

***WOLBACHIA* MODIFYING IMPACT ON *DROSOPHILA*
MELANOGASTER NEURODEGENERATIVE PHENOTYPE**

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Wolbachia species are obligate intracellular maternally inherited endosymbionts, presented in over than 60% of all insect, that can cause reproductive changes in various arthropoda. However, it is known that different *Wolbachia* strains can have various effects on host ontogenesis and survivability, especially *Wolbachia* strain with WMelPop genotype can lead to degeneration in different somatic tissues of *Drosophila* flies including central nervous system. We have found presence of *Wolbachia* strains in laboratory lines of X-linked *D. melanogaster* point mutants with strong neurodegenerative phenotype. *Wolbachia* eliminated after tetracycline treatment during 2–3 generations. Comparative analysis of neurodegenerative phenotype of adult flies after bacterium elimination has shown decrease of its manifestations after tetracycline treatment against *Wolbachia*. Also it was detected increase of life-span parameters of treated lines comparing to infected flies with the same genotypes. Obtained results confirm that a manifestation of neurodegenerative phenotype formation in *Drosophila* mutant flies could be a result of *Wolbachia* modifying influence.

Keywords: Wolbachia, Drosophila, neurodegeneration, life span, tetracycline.

Wolbachia species are obligate intracellular endosymbionts that are presented in over than 60% of all insect species. *Wolbachia* is known as maternally inherited, gram-negative α -proteobacterium that can cause reproductive changes in various arthropoda [6]. *Wolbachia* infection has influence on the reproductive processes in the host organism of different arthropoda to provide optimal conditions for surviving and spreading *Wolbachia* infection [3, 19]. That is why the main number of works is focused especially on this question. However, visible self-spreading effect have not been observed in some *Wolbachia* species, so question about the possibility of its maintenance in the host organisms remains still without answer [2]. There is a suggestion that *Wolbachia* infection presence in somatic cells can increase *Wolbachia* influence on somatic processes in the host organism [1]. Recently, it has been shown that *Wolbachia* infection could increase host resistance to viral pathogens in *Drosophila* [4]. Several less described effects of *Wolbachia* infection on parental strain, such as body size and life span parameters changes are also known [2]. However the range and intensity of *Wolbachia* produced symptoms can vary in different diapason depending on the special conditions of the host organism. Some behavioral changes (courtship activity, olfactory behavior) in some *Drosophila* lines are also associated with *Wolbachia* infection. Thus, *Wolbachia* infection can provide a positive effect on its host (comparing to uninfected organisms of the same lines) promoting survival rate and spreading of intracellular bacterium in that way.

However, number of investigations conducted on *Drosophila* model detected a presence of *Wolbachia* strains with different features. For example, *Wolbachia* microorganisms with WMelPop genotype, were presented in latent form during embrional stage, however their amount

became strongly increased in adults causing an intensive degeneration in different types of tissue, including brain, retina and muscles. Significant increasing of *Wolbachia* microorganisms inside the cells, probably, could lead to the strong injuring and as a result, to death of infected flies [13]. *W. popcorn* can multiply rapidly in different organs of adult flies, causing mass destruction of tissues and premature death, whereas it is observed in lesser amount in host tissues during larval, pupal and early adult stages. Probably, it can be explained by the lack or deficiency of limiting growth-factors in the host organism during mentioned developmental stages in *Drosophila* [13].

Thus, in previous studies it was shown that different strains of *Wolbachia* might have versatile influence on the living processes in the host organism under the different conditions. It is also possible that bacterium can behave in different way depending on the sex of its host. Host individual features can be determinative for *Wolbachia* infection progress or delay in different tissues [1]. It is possible, that some lines of *D. melanogaster* have developed some special properties to keep under the control bacterium proliferation and ontogenesis or have formed less favorable conditions for *Wolbachia* development in the organism of these lines. As an example, *Huonville* and *Oregon-R Drosophila* lines can be mentioned. In the former it was detected decrease of bacterium development on the period of "egg-adult fly" and dramatic reduction of host life span whereas in the second case it was observed an arrest of *Wolbachia* development in *Oregon-R* flies with all subsequent symptoms delay [2].

Thus, the *Wolbachia* infection can be harmful for its host and, as a result, can defeat the only endosymbiotic relationships between *Wolbachia* and its host organism, because of bacterium-insect interaction can lead to the fatal outcome. Examination of laboratory strains of *D. melanogaster* commonly used in genetic experiments revealed that large percentage of them carry *Wolbachia* in a nonvirulent form, which might affect their longevity and behavior [13]. It has been notified that 30% of the strains in the Bloomington Stock Center are *Wolbachia*-infected [3]. However, future investigations of *Wolbachia* phenomenon can help in creating of a new analysis systems of *Wolbachia/Rickettsia*-host interaction.

We have focused on the mutant lines of *Drosophila* with neurodegenerative phenotype as a model for *Wolbachia* role studying. Studied *D. melanogaster* mutants are characterized by brain neuron degeneration, glial malfunction, and premature death, caused by apoptotic processes in the CNS of studied flies [9, 10, 14, 17].

Infection is one of the modifying factors that can make a significant impact on the beginning and progress of neurodegenerative diseases [8, 11]. Determining of the *Wolbachia*-free or *Wolbachia*-infected status is important for ecological work, as well as for biochemical and genetic studies of host-pathogen interaction. However, the practical goal of our investigation is to understand the role of *Wolbachia* in the degenerative phenotype development in *Drosophila* neurodegenerative mutants.

Materials and methods

Fly lines. *Drosophila* point neurodegenerative mutants from our collection were used in this work. Fly lines with well-studied phenotypes, such as *28-11*, *76-15*, *72-7*, obtained in our laboratory after chemical mutagenesis [16] and *sws¹* mutant kindly provided by Dr. Doris Kretzschmar were used in the work [9]. As a control *D. melanogaster* wild type line *Oregon-R* (Bloomington *Drosophila* Stock Centre) was used.

Ovary dissection. Ovary tissue was used for DNA extraction. Flies were fed with concentrated yeast extract 1 - 2 days prior to dissection to fatten up the ovaries. Adult females were grabbed at its lower thorax with a pair of tweezers and tugged gently at the lower abdomen with another pair of tweezers until the internal organs in the abdomen were exposed. The pair of

ovaries were detached from other organs and transferred to the ice-cold IX Phosphate Buffered Saline (PBS) for storage [18,20].

PCR assay. Determination of *Wolbachia* infection status in *D. melanogaster* strains was made by PCR amplification of *Wolbachia*-specific fragments. Ovary tissue was crushed in Phire Animal Tissue Direct PCR Kit buffer with proteinase K and heated to 60°C for 45 min, followed by 10-min incubation at 95°C to inactivate the proteinase. After centrifugation 2 µl of supernatant was used for PCR-detection. The PCR reaction was carried out with Taq DNA Polymerase (Zymo Research). A temperature profile according to primers characteristics: 95°C for 1 min, 52°C for 1 min, and 72°C for 2 min was used for 30 cycles. *Wolbachia*-specific 16S rDNA primers 5'-TTGTAGCCTGCTATGGTATAACT-3' and 5'-GAATAGGTATGATTTTCATGT-3' were used in the reaction. The following primers specific for insect mitochondrial 12S rRNA were used as control to check for the quality of each DNA extraction: 5'-AAACTAGGATTAGATACCCT-ATTAT-3' and 5'-AAGAGCGACGGGCGATGTGT-3' [15]. The PCR products were separated on the 1% agarose gel.

Tetracycline treatment. Flies carrying the bacteria were treated by the tetracycline, which eliminates *Wolbachia* [12, 15]. Flies were treated by addition of 0.25 mg/ml of tetracycline to the standard flies meal for two–three generations. *Wolbachia* infection status in tested flies was confirmed by PCR-amplification.

Life-span detection. Longevity of investigated *Drosophila* lines was tested in male flies. The survival curves were built basing on the set of experiments, each involved 100 flies in similar conditions. The lifespan was assessed by counting a percentage of died flies from the total amount of flies every 2 days. Alive flies were transferred onto the fresh meal and action was repeated after two days.

Paraffin sections. The brain sections for histological examination were prepared according to a standard technique [5]. For this purpose, flies were placed in collars and fixed with Carnoy's solution (ethanol–chloroform–acetic acid in proportion 6:3:1) for 12 hours at 4°C. Then samples were dehydrated three times in ethanol (30 min), methyl benzoate (30 min), and paraffin (twice, 60 min) to form paraffin blocks, containing only heads of flies, and 7-µm sections were made. Tissue sections were covered by DPX Fluka and covered slides. Samples were analyzed in ultraviolet light using Carl Zeiss Jena microscope, 15×40.

Statistical analysis of the results. MS Excel was used for statistical analysis package of data analysis application. Results were presented as $M \pm m$, where m – standard error. Significant level marked “*” for $P \leq 0,05$, “**” for $P \leq 0,01$, “***” for $P \leq 0,001$

Results and Discussion

Confirmation of *Wolbachia*-infection in adult flies was done in laboratory lines of *D. melanogaster*: X-linked point neurodegenerative mutants and wild type flies *Oregon-R*. Using method of PCR-detection [15], it was shown presence of studied microorganisms in laboratory lines of *sws¹*, *76-15* and *28-11*, whereas mutant lines *72-2* and wild type *Oregon-R* were *Wolbachia*-free (fig. 1, A). The absence of *Wolbachia*-related microorganisms in the lines *72-7* and *Oregon-R* can be a result of internal changes in the genomic material of these lines, or switching-on some defense mechanisms that help host organism to prevent or lose *Wolbachia* infection [7].

Investigated lines of *Drosophila* were cultivated during 2–3 generations on tetracycline-including standard meal for microorganism treatment. *Drosophila* wild-type line *Oregon-R* was used as a control to determinate antibiotic influence on the flies ontogenesis.

PCR-analysis confirmed full healing of studied lines from *Wolbachia* infection after tetracycline treatment (fig. 1, B).

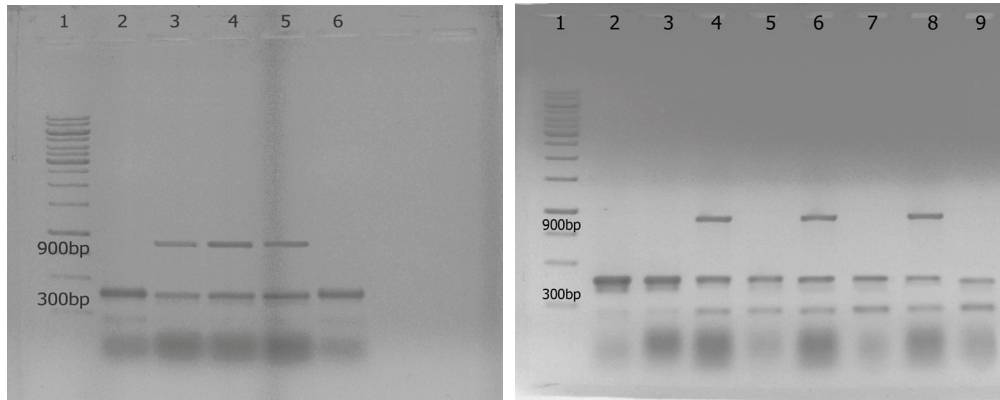


Fig. 1. PCR assay of *Wolbachia* infection using the specific primers for *Wolbachia* 16S rRNA (band of 936 bp). As a control for the quality of PCR primers to 12S rRNA were used (band of 400bp): A. Screening of *Wolbachia*-infected *Drosophila* laboratory lines with neurodegenerative phenotype: 1. DNA-marker; 2. DNA band of *Oregon-R* (wild type); 3. DNA band of *sws¹*-line; 4. DNA band of *76-15*-line; 5. DNA band of *28-11*-line; 6. DNA band of *72-7*-line. B. Confirmation of tetracycline treatment efficiency of the *Wolbachia*-infection in the infected *D. melanogaster* lines: 1. DNA-marker; 2. *Oregon-R*; 3. *Oregon-R tet⁺*; 4. *sws¹*; 5. *sws¹ tet⁺*; 6. *76-15*; 7. *76-15 tet⁺*; 8. *28-11*; 9. *28-11 tet⁺*.

We have analysed whether *Wolbachia* infection might have modification effect on phenotypic display in the brain tissue of adult flies of investigated lines with neurodegenerative disorders.

Neurodegenerative phenotype – is a complex of disorders, such as reduced life span compared to wild type flies, brain tissue vacuolization visible on paraffin sections, behavioral changes. Penetrance, expressivity and longevity of investigated lines are the main characteristics of neurodegenerative phenotype. Moreover, these parameters can be different and conservative for each of studied lines and can be used for laboratory lines description. It was described an incomplete penetrance for investigated *sws*-mutants and also for *28-11 Drosophila* point mutant [9, 10, 17].

Penetrance and expressivity of neurodegenerative phenotype of described neurodegenerative mutants *D. melanogaster* were analyzed to assess probable morphological changes in the flies brain, caused by *Wolbachia*, before and after tetracycline treatment. More than 60 males of each line were analyzed after tetracycline treatment during three generations and similar number of flies without antibiotic treatment was used as a control group. The main criteria for analysis were presence or absence of vacuoles in the brain tissue. We have not detected significant changes in the neurodegenerative phenotype of *Wolbachia* infected 20–23 days old adult flies before and after tetracycline diet (table 1). Penetrance of the neurodegenerative phenotype in *sws¹*-mutants was 53.5 ± 5.14 and $51.9 \pm 3.16\%$ without tetracycline and after tetracycline treatment, respectively. The average penetrance for *76-15* and *72-7 Drosophila sws*-mutants were 70.5 ± 3.47 vs $76.1 \pm 2.98\%$ and 83.3 ± 4.51 vs 88.1 ± 2.47 without and after treatment, respectively. Similar results were obtained for *28-11* point mutant that did not belong to the *swiss cheese* group of neurodegenerative mutants. The average penetrance of neurodegenerative phenotype was 41.4 ± 0.81 and $47.8 \pm 3.16\%$, respectively. Summarizing obtained results, it was observed slight, not significant, increase of neurodegenerative phenotype penetrance in tested flies after tetracycline diet (5–6%). Unchanged penetrance parameters can be explained, as an important evidence of mutant nature of neurodegeneration. Thus we suppose that degenerative changes in our investigated lines were

caused by point mutation in their genomes, but not by external infection agent, like *Wolbachia*.

Table 1

Comparison of neurodegenerative phenotype penetrance in *D. melanogaster* lines without and after tetracycline treatment

| Studied lines <i>D. melanogaster</i> | “tet ⁻ ” standard meal | | “tet ⁺ ” standard meal | |
|---|-----------------------------------|-----------|-----------------------------------|-----------|
| | wt, % | M, % | wt, % | M, % |
| Oregon-R | 100 | – | 100 | – |
| 76-15 | 29.5±2.86 | 70.5±3.47 | 23.9±1.1 | 76.1±2.98 |
| Sws1 | 46.5±2.74 | 53.5±5.14 | 48.1±1.4 | 51.9±3.16 |
| 28-11 | 58.6±4.18 | 41.4±0.81 | 52.2±2.33 | 47.8±3.61 |
| 72-7 | 16.7±2.14 | 83.3±4.51 | 11.2±0.92 | 88.1±2.47 |

Comment. wt – mutant flies without neurodegenerative phenotype (%); M – mutant flies with visible neurodegenerative phenotype (%).

Expressivity of phenotype can be more variable under environment influence. Therefore, analysis of expressivity changes can be very important to clarify a possible *Wolbachia* influence on degeneration intensity in studied flies. Analysis of the expressivity of neurodegenerative phenotype was done after additional observations and counting of total vacuoles number in the brain region. All investigated samples were separated for 3 groups: weak, medium and strong expression of the mutant phenotype. The main criteria of belonging to the one of three possible groups were size and number of vacuoles in the brain.

As it is visible from the figures 3 and 4, significant decrease of the neurodegenerative phenotype expressivity in 76-15, *sws¹* and 28-11 lines of *D. melanogaster* after tetracycline treatment was observed. We have not detected significant changes of expressivity in 72-7 mutant flies after tetracycline feeding (fig. 3). Strong downward tendency was detected for the number of flies with medium and strong expression of neurodegenerative phenotype based on size and total number of vacuoles in the brain of 20–21 days old initially infected *Drosophila* mutant lines.

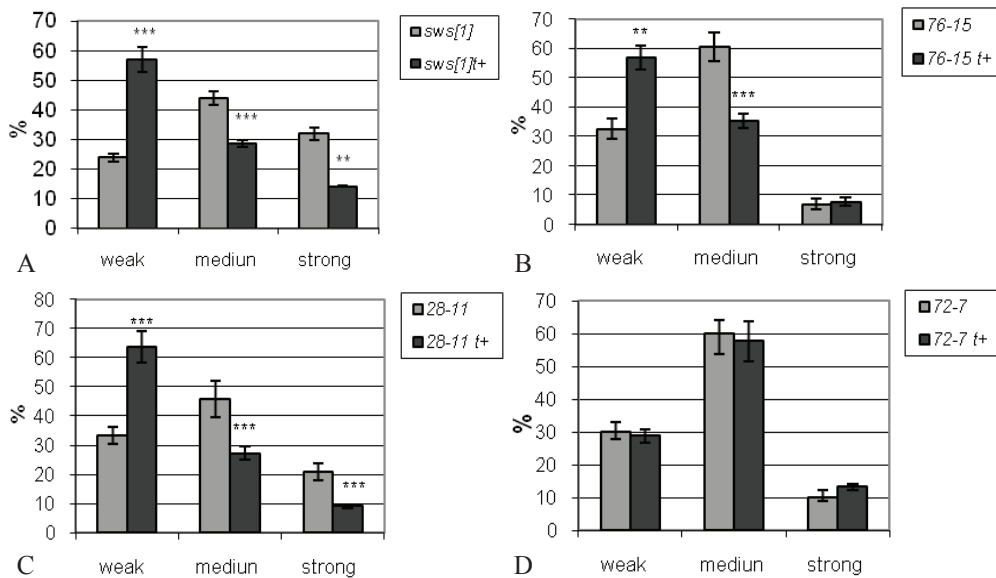


Fig. 3. Ratio of expressivity of brain tissue phenotype in 20–23-days old neurodegenerative mutants of *D. melanogaster*: A. *sws¹*; B. 76-15; C. 28-11; D. 72-7.

An absence of significant influence of tetracycline treatment on 72-7 *Wolbachia*-free mutants can be an additional evidence of *Wolbachia* modifying effect on changes of expressivity parameters in infected mutant lines. Moreover, these results confirm an absence of tetracycline influence on detected significant decrease of expression of degenerative phenotype.

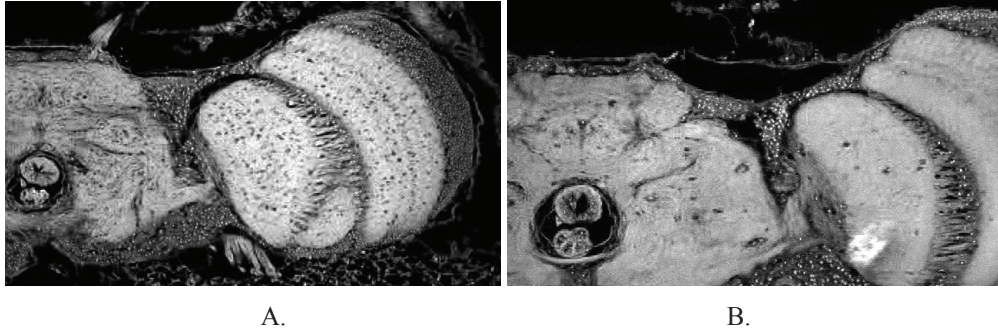


Fig. 4. Expressivity of neurodegenerative phenotype of 75-15 *Drosophila* mutant line before and after *Wolbachia* elimination: A. Strong expression of neurodegenerative phenotype of *Wolbachia*-infected flies; B. Weak expression of neurodegenerative phenotype of *Wolbachia*-free flies.

We have not detected any changes in the lifespan of *Wolbachia*-free wild type flies *Oregon-R* (fig. 5, A–C). However, a significant increase of longevity parameters in all tested mutant flies after tetracycline treatment was shown (fig. 5, A–C).

We suppose that tetracycline diet against *Wolbachia* lead to the longevity increase in the neurodegenerative mutant lines *sws¹*, 76-15 and 28-11.

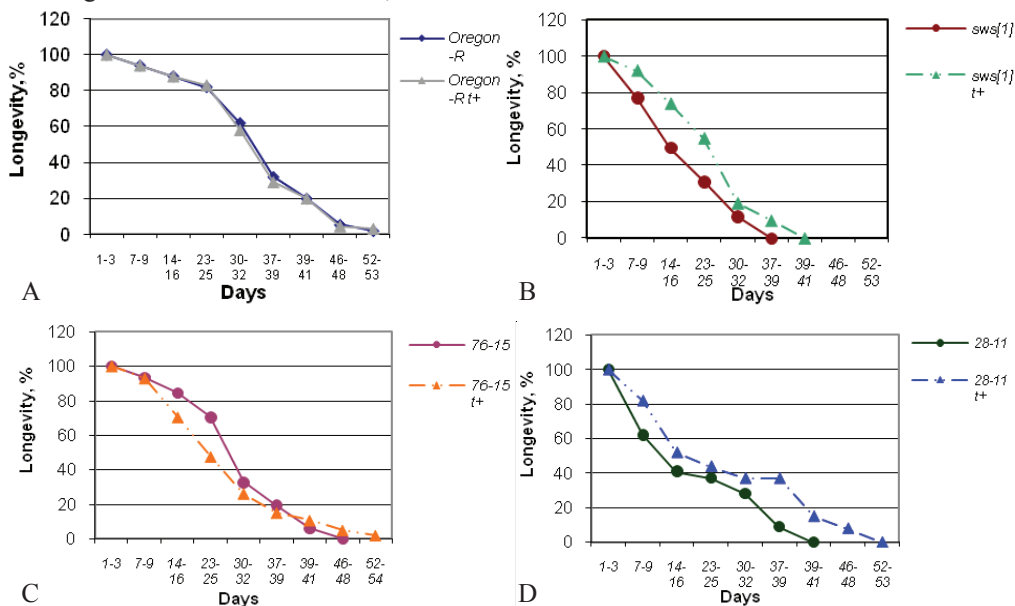


Fig. 5. Life span of *D. melanogaster* lines without tetracycline and after tetracycline treatment (t+): A. *Oregon-R* line; B. *sws¹* line; C. 76-15-line; D. 28-11-line.

Obtained results indicate that *Wolbachia* microorganisms, found in the investigated *Drosophila* imago, have not only a symbiotic effect on the host organism, but also can amplify the intensity of damage manifestation caused by mutations. As an example of this influence - the

higher expressivity of neurodegenerative phenotype in mutant flies before *Wolbachia* elimination. Bacteria can lead to the damage and destruction of various body tissues in *Drosophila*, including brain tissue due to uncontrolled increase of its abundance in the body of adult flies [2, 13].

Tetracycline treatment of studied neurodegenerative mutants infected with *Wolbachia* has a positive effect on flies, leading to treatment of *Wolbachia* infection and as a result decrease of neurodegenerative phenotype expressivity in studied *Drosophila* lines.

These data confirm that *Wolbachia* has a significant effect on neurodegenerative phenotype formation in mutant flies.

Absence of significant changes in the penetrance of the mutant phenotype suggest that pathogenic changes in the brain structure and nature of the mutation are not caused solely by the presence of bacteria in the organism of investigated mutants. However, obtained results give us evidence that presence of *Wolbachia* infection can have a modifying effect on the intensity of neurodegeneration in tested *D. melanogaster* lines. Thus, we can recommend to all researchers dealing with *Drosophila* viability and brain changes study, to consider possible *Wolbachia* influence.

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МОДИФІКУЮЧИЙ ВПЛИВ WOLBACHIA НА НЕЙРОДЕГЕНЕРАТИВНИЙ ФЕНОТИП *DROSOPHILA MELANOGASTER*

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Мікроорганізми роду *Wolbachia* – це облигатні внутрішньоклітинні ендосимбіонти у понад 60% усіх відомих видів комах, які передаються по материнській лінії та можуть спричиняти репродуктивні зміни у різних представників типу *Arthropoda*. Однак відомо, що різні штами *Wolbachia* можуть мати різний вплив на онтогенез і виживаність організму-господаря. Так, зокрема, бактерії роду *Wolbachia* з WMelPop генотипом можуть стати причиною нейродегенеративних процесів у різних тканинах дрозофіли, у тому числі нервовій тканині мозку. Нами було виявлено мікроорганізми роду *Wolbachia* у лабораторних лініях X-зчеплених нейродегенеративних мутантів *D. melanogaster*. Лікування від бактерій роду *Wolbachia* проводили шляхом личинкового згодовування тетрацикліну з їжею протягом 2–3 поколінь. Порівняльний аналіз нейродегенеративного фенотипу дорослих мух показав помітне зниження проявів

нейродегенеративного фенотипу у мутантів після вилікування від бактерії. Також було виявлено зростання тривалості життя у досліджуваних ліній *D. melanogaster* після виведення бактерії. Одержані результати можуть бути свідченням модифікуючого впливу вольбахії на формування нейродегенеративного фенотипу в мутантів *D. melanogaster*.

Ключові слова: *Wolbachia*, *Drosophila*, нейродегенерація, тривалість життя, тетрациклін.

МОДИФИЦИРУЮЩЕЕ ВЛИЯНИЕ *WOLBACHIA* НА НЕЙРОДЕГЕНЕРАТИВНЫЙ ФЕНОТИП *DROSOPHILA MELANOGASTER*

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Микроорганизмы рода *Wolbachia* – это облигатные внутриклеточные эндосимбионты в более чем 60% всех известных видов насекомых, которые передаются по материнской линии и могут вызывать репродуктивные изменения у разных представителей типа *Arthropoda*. Однако известно, что разные штаммы *Wolbachia* могут иметь различное влияние на онтогенез и выживаемость организма-хозяина. Так, в частности, бактерии рода *Wolbachia* с WMelPop генотипом могут стать причиной нейродегенеративных процессов в различных тканях дрозофилы, включая нервную ткань мозга. Нами были обнаружены микроорганизмы рода *Wolbachia* в лабораторных линиях X-сцепленных нейродегенеративных мутантов *D. melanogaster*. Лечение от бактерий рода *Wolbachia* проводили путем личиночного скармливания тетрациклина с пищей в течение 2–3 поколений. Сравнительный анализ нейродегенеративного фенотипа взрослых мух показал заметное снижение проявлений нейродегенеративного фенотипа у мутантов после излечения от бактерии. Также отмечен рост продолжительности жизни исследуемых линий *D. melanogaster* после полного излечения от микроорганизма. Полученные результаты могут быть свидетельством модифицирующего влияния вольбахии на формирование нейродегенеративного фенотипа мутантов *D. melanogaster*.

Ключевые слова: *Wolbachia*, *Drosophila*, нейродегенерація, продовжителність життя, тетрациклін.