

ся от японских перепелов. Для морфологических исследований от птиц отбирали кусочки кишечника и фиксировали в жидкости Бродского. Затем морфологический материал подвергали уплотнению путем заливки в парафин по общепринятым методикам [2]. Изготавливали гистологические срезы толщиной 3-5-7 мкм на санном МС-2 микро-томе, с последующей окраской гематоксилин-эозином.

Результаты исследований. В результате проведенных гистологических исследований установлено, что у перепелов, больных эймериозом, наблюдается острый катаральный энтерит. В 12-перстной кишке - отек и лимфоидно-эозинофильная инфильтрация слизистой оболочки, гиперемия (в некоторых случаях кровоизлияния), гиперсекреция бокаловидных желез и оголение части ворсинок. В тощей кишке - обнаружение ооцисты эймерий на разных стадиях развития. В местах скопления ооцист наблюдается частичная некротизация и лизирование слизистой оболочки эпителия, лимфоцитарная инфильтрация, эозинофилия и десквамация покровного эпителия подвздошной кишки. В слепой кишке наблюдается острое катаральное воспаление и сильная эозинофильная инфильтрация.

Заключение. При остром и хроническом течении эймериоза у перепела можно определить следующий патогистологический диагноз:

- 1) лимфоидно-эозинофильная инфильтрация, острое катаральное воспаление и гиперсекреция бокаловидных желез 12-перстной кишки;
- 2) острое катаральное воспаление и лимфоцитарная инфильтрация, а также наличие ооцист эймерий среди некротизированной и частично лизированной слизистой оболочки тощей кишки;
- 3) катаральное воспаление и эозинофильная инфильтрация слизистой оболочки слепой кишки.

Литература. 1. Биологические основы и технология выращивания перепелов: монография / А. М. Субботин, Д. Н. Федотов, М. С. Орда, М. П. Кучинский, Е. А. Жвилова. - Витебск : ВГАВМ, 2014. - 152 с. 2. Федотов, Д. Н. Гистология органов пищеварения : учебно-методическое пособие / Д. Н. Федотов. - Витебск : ВГАВМ, 2013. - 26 с.

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HISTOPATHOLOGICAL ALTERATIONS OF THE NATURALLY AEROMONAS-INFECTED SEA TROUT (*SALMOTRUTTA M. TRUTTA* L.) OF THE SLUPIA RIVER (NORTHERN POLAND)

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Introduction. Parasites invasion in fish are one of the well-recognized indicators of polluted aquatic environments [3]. Research in fish immune

system and parasite invasion mechanisms has advanced the knowledge of the mechanisms whereby parasites evade or cope with fish immune response [4]. Furunculosis induced by motile aeromonads is a problem in farming of salmonids (brown and rainbow trout) and various other fish species in Europe during last decades. Motile aeromonads cause diverse pathological conditions that include acute, chronic and covert infections [1]. Severity of disease is influenced by a number of interrelated factors, including bacterial virulence, the kind and degree of stress exerted on a population of fish, the physiological condition of the host and the degree of genetic resistance inherent within specific populations [1].

Pathological conditions attributed to members of the motile aeromonad complex may include dermal ulceration, tail or fin rot, ocular ulcerations, erythrodermatitis, haemorrhagic rot disease and scale protrusion disease. In the acute form of a motile aeromonad disease, a fatal septicæmia may occur so rapidly that fish die before they have time to develop anything but a few gross signs of disease. When clinical signs of infection are present, affected fish may show exophthalmia, reddening of the skin and an accumulation of fluid in the scale pockets. The abdomen may become distended as a result of an oedema and the scales may bristle out from the skin to give a 'washboard' appearance. The gills may haemorrhage and ulcers may develop on the dermis. Histopathologically, fish may exhibit epithelial hyperplasia in the foregut, leptomeningeal congestion in the brain, as well as a thrombosis and inflammation in the perisclerotic region and corneal epithelium of the eye [1].

Motile aeromonad septicæmias are generally mediated by stress. Elevated water temperature, a decrease in dissolved oxygen concentration, or increases in ammonia and carbon dioxide concentrations have been shown to promote stress in fish and trigger motile aeromonad infections [6]. The monitoring of environmental variables can therefore enable one to forecast stressful situations and possibly avoid problems before they arise [1].

The aim of the current study was to examine the histopathological alterations in the liver of healthy specimens of sea trout (*Salmo trutta m. trutta* L.) and naturally furunculosis-affected trout from Slupia river, the river of Baltic sea basin where trout are spawning (northern Poland, Central Pomeranian region).

Materials and methods of the research. Adult sea trout, 3-5 years of age, were collected from site on the Slupia river (Slupsk, northern Poland). The sampling for analysis from healthy males and females (control group), as well as those affected by furunculosis (study group) was collected directly after catch. After catching, microbiological tests were carried out. These tests confirmed that *Aeromonas hydrophila* complex caused furunculosis [5].

The hepatic tissue was fixed in a fresh solution of 4% paraformaldehyde (pH 7.4) at 4°C for 24 h and slides. Tissues were then placed in embedding cassettes dehydrated through graded ethanol, cleared in xylene and infiltrated with paraffin wax. Processed tissues were embedded in paraffin. Seven micron tissue sections were cut using a manual rotary microtome, stained with haematoxylin and eosin, mounted on glass slides and examined histologically. Images were obtained using a Micromed XS 5520

microscope (100/1.25x10) equipped with an eTREK DCM 320 3.0 M camera. Imaging and morphometric indices were examined using a graphical editor ImageJ (Image Processing and Data Analysis in Java).

The results of the research. There were no gender differences in the structure of the liver between healthy males and females. The liver had a tubular structure; the sections were presented in the form of balks. Veins and sinusoids between hepatic sections were noted. Hepatic balks consisted of polygonal and adjacent cells. Hepatocytes were polygonal, often 5-6-sided shape, ranging in size from 26 to 30 microns. The cytoplasm of most hepatocytes was compact, fine-grained, with finely granular basophilic inclusions. Hepatocytes in most sections had contained clear, pale stained nuclei with one to three nucleoli. Basophilic nucleus of spheric or elliptical shape with a diameter of up to 10 microns was located in the center of the hepatocytes. The chromatin in the nucleus was well structured, distributed around nuclear membrane or near well-marked with large nucleoli.

The morphological differences of liver preparations between furunculosis-affected trout and healthy individuals were observed. Hepatic parenchyma had a tubular structure with balks separated by veins and sinusoids. The balks were consisted of polygonal and adjacent hepatocytes. Intercellular spaces were greatly expanded due to edema. These alterations are more pronounced in liver of females than in males. Hepatic sinusoid and the intercellular spaces of the hepatic parenchyma were filled by erythrocytes. The cytoplasm of most hepatocytes was eosinophilic and foamy contained diffuse pockets of mild vacuolation. Basophilic nucleus had spherical or elliptical shapes with a diameter of 6 to 8 microns, located in the center of the hepatocyte. The well-structured chromatin in the nucleus was distributed around nuclear membrane or near large nucleoli. Number of nucleoli in the nucleus ranged from one to three. It should be noted decrease in the size of the nuclei in the liver of furunculosis-affected individuals. Some hepatocytes had no structural nuclei; it was represented by amorphous structureless mass. A few of hepatocytes had two nucleus after amitotic division or completed it.

Lesions in fish are associated with a variety of organisms including parasites and bacterial, viral, and fungal infectious agents. In addition, trauma, suboptimal water quality, and other abiotic stress factors may result in the loss of homeostasis [2]. The multifactorial pathways that operate at the ecological and the organism levels as well as the nonspecific response of the skin to insults make it very challenging to link epidemic skin ulcers to any single cause in natural aquatic populations [3].

Conclusion. The results of these studies provide evidence that *A. hydrophila* induce in *A. hydrophila*-infected fish appearance of a muscular haemorrhagic protuberance, which progressed into an extensive ulcerative dermatitis associated with focal haemorrhage, oedema, and dermal necrosis exposing the underlying muscle. The progression of the disease affected muscle, gills, liver, and finally the heart. Histopathological changes in *A. hydrophila*-infected liver were characterized by developed granulomatous inflammation and necrosis of hepatocytes. Multiple fibromas and macrophage granulomas were observed. The liver of infected fish showed deep vacuolisation and granulation in the cytoplasm, pyknosis of the hepatocyte nuclei, necrosis, granulomatous inflammation, and large number of

macrophages and fibroblasts. The liver exhibited focal necrosis of the hepatocytes with tubular degeneration of the intestinal microvilli and hepatocellular necrosis. The liver tissues appeared oedematous and were congested with necrotic foci showing fibrin deposition or slight haemorrhage in the pulp, inflammation, free pre-granulomatous tissue, and mature granuloma.

References. 1. Cipriano R., Austin B. 2011. *Furunculosis and other Aeromonad Diseases. In Fish Diseases and Disorders. Vol. 3, Viral, Bacterial and Fungal Infections.* Ed. by P.T.K. Woo and D.W. Bruno, 2011. P. 424-484. 2. Kane A.S., Dykstra M.J., Noga E.J., Reimschuessel R., Baya A., Driscoll C., Paerl H.W., Landsberg J. 2000. *Etiologies, observations and reporting of estuarine finfish lesions. Mar. Environ. Res., 50: 473-477.* 3. Noga E.J. 2000. *Skin ulcers in fish: Pfiesteria and other etiologies. Toxicol. Pathol., 28(6): 807-823.* 4. Sitja-Bobadilla A. 2008. *Living off a fish: a trade-off between parasites and the immune system. Fish Shellfish Immunol., 25(4): 358-372.* 5. Szewczyk E. 2005. *Bacteriological diagnostic. Warszawa, PWN (In Polish).* 6. Walters G.R., Plumb J.A. 1980. *Environmental stress and bacterial infection in channel catfish, Ictalurus punctatus Rafinesque. Journal of Fish Biology, 17: 177-185.*

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IN VITRO INHIBITION OF AEROMONAS HYDROPHILA GROWTH BY ETHANOLIC EXTRACTS OBTAINED FROM LEAVES OF VARIOUS FICUS SPECIES (MORACEAE)

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Introduction. *Aeromonas hydrophila* is an autochthonous species in freshwater environments and a member of the normal microflora in the fish intestinal tract [4]. On the other hand, *A. hydrophila* causes diverse pathologic conditions that include acute, chronic, and latent infections. Severity of disease is influenced by a number of interrelated factors, including bacterial virulence, the kind and degree of stress exerted on a population of fish, and the resistance and physiological condition of the host. Pathologic conditions attributed to members of the *A. hydrophila* complex include dermal ulceration, hemorrhagic septicemia, red sore disease, red rot disease, and scale protrusion disease [4]. In salmonids, *A. salmonicida* causes furunculosis, a disease characterized by skin ulcers and septicemia. Other *Aeromonas*