

**Introduction.** Entomopathogenic nematodes feed on the internal tissues of the insect with the help of bacteria that live symbiotically in the intestine, develop and reproduce. The term "Entomopathogen" also means the Greek "entomos" - insect, "pathogen" disease-causing, disease-causing insect. Representatives of the Steinernematidae and Heterorhabditidae families of entomopathogenic nematodes produce pathogenicity in more than 200 types of pest insects belonging to 100 genera in symbiosis with bacteria of Xenorhabdus and Photorhabdus genera, respectively, and kill their host within 2 days.

The first entomopathogenic nematodes were described by Steiner in 1923 as *Aplectana kraussei* (now *Steinernema kraussei* ), but this information was based on very little scientific evidence.

*Neoaplectana glaseri, was later*  reported by Glaser and Fox in 1930 . Steiner includes this species in the Oxyuridae family.

Only Yaroslav Weiser isolated and described *Neoaplectana carpocapsae moth larvae in Europe.* Also, in 1955, Dutki and Hough isolated the steinernematid strain DD-136 from moth larvae in the eastern part of North America, after which serious research on the pathogenicity and development of entomopathogenic nematodes began.

In 1965, with the help of morphological and physiological studies, Poinar noted that Weiser's *S. carpocapsae Czechoslovak strain and the North American DD-136 nematode are specific.*

in 1965 Symbiosis with *S. carpocapsae*  by Poinar and Thomas associated symbiotic bacterium is described.

Later, in 1966 - 1977, Poinar and Leutenegger described the location of bacteria in nematode larvae in the infectious stage using light and electron microscopy.

Poinar and Thomas, 1967, identified the role of bacteria in nematode developmental stages and killing insect pests and reported a new bacterial family, Xenorhabdus.

The genus Heterorhabditis was first described by Poinar in 1976, and its symbiotic bacterium *Xenorhabditis luminescens* was described by Thomas and Poinar in 1979. The luminescence ability of this symbiotic bacterium was so strong that all infected insect corpses glowed in the dark.

Later, in 1903, Boemari declared this bacterial species to be a representative of the genus Photorhabdus. The mechanism of action of bacteria was clarified by Milstead in 1977.

According to genetic analyzes conducted by Kiontke in 2007, the genus Heterorhabditis is considered a close group to vertebrate parasitic nematodes, and both groups diverged independently from the freeliving *Rhabditis group.*

The mutualistic existence of representatives of the genus Heterorhabditis with a unique group of luminescent symbiotic bacteria, its ability to enter the body of healthy insects, the alternation of sexual and hermaphrodite generations, and its unique morphology explain the fact that these species are given the status of a family.

Another feature not observed in Steinernema and other rhabditids is the presence of a dorsal "hook" on the tip of the head of third instar larvae of Heterorhabditis. Bedding and Molyneux (1982) noted in their research that this structure allows the potential host to enter its body cavity through the outer tegument or trachea and intestinal walls.

According to Akhurst and Boemare's research, all Steinernema species have a symbiotic relationship with Xenorhabdus bacteria, and Heterorhabditis species have a symbiotic relationship with Photorhabdus bacteria. It was the discovery of the symbiotic relationship between entomopathogenic nematodes and bacteria that marked a turning point in the use of nematodes as commercial biological control agents.

In 1959, Dutky was the first to realize the antibiotic properties of bacteria living in symbiosis with S. carpocapsae, when he determined the role of symbiotic bacteria in the reproduction of entomopathogenic nematodes developing inside the insect corpse.

Several antibiotics, including xenorhabdins, xenocaucins, hydroxystilbenes, indole and anthraquinone derivatives, were subsequently synthesized from Xenorhabdus and Photorhabdus cultures in 2002 by Webster et al.

Primary stage larvae of entomopathogenic nematodes grow maximally and carry out antibiotic production. But the primary phase goes directly to the second phase, in which nematode growth slows down and antibody production stops. Poinar noted in 1980 that this process is a major obstacle to the commercial use of nematodes.

In turn, insects have many protective reactions against entomopathogenic nematodes, and the most important of these are melanization and encapsulation. Usually, the bacteria are able to kill the insect much faster than the insect's defense mechanism can work. However, some experimental host organisms, such as flies, perform a very rapid melanization reaction that kills the nematodes before they can activate the symbiotic bacteria. Through this research, Bronskill and Welch presented many new scientific conclusions in 1962.

Furthermore, the fact that the infective larvae of S. carpocapsae can be easily destroyed even by Galleria mellonella, the most common experimental organism used for the propagation of entomopathogenic nematodes, if they do not have symbiotic bacteria, was demonstrated in Poinar's 1969 research work. announced.

Also, Veremchuk and Issi, 1970, natural combinations of nematodes in the infectious stage, for example, unicellular and fungal representatives, also quickly destroy nematodes in the insect organism. For example, when moths primarily infected with microsporidia are infected by S. carpocapsae, they note that the microsporidia also infect nematodes.

Poinar found that naturally occurring populations of entomopathogenic nematodes can also be infected by microsporidia, whose infective larval stages are susceptible to infection by several different common soil fungi. emphasizes the need to test for suspected nematophagous fungi.

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