DOI: 10.29326/2304-196X-2023-12-3-253-258



# Functional morphology of digestive organs of newborn calves and pathogenesis of escherichiosis

**S. M. Suleymanov**
Voronezh State Agrarian University named after Emperor Peter the Great (VSAU), Voronezh, Russia

**SUMMARY**

Despite numerous studies, the problem of escherichiosis in newborn calves remains one of the most urgent due to the extensive spread of the disease and high mortality of young animals. This paper presents results of experimental studies carried out at the FSBSI "All-Russian Veterinary Research Institute of Pathology, Pharmacology and Therapy". The aim of the work was to conduct complex morphological studies using modern methods for a deeper understanding of escherichiosis etiology and pathogenesis. For that, pathological samples were collected from 28 1–10 day-old calves diagnosed with colibacteriosis at the initial disease stage, demonstrating pronounced clinical signs and having the terminal stage of the disease. Samples from 6 clinically healthy calves of a similar age were used as control. It was found that newborn calves at the initial stage of escherichiosis demonstrated early structural changes in the ultrastructure of mucosa cells of the rennet and small intestine, as well as in the liver parenchyma, and mild changes – in the exocrine part of the pancreas. The most profound morphofunctional changes were observed in digestive organs with apparent clinical signs of the disease. As the condition developed, the range of pathological processes expanded and involved the structural organization of the rennet, small and large intestines, liver and pancreas. At the terminal stage of escherichiosis, deep inflammatory processes occurred not only in digestive organs, but also in other systems of diseased calves. Structural changes in digestive organs had an alterative nature at the initial disease stage, whereas in case of clinically pronounced disease signs there were manifestations of catarrhal-necrotic inflammation with multiple hemorrhages in the gastrointestinal tract and parenchymal organs. Digestive organ pathology plays the leading role in formation of the clinical and morphological picture at the initial stage of escherichiosis in calves. As the disease developed, the calves demonstrated changes at molecular and subcellular levels that were detected using histochemical and ultrastructural tests.

**Keywords:** calves, escherichiosis, digestive organs, rennet, intestine, liver, pancreas, histostructure, histochemistry, ultrastructure, morphometry

**Acknowledgements:** The author expresses gratitude to the staff of the FSBSI "All-Russian Veterinary Research Institute of Pathology, Pharmacology and Therapy".

**For citation:** Suleymanov S. M. Functional morphology of digestive organs of newborn calves and pathogenesis of escherichiosis. *Veterinary Science Today*. 2023; 12 (3): 253–258. DOI: 10.29326/2304-196X-2023-12-3-253-258.

**Conflict of interest:** The author declares no conflict of interest.

**For correspondence:** Suleiman M. Suleymanov, Doctor of Science (Veterinary Medicine), Professor, Professor of the Department of Obstetrics, Anatomy and Surgery, VSAU, 394087, Russia, Voronezh, ul. Michurina, 1, *e-mail: suleimanov@list.ru.*

УДК 619:616.33-002-091:579.842.11:636.22/.28.053.2

# К функциональной морфологии органов пищеварения новорожденных телят и патогенезу эшерихиоза

**С. М. Сулейманов**
ФГБОУ ВО «Воронежский государственный аграрный университет имени императора Петра I» (ФГБОУ ВО Воронежский ГАУ), г. Воронеж, Россия

**РЕЗЮМЕ**

Несмотря на многочисленные исследования, проблема эшерихиоза новорожденных телят по-прежнему остается одной из актуальных в связи с широким распространением заболевания и высокой смертностью молодняка. В данной статье приводятся результаты экспериментальных исследований, выполненных в ФГБНУ «Всероссийский научно-исследовательский ветеринарный институт патологии, фармакологии и терапии». Целью работы было проведение с помощью современных методов комплексных морфологических исследований для более глубокого понимания вопросов этиологии и патогенеза эшерихиозов. Для этого от 28 телят в возрасте 1–10 сут с установленным диагнозом «колибактериоз» был отобран патологический материал в начальной стадии заболевания, при наличии выраженных клинических признаков и в терминальной стадии болезни. Контролем служил материал от 6 клинически здоровых телят аналогичного возраста. Установлено, что в начальной стадии развития эшерихиоза у новорожденных телят ранние структурные изменения обнаруживались в ультраструктуре клеток слизистой оболочки сычуга и тонкого кишечника, а также в паренхиме печени и слабые – в экзокринной части поджелудочной железы. Наиболее глубокие морфофункциональные изменения наблюдались в органах пищеварения при выраженных клинических признаках заболевания. В этот период развития болезни диапазон патологических процессов расширялся с охватом структурной организации сычуга, тонкого и толстого кишечника, печени и поджелудочной железы. В терминальной стадии эшерихиоза глубокие воспалительные процессы развивались не только в органах пищеварения, но и в других системах организма больных телят. Изменения структуры в органах пищеварения в начальной стадии носили альтеративный характер, тогда как при клинически выраженных признаках болезни они проявлялись в виде катарально-некротического воспаления со множественными кровоизлияниями

в желудочно-кишечном тракте и паренхиматозных органах. В формировании клинико-морфологической картины в начальной стадии развития эшерихиоза у телят ведущее место принадлежало патологии органов пищеварения. В период становления болезни в организме телят развивались изменения на молекулярном и субклеточном уровнях, которые выявлялись гистохимическими и ультраструктурными исследованиями.

**Ключевые слова:** телята, эшерихиоз, органы пищеварения, сычуг, кишечник, печень, поджелудочная железа, гистоструктура, гистохимия, ультраструктура, морфометрия

**Благодарности:** Автор выражает благодарность сотрудникам ФГБНУ «Всероссийский научно-исследовательский ветеринарный институт патологии, фармакологии и терапии».

**Для цитирования:** Сулейманов С. М. К функциональной морфологии органов пищеварения новорожденных телят и патогенезу эшерихиоза. *Ветеринария сегодня*. 2023; 12 (3): 253–258. DOI: 10.29326/2304-196X-2023-12-3-253-258.

**Конфликт интересов:** Автор заявляет об отсутствии конфликта интересов.

**Для корреспонденции:** Сулейманов Сулейман Мухитдинович, доктор ветеринарных наук, профессор, профессор кафедры акушерства, анатомии и хирургии ФГБОУ ВО Воронежский ГАУ, 394087, Россия, г. Воронеж, ул. Мичурина, 1, *e-mail: suleimanov@list.ru*.

## INTRODUCTION

Escherichiosis is common among newborn calves. The infection occurs not only due to the presence of susceptible calves in the holding, but also due to some other factors, such as hypodynamia, keeping adult and young animals together in the same pen, non-compliance with the "all-in all-out" principle, etc. In some farms, the animal disease situation is so severe that nearly all born animals get diseased, and mortality reaches 5–30% and higher [1–9]. In the literature escherichioses are known as dyspepsia, toxic dyspepsia, diarrheal syndrome of infectious etiology (*Escherichia coli*), or colibacteriosis. These diseases cause great economic losses in animal husbandry. The development of a number of pathological conditions occurring only during the neonatal period is determined not only by the age-related anatomical, physiological and immunobiological features of newborn calves, but also by the impact of new environmental conditions on them [1, 4–6, 8, 10–16].

Despite numerous studies of the *E. coli* etiological role in the development of escherichiosis in calves and introduction of mass vaccination, the disease still remains relevant [17–20]. Assessing the scientific achievements in the study of disease issues in young calves, it can be assumed that researchers have discovered many features of escherichiosis etiology and pathogenesis in newborn calves, and also proposed evidence-based recommendations for its control. Following these recommendations makes it possible to significantly reduce the losses of young cattle. However, in general, the problem of morbidity in young cattle, particularly during colostrum feeding period, is far from being solved. A serious disadvantage is that no scientific theory has been developed yet that satisfactorily explains the patterns of this pathology occurrence and development mechanisms in young animals [9, 21–23]. Although the etiological factors of mass escherichiosis of newborn calves are diverse, they are primarily associated with the activation of opportunistic pathogenic microflora, as well as with incompliances with health and hygenic standards for animal keeping and feeding [1, 8, 10, 12], and reduced resistance of young stock against the disease. The escherichiosis morbidity rate in newborn calves in some farms of the Voronezh Oblast (OOO "Voronezhpishcheprodukt") is currently 54% [13].

In this regard, the aim of the research was to conduct comprehensive morphological studies for a deeper understanding of escherichiosis etiology and pathogenesis in newborn calves.

## MATERIALS AND METHODS

The study was performed at the FSBSI "All-Russian Veterinary Research Institute of Pathology, Pharmacology and Therapy" (FSBSI "ARVRIPP&T", Voronezh). Samples of pathological material were collected from 28 1–10 day old calves diagnosed with escherichiosis at the initial stage, including those demonstrating apparent clinical signs, and those at the terminal stage of the disease. Samples from 6 clinically healthy calves of the relevant age were used as control.

All experiments were carried out in strict accordance with the European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes (ETS No. 123).

Digestive organ samples were fixed in 10.0–12.0% neutral formalin solution, in Carnoy's and Rossman's liquids for light microscopy, and in 2.5% glutaraldehyde with postfixation in 1.0% osmium tetraoxide solution for electron microscopy. Fresh organ samples were frozen in a cryostat at –20 °C for testing the enzyme activity. The samples were poured into paraffin and Epon-812, followed by the preparation of appropriate sections for light and electron microscopy.

For overview information, the sections were stained with hematoxylin-eosin and hematoxylin-picrofuxin. Histochemical methods were used for detection of the following: lipids – with Sudan III and IV and Sudan black; hemosiderin – according to Perls, mucin – with Meyer's mucicarmine; Golgi complex –according to Elftman;

neutral mucopolysaccharides and glycogen – PAS staining according to Shabadash and Bauer; acid mucopolysaccharides – by alcyan blue staining; ribonucleoproteins (RNP) – by gallocyanin chromium alum staining according to Einarson; RNA – according to Brache; DNA – according to Felgen – Rozenbeck; alkaline phosphatase activity – according to Grokk – Pierce using diazonium salts, acid phosphatase – according to Gomori as modified by Bark; esterase – according to Nahlas – Zeligman; succinate dehydrogenase – using tetrazolium method according to Nachlas, etc.

A single-beam cytophotometer was used for determination of optical density of histochemical preparations in compliance with the manufacturing standards at all stages of the procedure. MOB-1-15$^{\times}$ ocular micrometer (AO "LOMO", Russia) was used for measuring the volume of hepatocyte nuclei, the height of the villi border epithelium and the mucosa thickness.

Hepatocytes, enterocytes and secretory epithelium of the pancreatic exocrine part were studied using ultrastructural methods. For this purpose, electronograms (at least 30 photonegatives at 6,100× magnification from each group of animals) were randomly selected. The volume of cytoplasmic organelles was measured according to the basic rule of stereology [24] using a morphometric grid [25, 26]. The 36-node grid was fixed on the screen of the 5PO-1 type microfilm reader. At the same time, the area of the photonegative (6.5×9 cm) accounted for more than 100 nodes. The number of nodes in the granular (rough) and agranular (smooth) endoplasmic reticula, mitochondria, lipids and hyaloplasm was calculated. After averaging the values for each organelle, the organelle volume was calculated as a percentage of the cytoplasm volume. Confidence intervals of averages were calculated for a confidence level of 0.95.

## RESULTS AND DISCUSSION

*Initial stage of escherichiosis.* At this stage of the disease newborn calves demonstrated early structural changes in the ultrastructure of mucosa cells of the abomasum and small intestine, as well as in the liver parenchyma, and mild changes – in the pancreatic exocrine part.

The abomasum mucosa was swollen with dystrophic changes in the epithelium, and its submucosa contained blood dilated capillaries. There was also an uneven increase and depolymerization of PAS-positive material and acidic mucopolysaccharides. The activity of succinate dehydrogenase slightly decreased in the area of abomasum bottom glands. Single dystrophic mast cells were found in the own plate of mucosa. The ultrastructure of the cylindrical epithelium demonstrated dystrophy of secretory granules and membranes of the endoplasmic reticulum with low activity of nonspecific carboxylic acid esterase (Fig. 1).

Desquamative catarrh of the villi border epithelium was observed in the mucosa of the small intestine, mainly jejunum, where a decreased activity of alkaline phosphatase, succinate dehydrogenase and nonspecific carboxylic acid esterase was noted. At the same time, the activity of acid phosphatase involved in the lysis of cell breakdown products under conditions of dystrophy increased. Vacuolization of cytoplasm, an increase in the number of lysosomes and swelling of mitochondria were observed in the ultrastructure of villi enterocytes.

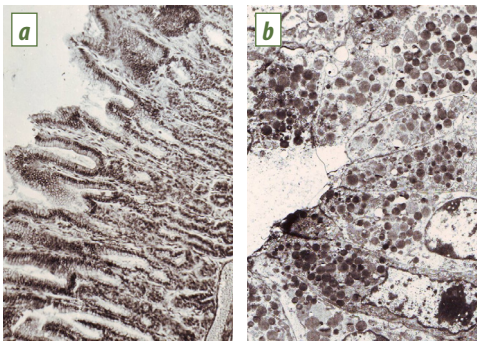


*Fig. 1. Structure of rennet mucosa at the initial stage of escherichiosis in calves:*
*a) pyloric glands of a 2-day-old calf in a state of dystrophy;*
*b) degeneration of secretory granules in fundic gland epithelium.*
*Staining with hematoxylin-eosin (a); 8× ocular magnification;*
*10× objective magnification (a); 6,100× (b)*

Small amounts of lymphocytes, histiocytes and mast cells were detected in the own plate of the mucosa villi. Many microvilli were fragmented on the brush border of enterocytes, and demonstrated moderate activity of nonspecific carboxylic acid esterase, succinate dehydrogenase and high activity of acid phosphatase. The RNP amount in the crypts decreased, there was a significant decrease in the villi cells. The number of polymorphic mitochondria with an enlightened matrix increased. The rough endoplasmic reticulum was fragmented and its total volume was 17.7%, and the volume of smooth endoplasmic reticulum increased to 47.0%. Lysosomes were found everywhere. The enterocyte nuclei became hyperchromic. Generally, low secretory activity of enterocytes was detected at the early stage of escherichiosis (Fig. 2).

A moderate amount of glycogen was detected in the liver, weakly expressed fatty and granular dystrophy of hepatocytes and decreased activity of succinate dehydrogenase and nonspecific carboxylic acid esterase were noted. Electron microscopic studies have shown that there was a decrease in the number of glycogen granules in the liver cell cytoplasm. The amount of rough endoplasmic reticulum in hepatocytes decreased to 3.4% as compared to 18.0% in normal. At the same time, the tubules of the endoplasmic reticulum were fragmented and

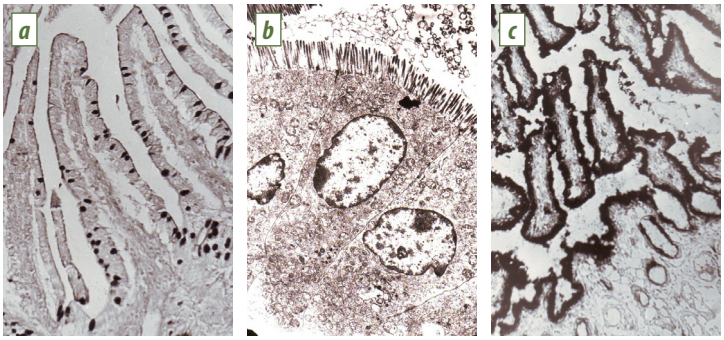


*Fig. 2. Histochemical and ultrastructural changes in small intestine mucosa at the initial stage of escherichiosis in calves:*
*a) degranulation of acid mucopolysaccharides in duodenum goblet cells;*
*b) cytoplasm vacuolization and enterocyte microvilli fragmentation;*
*c) irregular activity of succinate dehydrogenase in jejunum.*
*Staining with alcian blue (a); azo-coupling according to Grokk – Pierce (c);*
*8× ocular magnification; 10× objective magnification (a, c); 6,100× (b)*

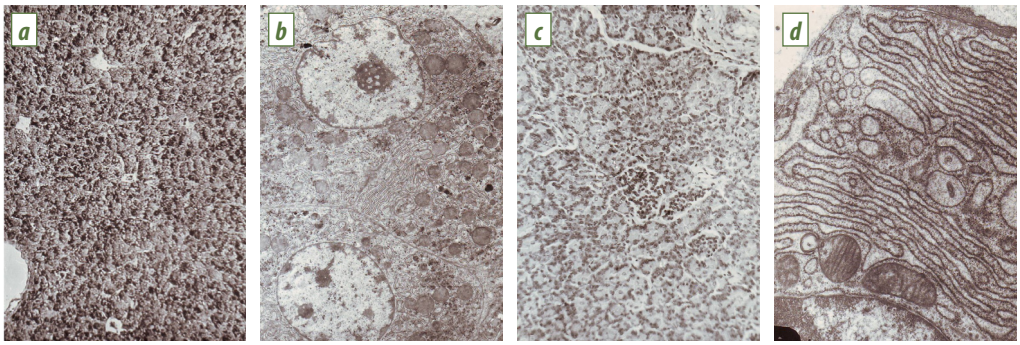


*Fig. 3. Histochemical and ultrastructural changes at the initial stage of escherichiosis in calves: a) a moderate amount of glycogen in the liver; b) hepatocyte cytoplasm vacuolization; c) dystrophy of exocrine pancreatic gland acini; d) vacuolization of the acinar cell. Periodic-Acid-Schiff (PAS) staining (a); hematoxylin-eosin staining (c); 8× ocular magnification; 10× objective magnification (a, c); 6,100× (b); 20,000× (d)*

turned into large vacuoles. The membranes lost ribosomes. Autophagic vacuoles appeared. Meanwhile, there was an increase in lipid inclusions contacting with mitochondria and endoplasmic reticulum membranes.

No noticeable changes were observed in the pancreas at the initial stage of escherichiosis in calves, but the volume of the granular endoplasmic reticulum and mitochondria in acinar cells significantly decreased, which was 10.0 and 5.0%, respectively (Fig. 3).

*In case of pronounced clinical signs of escherichiosis*, the most profound morphofunctional changes were observed in the digestive organs. During this period of disease development, the range of pathological processes expanded involving the structural organization of the abomasum, small and large intestines, liver and pancreas.

In the mucous membrane of the abomasum there were foci of epithelial cell desquamation and necrobiosis of the fundal and pyloric gland apex with a sharp decrease in the amount of neutral and acid mucopolysaccharides, nucleoprotein complex and a decrease in the activity of succinate dehydrogenase and nonspecific esterase of carboxylic acids. Conjested blood vessels and submucosa swelling were noted. This led to swelling of the abomasum mucosa, its thickness reached an average of 406.1 microns.

Uncovered villus tips, blood filling and swelling of the own plate were revealed in the small intestine in most cases. The activity of alkaline phosphatase, succinate dehydrogenase and nonspecific carboxylic acid esterase sharply decreased while the acid phosphatase activity in the area of the villi border epithelium increased. Dystrophic changes in enterocytes were accompanied by a significant decrease in the nucleoprotein number, mainly in the villi apical part. The ultrastructure of many villous cells revealed lysis of cytoplasmic organelles with nuclei pyknosis. The apical part of the terminal layer became clearer. Bundles of myofibrils were found in the sites of cell microvilli, and myelin figures and damaged mitochondria appeared in the cytoplasm.

The destruction of the beamed structure was observed in the liver, and hepatocytes were in a state of necrobiosis. Persistent fatty necrobiosis developed in the ultrastructure of hepatocytes, the number of glycogen granules decreased significantly, the membranes of the endoplasmic reticulum and mitochondria were lysed.

Pancreatic acinar cells demonstrating cytoplasm vacuolization and lysis of zymogen granules suffered greatly (Fig. 4).

*Terminal stage of escherichiosis.* At this stage, deep inflammatory processes developed not only in the digestive organs, but also in other body systems of diseased calves.

Further on, as a rule, the initial damage in the digestive organs progressed due to opportunistic pathogens in the gastrointestinal tract. The damage was characterized by profound changes in the structural organization of cells and tissues with involvement of the large intestine and pancreas in the pathological process. There was a significant decrease in the activity of hydrolytic enzymes and the content of mucopolysaccharide compounds in the gastrointestinal tract and liver. Deeply damaged membrane structures, occurrence of myelin figures, cytolysis and karyolysis were observed in the ultrastructure of abomasum, small intestine and liver mucosa cells. A large number of lipid inclusions accumulated in hepatocytes. At the terminal stage of escherichiosis, microbial intoxication caused irreversible pathological processes in the digestive organs of diseased calves, which in most cases resulted in their death.

## CONCLUSION

The digestive organs were structurally and functionally involved in the pathological process in calves at the initial disease stage, as well as those demonstrating clinically pronounced signs of escherichiosis. However, in terms of profoundness of changes and their reversibility, the digestive organs had their own peculiarities. Structural changes in digestive organs had an alterative nature at the initial disease stage, whereas in case of clinically pronounced disease signs there were manifestations of catarrhal-necrotic inflammation with multiple hemorrhages in the gastrointestinal tract and parenchymal organs.

The developing morphological changes corresponded to general pathological processes in newborn calves diseased with escherichiosis. At the same time, a complex of morphological changes characteristic of this pathology developed. The leading role in the formation of the clinical and morphological picture at the initial stage of escherichiosis in calves belonged to the pathology of the digestive organs. As the disease progressed,

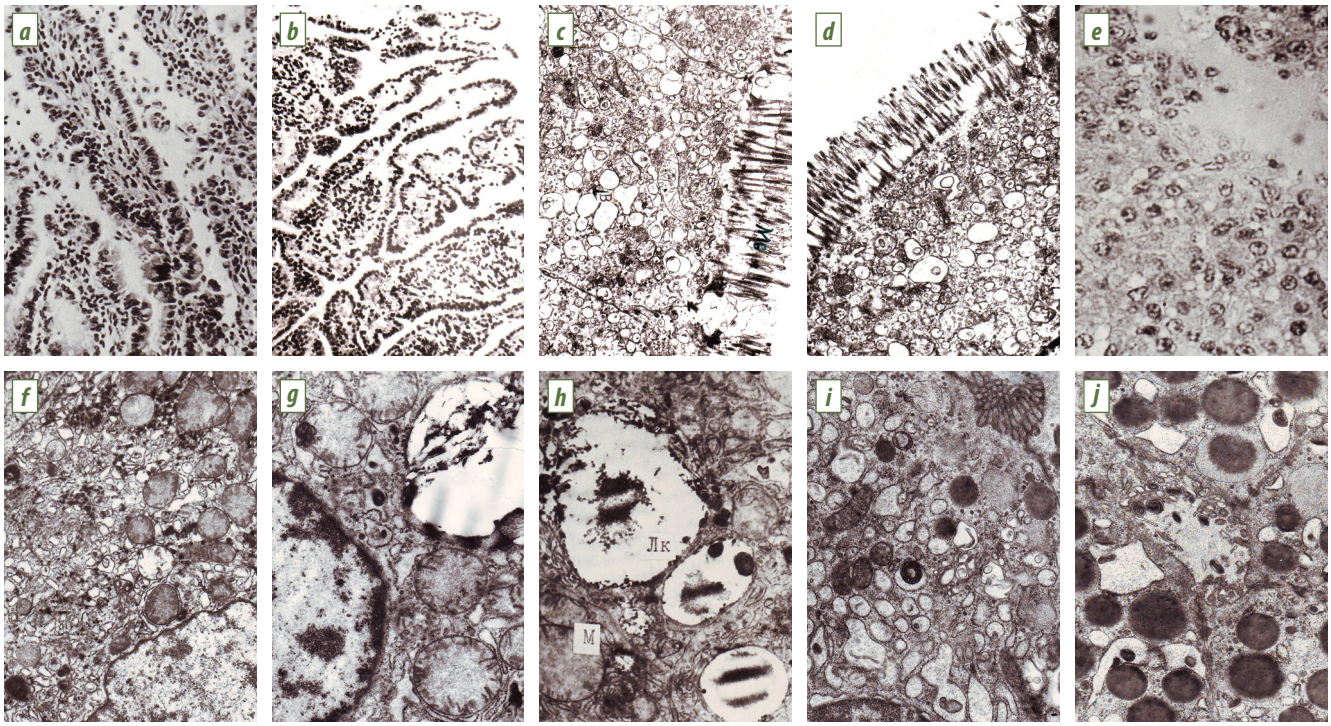


*Fig. 4. Structural changes in digestive organs of calves with pronounced clinical signs of escherichiosis:*
*a) desquamative catarrh of the small intestine border epithelium;*
*b) inflammatory-necrotic enteritis;*
*c) lysis of enterocyte cytoplasmic organelles;*
*d) enterocyte necrobiosis and microvilli lysis;*
*e) destruction of the colomnial structure with deep necrobiotic changes in the liver;*
*f) reduction of glycogen granules in hepatocyte cytoplasm and onset of dystrophic changes;*
*g) onset of hepatocyte fatty degeneration;*
*h) progression of hepatocyte fatty necrobiosis;*
*i) vacuolization and absence of zymogen granules in pancreatic acinar cell cytoplasm;*
*j) lysis of microvilli of pancreatic acinar cells and necrobiosis of zymogen granules.*
*Staining with hematoxylin-eosin (a, b, e);*
*8× ocular magnification;*
*10× objective magnification (a, b, e);*
*6,100× (c, d, f); 10,000× (g, h, i, j)*

the calves demonstrated changes at molecular and subcellular levels that were detected using histochemical and ultrastructural tests.

## REFERENCES

1. Anokhin B. M., Suleymanov S. M. Digestive organs in dyspepsia in newborn. *Veterinariya*. 1977; 5: 82–87. EDN: VNSENP. (in Russ.)

2. Dzhupina S. I. Etiologiya i profilaktika massovykh zheludochno-kishechnykh boleznei telyat = Etiology and prevention of mass gastrointestinal diseases of calves. *Veterinary Pathology*. 2003; 2: 28–30. EDN: HSOBLX. (in Russ.)

3. Zaroza V. G., Burova G. A., Burov V. G. Vozbuditeli kolibakterioza zhivotnykh i ikh laboratornaya identifikatsiya = Colibacillosis pathogens in animals and their laboratory identification. *Veterinariya sel'skokhozyaistvennykh zhivotnykh*. 2008; 3: 29–33. (in Russ.)

4. Koliakov Ia. E. Colibacillosis in calves. *Veterinariya*. 1978; 4: 48–53. PMID: 349859. (in Russ.)

5. Makarov V. V. O probleme prichinnosti infektsionnykh zabolevanii = Aspects of infectious disease causation. *Vestnik of the Russian agricultural science*. 2003; 5: 11–14. EDN: DMITSK. (in Russ.)

6. Tsion R. A. The differential diagnosis of dyspepsion, colibacteriosis and paratyphoid fever of calves. *Veterinariya*. 1966; 5: 50–53. PMID: 4975345. (in Russ.)

7. Chernukha V. K., Pustovar Ya. P. Kliniko-patomorfologicheskie izmeneniya pri toksicheskoi dispepsii teliat = Clinical and pathomorphological changes in calves with toxic dyspepsia. *Veterinariya*. 1965; 8: 104–111. (in Russ.)

8. Shaimukhametov M. A. Epizootology and therapeutic and preventive measures for escherichiosis of calves in the Republic of Bashkortostan: Author's Abstract of Thesis for degree of Candidate of Science (Biology). Ufa; 2019. 20 p. EDN: NJMMYS. (in Russ.)

9. Shishkov V. P. Pathomorphological diagnosis of toxic dyspepsia of calves. *Veterinariya*. 1964; 4: 64–66. (in Russ.)

10. Alikaev V. A. Scientific and practical achievements and problems in controlling diseases of young animals. *Veterinariya*. 1967; 11: 73–77. PMID: 5630024. (in Russ.)

11. Ivanov A. V., Matrosova L. Ye., Tremasova A. M., Titova V. Yu. Study of therapeutic efficacy of probiotic Enterosporin at colibacteriosis of calves. *Veterinary Medicine*. 2011; 95: 201–202. EDN: SMUKSV. (in Russ.)

12. Mityushin V. V. Dyspepsia of newborn calves. Moscow: Rossel'khozizdat; 1979. 111 p. (in Russ.)

13. Suleimanov S. M. Primenenie Rediara pri zheludochno-kishechnoi patologii u novorozhdennykh telyat v OOO «Voronezhpishcheprodukt» = Use of Rediar in gastrointestinal pathology in newborn calves in OOO "Voronezhpischeprodukt". *Veterinarno-sanitarnye aspekty kachestva i bezopasnosti sel'skokhozyaistvennoi produktsii:*

*materialy III Mezhdunarodnoi konferentsii po veterinarno-sanitarnoi ekspertize (15 noyabrya 2018 g.) = Veterinary and sanitary aspects of agricultural product quality and safety: proceedings of the III International Conference on Veterinary and Sanitary Expertise (November 15, 2018)*. Voronezh: Voronezh SAU; 2019; 4: 204–206. EDN: EQRFMB. (in Russ.)

14. Shachov A. G., Masjanov J. N., Sashnina L. J., Zolotarev A. I. The immune status of calves with diarrhea syndrome of infectious etiology. *Veterinary Pathology*. 2010; 1: 35–39. EDN: OBMNEF. (in Russ.)

15. Zufarov K. A., Shishova E. K., Tashkhodzhaev P. I., Baibekov I. M. Electron microscopic study of mucosa epithelium of the large intestine after resection of the jejunum. *Arkhiv Patologii*. 1970; 32 (4): 57–62. PMID: 5459109. (in Russ.)

16. Suleimanov S. M. Morphological changes in dyspepsia in calves. *Veterinariya*. 1976; 9: 69–71. PMID: 997244. (in Russ.)

17. Zufarov K. A., Baibekov I. M., Khodzhimetov A. A. Compensatory adaptive processes in the intestine. Moscow: Meditsina; 1974. 208 p. (in Russ.)

18. Zhukova S. V., Panina T. A., Vostroilova G. A., Ermakova T. I. Antimicrobic activity and clinical efficiency of a new complex antibacterial preparation at colibacteriosis of calfs. *Scientific notes Kazan Bauman State Academy of Veterinary Medicine*. 2012; 211: 71–75. EDN: PCAQZV. (in Russ.)

19. Suleimanov S. M. Morphofunctional relationship of the digestive organs and adrenal cortex in dyspepsia in calves. *Veterinariya*. 1978; 3: 91–94. PMID: 644876. (in Russ.)

20. Gebregiorgis A., Tessema T. S. Characterization of *Escherichia coli* isolated from calf diarrhea in and around Kombolcha, South Wollo, Amhara Region, Ethiopia. *Trop. Anim. Health Prod*. 2016; 48 (2): 273–281. DOI: 10.1007/s11250-015-0946-9.

21. Porokhov F. F. Prevention of diseases in young stock. *Veterinariya*. 1974; 11: 97–100. PMID: 4456757. (in Russ.)

22. Shishkov V. P. Histochemical studies of dyspepsia in newborn calves. *Veterinariya*. 1966; 4: 78–79. PMID: 6010634. (in Russ.)

23. Il'in P. A. Morphofunctional differentiation of tissues of oropharynx, esophagus and multichamber stomach of cattle in ontogenesis: Author's Abstract of Thesis for degree of Doctor of Science (Biology). Omsk; 1972. 43 p. (in Russ.)

24. Saltykov S. A. Stereometric metallography. 3rd ed., revised and supplemented. Moscow: Metallurgiya; 1970. 375 p. (in Russ.)

25. Avtandilov G. G. Morphometry in pathology. Moscow: Meditsina; 1973. 230 p. (in Russ.)

26. Weibel E. R. Stereological principles for morphometry in electron microscopic cytology. *In: International Review of Cytology. Ed. by G. H. Bourne, J. F. Danielli. Vol. 26.* New York, London: Academic Press; 1969; 235–302. DOI: 10.1016/s0074-7696(08)61637-x.

*Received 31.07.2023*
*Revised 09.08.2023*
*Accepted 02.09.2023*

## INFORMATION ABOUT THE AUTHOR / ИНФОРМАЦИЯ ОБ АВТОРЕ

**Suleiman M. Suleymanov,** Doctor of Science (Veterinary Medicine), Professor, Honored Scientist of the Russian Federation, Professor of the Department of Obstetrics, Anatomy and Surgery, VSAU, Voronezh, Russia; *https://orcid.org/0000-0002-0461-9885, e-mail: suleimanov@list.ru*.

**Сулейманов Сулейман Мухитдинович,** доктор ветеринарных наук, профессор, заслуженный деятель науки Российской Федерации, профессор кафедры акушерства, анатомии и хирургии ФГБОУ ВО Воронежский ГАУ, г. Воронеж, Россия; *https://orcid.org/0000-0002-0461-9885, e-mail: suleimanov@list.ru.*