail.com

PLECTOSPHAERELLA MELONIS (SYN. *ACREMONIUM CUCURBITACEARUM*) — PLANT PATHOGENIC ORGANISM

Fungal diseases cause significant damage to agriculture. Plectosphaerella melonis (syn. Acremonium cucurbitacearum and Nodulisporium melonis) is a pathogen of cultivated plant diseases in Spain, Italy, Japan, USA, Egypt, and Ukraine. This review discusses the main results of research related to this phytopathogen. By morphological and cultural features, P. melonis is a morphologically intermediate species between A. strictum and A. charticola, however, 5.8S-ITS region-based phylogenetic analysis showed that P. melonis is a monophyletic taxon more closely related to Plectosphaerella than to other species of the genus Acremonium. The most susceptible plants are at the stage of germination; however, the development of the disease is manifested in the fruiting period. For a comprehensive assessment of virulence, real leaf area (RLA) of the first two leaves, lesion of hypocotyl (RH), root collar (RSR), primary (R1R) and secondary roots (R2R) are measured. P. melonis affects the root system, in particular the root collar and hypocotyl, and colonizes the epidermis and cortex of the root centrographically towards the stem. The range of host plants includes Cucurbitaceae, however, peppers, tomatoes, basil, and parsley are infected as well. Plants vary in susceptibility depending on the species and even variety. The pathogenic response of plants differs depending on the growing conditions (protected and open soil), the interaction between the pathogen and competing microorganisms, and other ecological and trophic relationships. The main means of control are the use of long-term crop rotations and the selection of resistant varieties. In Ukraine, a strain of the antagonist fungus Trichoderma viride was selected, which is an effective means for controlling P. melonis 502. The aim of our work was to establish the role of P. melonis in the development of diseases of cultivated plants.

Keywords: *Acremonium cucurbitacearum, Plectosphaerella melonis, Nodulisporium melonis, Cucumis.*

Citation: Tsekhmister H.V., Kyslynska A.S. *Plectosphaerella melonis* (Syn. *Acremonium cucurbitacearum*) — Plant Pathogenic Organism. *Microbiological journal.* 2022 (3). P. 92—100. https://doi.org/10.15407/microbiolj84.03.092

Over the last 30 years, cases of a new plant disease of Cucurbitaceae, caused by *Plectosphaerella melonis* (syn. *Acremonium cucurbitacearum* and *Nodulisporium melonis*), have been reported worldwide [1—14]. There are a number of names in the literature for this disease, in particular, "vine decline", sudden wilting, but the most popular is "acremonium collapse" [4]. In 1982, in California (USA), Gubler described early melon plant disease caused by *Cephalosporium cucurbitacearum* [15]; however, Alfaro-Garcia believes that Gubler most likely dealt with *P. melonis* [4]. Further cases of the disease were reported in California in 1993 and 1994, where the pathogen was isolated from collapsed watermelon and musk melon [2], which formed further grounds to study the pathogenic properties of *P. melonis* California isolates [2, 12]. In 1996, the same disease was reported on melon plants in Texas (USA) [16]. In Spain, after the mass death of early melons at the beginning of fruiting, Garsia-Jimenez et al. found that the causative agent was the fungus *P. melonis* [1, 4]. In 2002, *P. melonis, A. crotonigenum* (Schol-Schwarz) W. Gams, and A. sclerotigenum were isolated from affected melon plants in Italy, but only inoculation by *P. melonis* reproduced the disease symptoms [14, 17]. In 2012, a case of pathogenicity of *P. melonis* 502 against cucumber plants in Ukraine was reported [18]. In 2017, the disease was registered in melons in Egypt [19], and in 2021 in Italy [20].

Taxonomic features. When the pathogen was isolated for the first time, it was morphologically and culturally identified by Alfaro-García, W. Gams & García-Jiménez as a new species of *A. sucurbitacearum* and included in the *Acremonium* section [4]. The fungus is a morphologically intermediate species between *A. strictum* and *A. charticola* (Lindaau) W. Gams, and other related species are *A. sclerotigenum* and *A. kiliense* Grfitz [4]. In 2004, molecular genetic methods confirmed the affiliation of the pathogen to a new species of *A. sucurbitacearum* [13]. 5.8S-ITS region-based phylogenetic analysis showed that *A. cucurbitacearum* is a monophyletic taxon more closely related to *P. cucumerina* (92%) than to the other species of the genus *Acremonium* (68—72%), and all isolates of *A. cucurbitacearum* form a monophyletic group, as well as isolates of *P. cucumerina* are clustered nearby. The fungi *A. strictum, A. charticola, A. sclerotigenum, A. kiliense*, and *A. crotocinigenum* are significantly separated from *A. cucurbitacearum* [13]. Carlucci et al. have shown that A. cucurbitacearum is synonymous with *Nodulisporium melonis* [21] and *P. melonis* comb. nov. [22].

It is noteworthy that the phytopathogen *Plectosporium tabacinum* (teleomorph of *P. cucumerina*) resembles some species of *Acremonium*, but they differ in teleomorphs and morphology. Furthermore, the results of genetic studies indicate a higher genetic affinity between *A. cucurbitacearum* and *P. tabacinum* than with the other species of *Acremonium* [23, 24]. Therefore, currently, it is customary to call *A. cucurbitacearum* as *P. melonis*, although both names are legitimate in the MYCOBANK database.

Origin of isolates. The geographical origin of *P. melonis* is unknown, but the disease was detected in North America and Europe at the same time. Abad et al. have investigated the relatedness between European and American isolates of A. cucurbitacearum. For this purpose, they used the method of vegetative compatibility analysis (VCG), which allows studying the genetic diversity among populations of phytopathogenic fungi. This method is based on the ability of two physiologically different strains of the same species to connect asexually, thus forming a stable heterokaryon. In general, strains of fungi that anastomose and form heterokaryons with each other are considered vegetatively compatible and belong to the same group [25]. In general, Abad et al. obtained 10

different VCGs of *P. melonis* [24, 26]. Molecular genetic studies and analysis of the vegetative compatibility of isolates revealed that American isolates of *P. melonis* are similar or identical to Spanish ones [27, 28]. However, RAPD model-based cluster analysis obtained using 25 primers divided *P. melonis* isolates from Spain and the United States into two main groups, most isolates from the United States fell into group 1, but genetic similarity did not correlate with the geographical origin or previously established VCG groups [13].

Symptoms. Lesions of the root system of plants infected with *P. melonis* were detected at the stage of germination. After sowing the seeds in the soil, the formation of light yellow-brown spots was registered on the hypocotyl, which eventually roughened. Hypocotyl discoloration is a differential symptom caused by *P. melonis*, which makes it possible to diagnose the disease at the early stages. At the same time, the seedlings discolor and die. Two weeks after sowing the seeds, the formation of necrotic spots was registered on the root hairs of plants. At the end of the growing season, the main roots had a bare appearance and ulcers in the places where the secondary roots were attached. Later on, lesions appeared on small seedlings, while the formation of transverse circles was observed on the roots of mature plants [1, 29]. Additional roots, which branched from the hypocotyl were formed above the lesion site [4].

In field experiments, aboveground symptoms were manifested during the period of the maximum need of plants for moisture [1], this mainly took place at the stage of fruiting, i.e. 3 months after planting in the soil [30, 31]. The affected plants showed yellowing of old leaves, necrosis of leaf blade tissue, loss of turgor of young plants, wilting, and death of the stem. It is characteristic that plant growth retardation or soaking was not observed [1].

The pathogenicity test in the vegetation experiment confirmed the sequence of symptoms: there were distinct yellowish-brown areas after about 10 days between the hypocotyl and the root system; root collar bark covering and secondary root death, which was accompanied by the formation of lateral roots from the hypocotyl, was registered on Day 20 of vegetation [1]. This is a kind of protective reaction to pathogens of root diseases [30—32]. It is important to note that aboveground symptoms did not appear before the fruiting phase [1]. In the works of other authors, it is noted that *P. melonis* causes the greatest damage to seedlings in the root collar [11].

Symptoms of damage to the root system of cucumbers in the vegetation experiment were observed 14 days after sowing seeds in the soil infected with infected *P. melonis* 502. Cucumber plants of Nizhynskyi 12 variety were very susceptible to the pathogen at the stage of growing two real leaves (2 weeks after sowing). Terrestrial symptoms were absent after 14 days, although at this time there were noticeable lesions on the root system [18].

At the early stages of growth, the ability to absorb water decreases, which leads to gradual yellowing and wilting, or even complete collapse [30—32]. At the beginning of ontogenesis of such plants, moisture supply is sufficient for growth and development, however, at the stage of mass fruiting of Cucurbitaceae, when water demand is maximum, the affected root system is unable to fully provide the plant with moisture [30, 31, 33].

Plants inoculated with *P. melonis* had a larger diameter of the main root. At the same time, there was a decrease in the length of roots with a diameter of less than 0.5 mm and in the total number of roots, as well as a reduction in the rhizosphere volume and leaf surface area [34]. Biernacki notes that *P. melonis* caused the most damage to small roots, reducing the area of the root system rather that its mass [34].

Determining the area of the first two real leaves may be more indicative than the mass

of the root system to study the harmfulness of root pathogens. Bruton et al. have shown that the study of only the mass of the root system to assess the harmfulness of *P. melonis* is insufficient because the additional roots that branch from the hypocotyl of affected plants offset the actual decrease in the root mass of primary and secondary roots [11]. For a comprehensive assessment of the virulence of *P. melonis* isolates, it was proposed to measure the area of the first two real leaves (RLA) to assess the lesion of hypocotyl (RH), root collar (RSR), primary (R1R), and secondary roots (R2R) [11].

Location. The pathogen is always isolated only from the root system. Moreover, *P. melonis* colonizes the epidermis and cortex of the root of plants, and hyphae form a continuous and branched network, which spreads through these tissues. Colonization of the root system by hyphae of *P. melonis* occurs centrographically towards the stem, with the development of hyphae inside cells and intercellular space, and a decrease in the density of the pathogen towards the endoderm, which serves as a barrier to fungal infections. Hypertrophied and hyperplastic cells of the parenchyma are further necrotized [35].

In the experiments with melons, the first visible effect of *P. melonis* penetration into the root-hypocotyl zone of melons was atypical reddening of the epidermis and external parenchymal cells of the cortex due to suberin deposition (large-scale plant response to pathogens). The suberized layer was observed 30 days after infection of the soil with the fungus. After 42 days, the outer cortical cells of the hypocotyl were somewhat distorted and discolored. Xylem vessels did not change in 30-day-old infected plants, however, after 42 days calluses were found on the inner walls of the xylem. The etiology of callus formation is debatable, but it is known that these structures reduce the supply of water to the plant in conditions of high demand for moisture, which may be the reason for their depletion [35, 36].

After 42 hours, pumpkin plants, which were characterized by partial resistance to *P. melonis*, have shown a slight deposition of suberin, which is associated with slower colonization compared to melon plants. Pumpkin plant tissues contain a high amount of cucurbitacins — inhibitors of fungi, which in turn determines their tolerance to acremoniosis [8, 35].

The internal tissues of the root of melons and watermelons were rarely colonized, but hyphae were sometimes fixed even inside the lumen of the xylem vessels. Alfaro-Fernández et al. suggest that this is the result of extremely severe complications of the infection. Despite the colonization of the xylem, no fungal structures were found in the vessels of the hypocotyl, which indicates the impossibility of spreading *P. melonis* in the vessels of host plants [35].

Host plants. The range of *P. melonis* host plants is limited. It was previously thought that the fungus was pathogenic only to the Cucurbitaceae family, although it is commonly isolated from plants of other families. *P. melonis* is capable of limited colonization of the roots of some dicotyledonous plants of the families Asteraceae, Fabaceae, Malvaceae, Poaceae, and Solanaceae [7]. However, it is now known that *P. melonis* caused disease in basil, parsley, tomato, and pepper [37—39].

Garsia-Jimenez et al. have tested 41 wild plant species for susceptibility to *P. melonis*. All studied representatives are excluded from the possible range of host plants. It is also possible to grow other crops, including watermelons and cucumbers, on soils infected with *P. melonis*, as they have slower development of symptoms [1].

Armengol et al. have studied the pathogenicity of Spanish isolates of *P. melonis* to 31 species of plants of the Cucurbitaceae family, 18 species of cultivated plants and 15 species of weeds. They found that a wide range of plants of the Cucurbitaceae family could be potential host plants with different levels of susceptibility. Almost all varieties of melons (*Cucumis melo*) and water-

melons (*Citrullus lanatus*) are classified as susceptible or very susceptible. Pumpkin (*Cucurbita maxima*), luffa (*Luffa acutangula*, *L. aegyptiaca*) and wax gourd (*Benincasa hispida*) were highly resistant, and stuffing gourd (*Cyclanthera pedata*) — highly endurable. Other cultivated plants and weeds grown on contaminated soils are not affected by the fungus. It is interesting to note that the response to pathogen infection in melon and watermelon plants is the same, but to date, there are no special issues with watermelon acremoniosis. In fact, musk melon fields, where the disease was reported, are recommended for growing watermelons. In addition, in Spain, cucumbers (*C. sativus*) are less affected, although cucumber plants range from resistant to highly susceptible [7].

The pathogenicity of Texas isolates of *P. melonis* against Ciucurbitacea was studied further. Of the 37 representatives studied, only 22 were very resistant (of which the most resistant are representatives of the genus *Cucurbita — C. moschata* and *C. maxima*). Virtually all plants of the genera *Cucurbita*, *Luffa* and *Lagenaria* were classified as highly resistant. From the genus *Cucumis*, three varieties of cucumber plants (*C. sativus cultigens*) were evaluated as very resistant. *C. lanatas* and *C. melo* species are moderately resistant and susceptible. There were no very susceptible plants among the representatives of Cucurbitaceae [11]. Symptoms ranged from minor root damage or discoloration (in resistant species) to moderately or severely damaged hypocotyl, primary and/or secondary roots (in susceptible species). *P. melonis* was isolated from the lesion site of all tested Cucurbitaceae [11].

Iglesias et al., studying the disease, in a field small-scale experiment found that there is intraspecific susceptibility and the spread of the disease varies depending on the variety of melon. After 50—69 days of planting in the soil, they recorded the death of 10—20 % of plants of susceptible melon varieties. In contrast, Pat 8 variety musk melon was characterized by partial resistance (delayed onset of symptoms, reduced spread and development of the disease). At the end of ontogenesis, the number of healthy Pat 81 melon plants was 45—85 %, while in the susceptible melon varieties VC-185, VC-186, VC-187, and BG-13819 it was 0—15 % [30]. Iglesias et al. note that the size and morphology of the root system of plants is one of the key factors in their resistance to root pathogens. Thus, the resistant Pat 81 variety of musk melon is characterized by a well-developed and branched root system, which promotes the formation of new roots [31]. The plants of this variety presented a large number of additional roots that branched from the hypocotyl over the affected areas [30]. Therefore, determining the range of virulence of pathogens is a necessary factor for the effective cultivation of crops, which requires reliable ways to assess their virulence.

Environmental effect on disease development. The pathogenic response of plants differs depending on the growing conditions (protected or open soil), the interaction between the pathogen and competing microorganisms, and other ecological and trophic interaction [11]. For example, California isolates of *P. melonis* in field small-plot experiments were found to be slightly pathogenic for melons, however, in vegetation experiments, the degree of plant damage was much higher, and the dry weight of the aboveground part decreased by 23 %. Aegerter notes that despite the colonization of the root system with *P. melonis*, the affected plants in some cases continued to bear fruit [12].

Bruton, who studied the pathogenicity of *P. melonis*, noted that the response of muskmelon to acremoniosis can vary depending on the stage of development and level of plant resistance, density and virulence of pathogens, and environmental conditions. The development of *P. melonis* in plant roots is significantly influenced by the depth of seedling planting. The optimum for the uniform spread of *P. melonis* was

4 cm [11]. Heat stress is also an important factor influencing the development of acremoniosis symptoms [12].

At the same time, Grogan et al. [40] note that it is impossible to achieve 100% spread of the disease. Even with a high density of infectious material, the disease spread curve approaches the plateau, as part of the plant population may be highly resistant. Also, only certain areas of the root are susceptible to acrimoniosis [11].

The significant influence of environmental conditions on the development of this disease has made its study quite difficult because the lack of control of environmental factors makes it necessary to repeat field experiments in different years [30, 31].

Control means. This is a relatively new and little-studied disease. Information about its biology and epidemiology is not enough to develop a control strategy. Quite large areas of crops are occupied under protected soil, and crop rotation in most cases is not used, which in turn leads to the accumulation of pathogenic microbiota. For example, in Spain, the disease was registered in fields that were previously occupied by citrus monoculture for 8 years [7]. Schmidt et al. showed that cover crops increase *P. melonis* in the soil regardless of the soil processing mode [41]. Since *P. melonis* is able to form chlamydospores [4, 5], it is effective to use long-term crop rotations to limit the spread of the disease [7]. Given the narrow range of *P. melonis* host plants [7, 15], seedlings of other crops and weeds can limit the accumulation of infectious material in the soil. Given not only the interspecific but also the intraspecific tolerance, the selection of resistant varieties is not inferior [11].

Information on the biological control of the disease spread, in particular the use of microbial preparations, is limited. In Ukraine, a strain of the antagonist fungus *Trichoderma viride* 017, which is an effective means against *P. melonis* 502 has been selected [42—45]. *In vitro* screening of antagonist microorganisms against *P. melonis* 502 allowed selecting a strain of *Bacillus* sp. 23 (restrained colony growth even after 25 days) and micromycetes *C. cochliodes* 3250 (completely consumed the pathogen colony, showing hyperparasitism on Day 15 of co-cultivation) and *T. viride* 017 (showed hyperparasitism on Day 15 of cultivation) as the most active [42]. The biological efficiency of the pre-sowing treatment of cucumber seeds with *T. viride* 017 on an artificial infectious background (AIB) of *P. melonis* 502 was 61%. Inoculation of cucumber seeds with the fungus *T. viride* 017 contributed to the formation of a stronger root system and increased yields by 34 % (15.4 t/ha) compared to the variant with AIB [45].

The composition of Se and I nanoparticles against *P. melonis* 502 was also investigated. It was found that the composition slows down the growth and reduces the diameter of the colony, but does not lead to mycelial death. The composition of Se and I nanoparticles shows high fungistatic activity throughout the cultivation period. Reduction of colony diameters under the action of Se and I composition reaches 94.13% [45].

Conclusions. The phytopathogen *P. melonis* is the causative agent of root rot in cultivated plants and causes the greatest damage to young plants at the stage of germination. The root collar and hypocotyl are involved the most commonly. The range of host plants includes mainly Cucurbitaceae. Plants vary in susceptibility depending on the species and even variety. The main control means is the use of long-term crop rotations and the selection of resistant varieties.

Acknowledgements. The authors express their sincere gratitude to Olena Volodymyrivna Nadkernychna, the Head of the Laboratory of Plant-Microbial Interactions at the Institute of Agricultural Microbiology and Agroindustrial Manufacture of NAAS of Ukraine for the critical analysis of the manuscript and comprehensive support.

REFERENCES

1. Garsia-Jimenez J, Velazquez MT, Jorda C, Alfaro-Garcia A. Acremonium species as the causal agent of muskmelon collapse in Spain. Plant Dis. 1994; 78:416—419.
2. Bruton BD, Davis RM, Gordon TR. Occurrence of Acremonium sp. and Monosporascus cannonballus in the major cantaloupe and watermelon growing areas of California. Plant Dis. 1995; 79:754.
3. Gubler WD, Zitter NA, Hopkins DL, Thomas CE. Acremonium hypocotyl rot. Compendium of cucurbit diseases. 1 ed. St. Paul: Amer. Phytopathological Society; 1996.
4. Alfaro-García A, Armengol J, Bruton BD, Gams W, García-Jiménez J, Martínez-Ferrer G. The taxonomic position of the causal agent of Acremonium collapse. Mycologia. 1996; 88:804—808.
5. Cluck T, Bruton C, Biles C. Effect of carbohydrates on chlamydospore production of Acremonium cucurbitacearum. In: Proc. Okla. Acad. Sci. 85th Technical Meeting Oklahoma State University; 1997 November 08; Oklahoma, USA. 1997. p. 136.
6. Armengol J. Aspectos patológicos, epidemiológicos y culturales de Acremonium cucurbitacearum Alfaro-Garcia, W. Gams et J. Garcia-Jiménez. Ph.D. diss. Universidad Politecnica de Valencia, Valencia, Spain, 1997.
7. Armengol J, Sanz E, Martínez-Ferrer G, Sales R, Bruton BD, García-Jiménez J. Host range of Acremonium cucurbitacearum, cause of Acremonium collapse of muskmelon. Plant Pathol. 1998; 47:29—35.
8. Bruton BD. Soilborne diseases in Cucurbitaceae: Pathogen virulence and host resistance. In: Cucurbitaceae '98: evaluation and enhancement of Cucurbit germplasm; 1998 November 30 — December 4; Alexandria, VA. ASHS Press. 1998. p. 143—66.
9. Bruton BD, Garcia-Jimenez J, Armengol J. Analysis of the relationship between temperature and vine declines caused by Acremonium cucurbitacearum and Monosporascus cannonballus. Subtrop. Plant Sci. 1999; 51:23—28.
10. Bruton BD, Garcia-Jimenez J, Armengol J, Popham TW. Assessment of virulence of Acremonium cucurbitacearum and Monosporascus cannonballus on Cucumis melo. Plant Dis. 2000; 84:907—913.
11. Bruton BD, Popham TW, García-Jiménez J, Armengol J, Miller ME. Disease reaction among selected Cucurbitaceae to an Acremonium cucurbitacearum isolate from Texas. Hortscience. 2000; 35(4):677—680.
12. Aegerter BJ, Gordon TR, Davis RM. Occurrence and pathogenicity of fungi associated with melon root rot and vine decline in California. Plant Dis. 2000; 84(3):224—230.
13. Martinez-Culebras PV, Abad-Campos P, Garcia-Jimenez J. Molecular characterization and PCR detection of the melon pathogen Acremonium cucurbitacearum. Eur J Plant Pathol. 2004; 110:801—809.
14. Chilosi G, Reda R, Aleandri MP, Camele I, Altieri L, et al. Fungi associated with root rot and collapse of melon in Italy. OEPP/EPPO Bulletin. 2008; 38:147—154.
15. Gubler WD. Epidemiology and control of Cephalosporium root and hypocotyl rot of melon. Davis: University of California; 1982.
16. Bruton BD, Miller ME, Garcia-Jimenez J. Comparison of Acremonium sp. from the Lower Rio Grande Valley of Texas with Acremonium sp. from Spain. Phytopathology. 1996; 3:86.
17. Infantino A, Uccelletti A, Stefano G, Ciuffreda G, Frisullo S. First report of Monosporascus cannonballus on melon in Italy. J Plant Pathol. 2002; 84:139—140.
18. Kopilov E, Tsekhmister H, Nadkernychna O, Kyslynska A. Identification of Plectosphaerella melonis from cucumber plants in Ukraine. Phytopathologia Mediterranea. 2021; 60(2):259—263.
19. Mostafa MA, Attia MF, Merghany MM, Salama RM. Association of Plectosphaerella melonis with cantaloupe decline for the first time in Egypt. Plant Archives. 2019; 19(2):2565—2573.
20. Infantino A, Balmas V, Schianchi N, et al. Diversity of soil-borne fungal species associated to root rot and vine decline of melon in Sardinia (Italy). J Plant Pathol. 2021; 103:421—432. doi:10.1007/s42161-021-00774-9.
21. Watanabe T, Sato M. Root rot of melon caused by Nodulisporium melonis in Japan. II: Identification. Annals Phytopathological Society of Japan. 1995; 61:330—333.
22. Carlucci A, Raimondo ML, Santos J, Phillips AJL. Plectosphaerella species associated with root and collar rots of horticultural crops in southern Italy. Persoonia — Molecular Phylogeny and Evolution of Fungi. 2012; 28(1):34—48.
23. Vicente MJ, Cifuentes D, Cenis JL, Abad P. RAPD-PCR polymorphism and vegetative compatibility group variation in Spanish isolates of Acremonium cucurbitacearum. Mycol Res. 1999; 103:1173—1178.
24. Abad P, Pérez A, Marqués MC, Vicente MJ, Bruton BD, et al. Assessment of vegetative compatibility of Acremonium cucurbitacearum and Plectosphaerella cucumerina isolates from diseased melon plants. OEPP/EPPO Bulletin. 2000; 30: 199—204.

25. Joaquim TR, Rowe RC. Vegetative compatibility and virulence of strains of Verticillium dahliue from soil and potato plants. Phytopathology 1991; 81:552—558.
26. Abad P, Hack T, Vicente MJ, Bruton BD, Garcı´a-Jime´nez J. Vegetative compatibility groups in Acremonium cucurbitacearum. In: Diagnosis and Identification of Plant Pathogens. Proceedings of the 4th Symposium of the European Foundation for Plant Pathology; 1997 September 9—12; Bonn, Germany. Dordrecht, the Netherlands: Kluwer Academic Publishers. 1997. p. 287—9.
27. Vicente MJ, Abad P, Cenis JL. RFLPs del ADN mitocondrial, RAPDs y VCGs de Acremonium sp.: marcadores genéticos para su identificación taxonómica. In: La Protección Vegetal en España; 1994; Madrid. Vicente: Instituto Nacional de Investigación y Tecnología Agraria y Alimentaria. 1995. p. 401—15.
28. Abad P, Hack T, Vicente MJ, Bruton BD, Garcia-Jimenez J. Vegetative compatibility groups in Acremonium cucurbitacearum. In: Diagnosis and Identification of Plant Pathogens. Proceedings of the 4th Symposium of the European Foundation for Plant Pathology; 1996 September 9—12; Bonn, Germany. Dordrecht, the Netherlands: Kluwer Academic Publishers. 1997. p. 287—9.
29. Chilosi G, Reda R, Aleandri MP, Camele I, Marcone C, Altieri L, Montuschi C, Rossi V, Carlucci A, Lops F, Mucci M, Raimondo ML, Frisullo S. Occurrence of fungi associated with melon colapse in Italy. In: 12th Congress of the Mediterranean Phytopathological Union; 2006 June 11—15; Rhodes Island, Greece. p 133—135.
30. Iglesias A, Picó B, Nuez F. Pathogenicity of fungi associated with melon vine decline and selection strategies for breeding resistant cultivars. Ann Appl Biol. 2000; 137:141—151. doi:10.1111/j.1744-7348.2000.tb00046.x
31. Iglesias A, Pico B, Nuez F. Resistance to melon dieback in C. melo ssp. agrestis Pat 81. Phytopathol. 1999; 89:35.
32. Garcia-Jimenez J, Velazguez T, Alfaro-Garcia A. Secuencia de sintomas en el colapso del melon. Bol San Veg Plagas. 1989; 15:333—342.
33. García-Jiménez J, Martínez-Ferrer G, Armengol J, Velázquez MT, Orts M, et al. Agentes asociados al colapso del melon en distintas zonas españolas. Bol San Veg Plagas. 1993; 19:401—423.
34. Biernaki M, Bruton BD. Quantitative response of Cucumis melo inoculated with root rot pathogens. Plant Dis. 2001; 85:65—70.
35. Alfaro-Fernández A, García-Luis A. Colonisation and histological changes in muskmelon and autumn squash tissues infected by Acremonium cucurbitacearum or Monosporascus cannonballus. Eur J Plant Pathol. 2009; 125(1):73—85.
36. Pivonia S, Cohen R, Kafkafi U, Ben Ze'ev IS, Katan J. Sudden wilt of melons in Southern Israel: fungal agents and relationship with plant development. Plant Dis. 1997; 81:1264—1268. doi:10.1094/PDIS.1997.81.11.1264.
37. Carlucci A, Raimondo ML, Santos J, Phillips AJL. Plectosphaerella species associated with root and collar rots of horticultural crops in southern Italy. Persoonia — Molecular Phylogeny and Evolution of Fungi. 2012; 28(1):34—48.
38. Raimondo ML, Carlucci A. Characterization and pathogenicity of Plectosphaerella spp. collected from basil and parsley in Italy. Phytopathologia Mediterranea. 2018; 57(2):284–295.
39. Raimondo ML, Carlucci A. Characterization and pathogenicity assessment of Plectosphaerella species associated with stunting disease on tomato and pepper crops in Italy. Plant Pathology. 2018; 67(3):626–641.
40. Grogan RG, Sall MA, Punja ZK. Concepts for modeling root infection by soilborne fungi. Phytopathology 1980; 70:361—363. doi: 10.1094/Phyto-70-361.
41. Schmidt R, Mitchell J, Scow K. Cover cropping and no-till increase diversity and symbiotroph: saprotroph ratios of soil fungal communities. Soil Biology and Biochemistry. 2019; 129:99–109.
42. Tsekhmister HV, Kyslynska AS, Pavlenko AA. Antahonistychna aktyvnist gruntovykh mikroorhanizmiv yak efektyvnyi zasib zakhystu roslyn vid akremoniozu. Silskohospodarska mikrobiolohiia. 2019; 30:46–53. Ukrainian.
43. Tsekhmister H, Khalep Yu, Khareba O. Ekonomichna y enerhetychna efektyvnist peredposivnoi obrobky nasinnia ohirkiv hrybom Trichoderma viride. Visnyk ahrarnoi nauky. 2021; 99(9):72–79. Ukrainian.
44. Pavlenko AA, Kopylov EP, Tsekhmister HV. [Efficacy of Trichoderma viride strain with high antagonistic and cellulolytic activity]. Agricultural microbiology. 2021; 33:88–95. Ukrainian.
45. Kopylov YeP, Tsekhmister HV. Vplyv hryba-antahonista Trichoderma viride 017 na produktyvnist roslyn ohirkiv. Silskohospodarska mikrobiolohiia. 2018; 27:74—79. Ukrainian.
46. Derevianko SV, Vasylchenko AV, Tsekhmister HV. Biolohichna aktyvnist kompozytsii nanochastynok nemetaliv. Ahroekolohichnyi zhurnal. 2020; 1:111–115. Ukrainian.

Received 22.12.2021

Г.В. Цехмістер, А.С. Кислинська

Інститут сільськогосподарської мікробіології та агропромислового виробництва НААН України,
лабораторія рослинно-мікробних взаємодій,
вул. Шевченка, 97, Чернігів, 14027, Україна

PLECTOSPHAERELLA MELONIS (SYN. *ACREMONIUM CUCURBITACEARUM*) —
ЗБУДНИК ХВОРОБ РОСЛИН

Грибні хвороби завдають значних збитків сільському господарству. *Plectosphaerella melonis* (синоніми — *Acremonium cucurbitacearum* і *Nodulisporium melonis*) є збудником хвороб культурних рослин Іспанії, Італії, Японії, США, Єгипта та України. В цьому огляді обговорено основні результати досліджень, пов`язаних із цим фітопатогеном. За морфолого-культуральними ознаками, *P. melonis* є морфологічно проміжний вид між *A. strictum* та *A. charticola*, проте філогенетичний аналіз, що базується на регіоні 5.8S-ITS, показав, що *P. melonis* є монофілетичним таксоном, більш тісно пов'язаним з *Plectosphaerella*, ніж з іншими видами роду *Acremonium*. Найбільш чутливими для ураження є рослини на стадії сходів, але розвиток захворювання проявляється в період плодоношення. Для комплексної оцінки вірулентності вимірюють площу перших двох справжніх листів (RLA), ураження гіпокотилю (RH), кореневої шийки (RSR), первинного (R1R) та вторинних коренів (R2R). *P. melonis* уражує кореневу систему, причому найчастіше страждає коренева шийка і гіпокотиль, при цьому колонізуються епідерміс і кортекс кореня центрографічно до стебла. Спектр рослин-хазяїнів включає Cucurbitaceae, проте уражуються перець, томат, базилік і петрушка. Рослини різняться за сприйнятливістю залежно від виду і навіть сорту. Хвороботворна реакція рослин відрізняється залежно від умов вирощування (закритий або відкритий грунт), взаємодії патогена з конкуруючими мікроорганізмами та іншими еколого-трофічними зв'язками. Основним засобом боротьби є застосування довготривалих сівозмін та селекція стійких сортів. В Україні селекціоновано штам гриба антагоніста *Trichoderma viride*, який є ефективним засобом боротьби проти *P. melonis* 502. Метою нашої роботи було дослідити роль *P. melonis* у розвитку захворювань культурних рослин.

Ключові слова: *Acremonium cucurbitacearum, Plectosphaerella melonis, Nodulisporium melonis, Cucumis.*