URRENT ISSUES OF CATTLE PRODUCTION TECHNOLOGIES AND QUALITY ENSURING DEVELOPMENT IN UKRAINE (dedicated to the 100th anniversary of the National Academy of Agrarian Sciences of Ukraine foundation)

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The article highlights the results of scientific research, as well as the practical use of the achievements of scientists of the Institute of Animal Science NAAS in the fields of dairy and beef cattle breeding and genetics, biotechnology, reproduction, maintenance technologies, mechanization, an animal's feeding and nutrition standards development, as well as economical point, feed resources and livestock products quality ensuring and safety. The problems and prospects of scientific research in modern economic and climatic conditions of industry development were discussed in the article.

Institute scientists belong the priority theoretical and applied developments, which played a decisive role in the development of high-tech livestock production in the historical aspect, the new breeds and types of dairy and beef cattle creation, the modern system of large-scale breeding introduction, based on domestic competitive technologies of herd reproduction, uniform full-feeding during whole production cycle. The modern developments of the institute team influence to the strategy and methodology formation of competitive high-quality livestock products technological support in different organizational production farms.

Key words: production technologies, cattle breeding, breeds, feeding, maintenance, reproduction, products quality.

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LIPID PEROXIDATION IN THE HEART AND LIVER OF RAINBOW TROUT (*ONCORHYNCHUS MYKISS*) UPON DISINFECTION BY FORMALIN

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To evaluate the effect of formalin disinfection on the 2-thiobarbituric acid reactive substances (TBARS) aslipid peroxidation biomarker in the cardiac and hepatic tissue of rainbow trout (Oncorhynchus mykiss Walbaum) were assigned into test and control groups. The test group was exposed to formalin in final concentration 200 mL per m^3. Fish were bathed for 20 min, three times, every 3 days. Two days after the last bathing fish were sampled. The trout exposed to formalin expressed a significantly higher TBARS level in the heart by 37.2 % (p=0.020) compared to the control group. No significant differences in lipid peroxidation in the liver between control and the formalin-exposed group were found. Thus, it might be concluded that the formalin disinfection can increase oxidative stress in the heart of rainbow trout which in turn will lead to cardiac problems. Recognizing the role of biochemical changes in the tissues of formalin-exposed trout has important implications for understanding the complexity of the

physiological changes that occur during disinfection but also for improving aquaculture practices to maximize tissues growth and health of treated trout. The results of the studies are consistent with other studies that show that formaldehyde can cause oxidative stress, increasing the formation of ROS. Formaldehyde produces ROS, which causes changes in the structure of DNA, breaking DNA strands, in turn, contributes to mutagenesis and other pathological processes. It is known that non-specific immune parameters of rainbow trout after exposure to formalin undergo changes in General, in particular, there is an increase in the levels of hematocrit, leukocrit and glucose in the serum of fish exposed to formalin.

Thus, it can be concluded that disinfection with formalin can increase oxidative stress in the heart of rainbow trout, which in turn will lead to heart problems. Recognition of the role of biochemical changes in the tissues of trout exposed to formalin is important for understanding the complexity of physiological changes occurring during disinfection, as well as for improving aquaculture practices for maximum tissue growth and health of treated trout.

Keywords: **rainbow trout *Oncorhynchus mykiss*, disinfection, formalin, oxidative stress, liver, heart.**

In aquaculture, formalin (in a 1-h static bath at 200 mg per L repeated twice weekly) can be used as a disease prophylaxis regime with no negative effects on growth of juvenile rainbow trout [23]. Speare and Macnair (1996) assessed the effects of twice-weekly exposure to formalin (200 mg per L in a 1-h static bath) on juvenile rainbow trout (57.4 g initial weight) in a completely random, matched-pairs, 12-week growth trial. Growth rates, appetite, feed conversion, and body condition index of the fish were not significantly affected by formalin treatment after 6 and 12 weeks. There was no evidence of a cumulative effect of formalin treatments over time because the similarities between treated and untreated groups of fish persisted over the 12-week trial [23].

Formalin treatments are used to control fungal infections in eggs of rainbow trout *Oncorhynchus mykiss* [1]. Waterstrat and Marking (1995) evaluated the effectiveness of formalin, hydrogen peroxide and salt (NaCI) in controlling fungal infections in eggs of fall chinook salmon (*Oncorhynchustshawytscha*) under hatchery conditions. The clinical trial involved the treatment of eggs exposed to *Saprolegniaparasitica* with daily 15-min treatments of either 500 ppm or 1,000 ppm formalin. Both agents at concentrations of either 500 ppm and 1,000 ppm appeared effective in controlling infections. Hydrogen peroxide and formalin at concentrations of 500 ppm and 1,000 ppm appear to be effective alternatives to the standard hatchery practice of treating eggs with formalin at a concentration of 1,667 ppm [29]. Rach and co-workers (2005) suggested that both therapeutants were effective in increasing lake trout egg survival up to the eyed egg stage; however, formalin was the most efficient [19].

Although formalin may continue to be useful in the aquaculture industry it causes potentially harmful alterations to fish skin [21] and induces bronchial lesions [22]. It was reported that rainbow trout exposure to various concentrations of formalin affected the mucous cells resulting in increased release of mucus [4]. Blebbing of epithelial cell membranes was the first sign of the injury. Highly irregular organization of the cells followed, with regional differences occurring in different parts of fins [4]. Moreover, significant pathological changes and cell damage violence in the different formaldehyde concentration were detected [7]. Hyperplasia, epithelial disruption and necrosis cloudy swelling, hemorrhage and the accumulation of pigments in gill, necrosis in the liver parenchyma and renal tubules and degeneration as histological effect of applying of different formaldehyde concentration ranging between 50 to 500 ppm to the fry (with 6 g

average weight) of *Chanoschanos* were observed [7]. Degeneration in the epithelial cells and pilar in the gill lamellae, lymphoid infiltration, interlamellar necrosis and degeneration of the muscle tissue, dilatation in the liver, congestion in veins, degeneration in hepatocytes, damage in the blood vessels of rainbow trout treated with formaldehyde were determined by Bulut and co-workers (2015) [5].

Toxicity of formaldehyde has been attributed to its ability to form adducts with DNA and proteins [27]. Formaldehyde enters the single-carbon cycle and is incorporated as a methyl group into nucleic acids and proteins. formaldehyde reacts chemically with organic compounds (e.g., deoxyribonucleic acid, nucleosides, nucleotides, proteins, amino acids) by addition and condensation reactions, thus forming adducts and deoxyribonucleic acid-protein crosslinks [28]. It causes oxidative DNA damage in cells by increasing the production of reactive oxygen species (ROS) [6]. On the other hand, formaldehyde covalently binds with proteins to form formaldehyde-protein conjugates, which may lead to the formation of formaldehyde-specific antibodies [13].

ROS, chemically reactive molecules containing oxygen, including hydroxyl radicals, superoxides, peroxynitrites and lipid peroxyl radicals, can form as a natural by-product of the normal metabolism of oxygen and also have their crucial roles in cell homeostasis [16]. The balance between ROS production and their removal by antioxidant systems is the "redox state". Oxidative stress is defined as an excess production of ROS relative to the levels of antioxidants. When the production of ROS exceeds the capacity of antioxidant defense, oxidative stress has a harmful effect on the functional and structural integrity of biological tissue [12]. The present study aims to explore potential contributions of disinfection by formalin to the development of oxidative stress in the cardiac and hepatic tissue of rainbow trout. In this study, we sought to determine whether the profile of 2-thiobarbituric-acid-reacting substances (TBARS) in cardiac and hepatic tissues of juvenile rainbow trout changed following exposure to formalin. TBARS assay for oxidative stress was used to identify potential biomarkers in the assessment of formalin disinfection of rainbow trout.

Materials and methods. Experimental Fish. Twenty one clinically healthy rainbow trout specimens with a body mass of 45.0±2.2 g were used in the experiment. The study was carried out in a Department of Salmonid Research, Inland Fisheries Institute inRutki village, Poland. The experiment was performed at a water temperature of 16±2°C and the pH of 7.5. The dissolved oxygen level was about 12 ppm with additional oxygen supply with a water flow of 25 L/min and a photoperiod of 7 hours per day. Fish were fed with commercial pelleted diet. All enzymatic assays were carried out at Department of Zoology, Institute of Biology and Environmental Protection, Pomeranian University in Słupsk (Poland).

Experimental groups. The fish were divided into two groups and held in 250-L square tanks (70 fish per tank) supplied with the same water as during the acclimation period (2 days). Water supply to each tank was stopped on alternate days. Fish were disinfected using formalin in a final concentration of 200 mL per m^3 (Group II, n=10). The control group (Group I, n=11) was handled in the same way as a formalin-exposed group with the same water. Fish were bathed for 20 min and the procedure repeated three times every 3 days. Two days after the last bathing fish were killed and decapitated. No anesthetic agent was used before killing, decapitation and tissue sampling of specimens.

Tissue isolation. Heart and liver were excised from trout after decapitation. One specimen was used for each homogenate preparation containing a sample (10 % w/v). Hearts and livers were excised, weighted and washed in ice-cold buffer. The tissue was rinsed clear of blood with cold isolation buffer and homogenized in a glass Potter-

Elvehjemhomogenizing vessel with a motor-driven Teflon pestle on ice. The isolation buffer contained 100 mMTris-HCl; pH of 7.2 was regulated with HCl.

All enzymatic assays were carried out at 25±0.5 °C using a Specol 11 spectrophotometer (Carl Zeiss Jena, Germany). Adding the homogenate suspension started the enzymatic reactions. The specific assay conditions are presented subsequently. Each sample was analyzed in triplicate. The protein concentration in each sample was determined according to Bradford (1976) using bovine serum albumin as a standard [3].

Assay of 2-thiobarbituric acid reactive substances (TBARS). An aliquot of the homogenate was used to determine the lipid peroxidation status of the sample by measuring the concentration of 2-thiobarbituric-acid-reacting substances (TBARS), according to the method of Kamyshnikov (2004) [12]. Reaction mixture contained sample homogenate (2.1 mL, 10 % w/v) in tris-HCl buffer (100 mM, pH 7.2), 2-thiobarbituric acid (TBA; 0.8 %, 1.0 mL), and trichloracetic acid (TCA; 20 %, 1.0 mL). The total volume was kept in a water bath at 100 ^{o}C for 10 min. After cooling, the mixture was centrifuged at 3,000g for 10 min. The absorbance of the supernatant was measured at 540 nm. TBARS values were reported as nmoles malonic dialdehyde (MDA) per mg protein.

Statistical analysis. Results are expressed as mean ± S.E.M. All variables were tested for normal distribution using the Kolmogorov-Smirnov test ($P>0.05$). The significance of differences in the lipid peroxidation biomarker in the heart and liver tissue of rainbow trout between control and formalin-exposed groups (significance level at $p<0.05$) was examined using Mann-Whitney *U* test [31]. All statistical calculations were performed on separate data from each individual with STATISTICA 8.0 software (StatSoft, Krakow, Poland).

Results and discussion. The results indicate that the trout exposed to formalin expressed a significantly higher TBARS level in the heart by 37.2 % ($p=0.020$) compared to the control group. No significant differences in lipid peroxidation in the liver between control and the formalin-exposed group were found (Fig. 1).

Results of this study showed that formalin disinfection activated oxidative stress given as increased lipid peroxidation in the cardiac tissue (Fig. 1). Our results are in agreement with other research, that suggests that formaldehyde can induce oxidative stress by increasing the ROS formation [2, 9, 11, 20, 25, 32]. Formaldehyde generates ROS that induces DNA base modifications and DNA strand breakage contributes to mutagenesis and other pathological processes [32].

Moreover, excessive ROS production can cause developmental toxicity through oxidative damage to key cellular components such as DNA, proteins,and lipids [8]. Saito and co-workers (2005) using Jurkat cells, assessed oxidative stress markers such as cellular glutathione (GSH) content and cellular ROS and DNA-protein cross-links, which formed as a result of formaldehyde treatment. Cellular ROS were synergistically increased before cell death. The formation of DNA-protein cross-links was observed in the presence of formaldehyde. Co-incubation with semicarbazide, which inactivates formaldehyde, prevented this cell death induced by a combination of formaldehyde and a water-soluble radical initiator, 2,2'-azobis-[2-(2-imidazoline-2-yl)propane] dihydrochloride. Semicarbazide also exhibited an inhibitory effect on the synergistic increment of cellular ROS and the formation of DNA-protein cross-links [20].

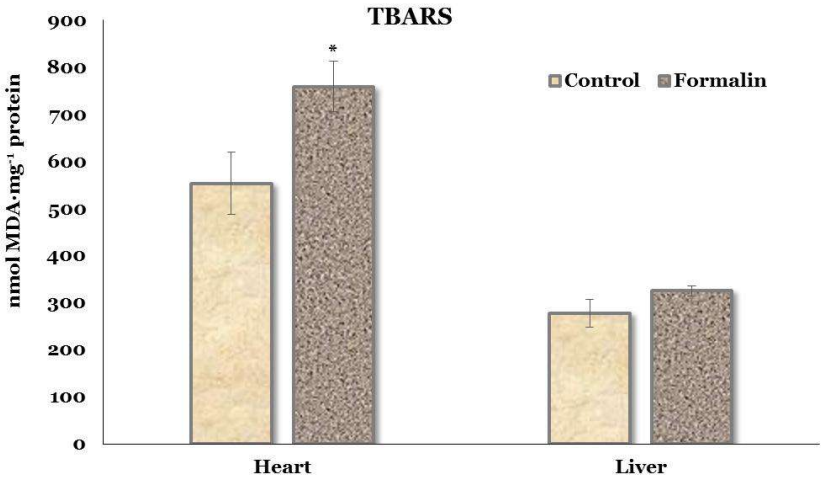

Fig. 1. Lipid peroxidation measured by the quantity of TBARS level (nmol MDA·mg^{-1} protein) in the heart and liver of rainbow trout disinfected by formalin.

Values expressed as mean ± S.E.M.
** the significant change was shown as p<0.05 when compared to untreated group values*

The biological action of formaldehyde is dose-dependent [25]. *In vitro* studies on a tumor cell and endothelial cell cultures showed that formaldehyde in the concentration of 10.0 mM caused necrotic cell death, 1.0 mM resulted in enhanced apoptosis and reduced mitotic activity while 0.5 and 0.1 mM enhanced cell proliferation and reduced apoptotic activity [25]. Among formaldehyde organic compounds N-hydroxymethyl-L-arginine, 1'-methyl ascorbigen and the formaldehyde donor resveratrol may be considered as potential inhibitors of cell proliferation. The genotoxic and carcinogenic effects of formaldehyde are due to the production of DNA-protein cross-links. Low doses of formaldehyde by reducing apoptotic activity may also accumulate cells with such cross-links [25]. Ozen and co-workers (2008) investigated formaldehyde-induced oxidative damage and apoptosis in rat tests. The activities of SOD and GPx decreased significantly, whereas the level of malondialdehyde (MDA), a lipid peroxidation product commonly used as a biomarker of oxidative damage, significantly increased in testes of male Wistar rats treated with formaldehyde. Apoptosis of spermatogenetic and Leydig cells of testicular tissues was observed [17]. MDA was significantly increased in the testicular tissues of male mice treated with formaldehyde at 20 mg per kg [26].

TBARS, a lipid peroxidation biomarkers commonly used as a biomarker of oxidative damage, were also significantly increased in the cardiac tissues of male rats exposed to formaldehyde in subacute and subchronic studies of Güleç and co-workers (2006) [9]. They evaluated the oxidant and antioxidant status as well as lipid peroxidation in the heart of rats exposed to formaldehyde inhalation for four weeks (subacute) or 13 weeks (subchronic) continuously. They revealed that subacute and subchronic formaldehyde inhalation may stimulate oxidative stress and thus, some secondary toxic effects in cardiac cells and tissue [9]. A marked formation of ROS in isolated rat hepatocytes incubating with low concentrations of formaldehyde was observed by Teng and co-workers (2001) [27]. A marked decrease in mitochondrial membrane potential and inhibition of mitochondrial respiration that was accompanied by ROS formation occurred when isolated rat hepatocytes were incubated with low concentrations of formaldehyde in a dose-dependent manner [27]. Hepatocytic GSH level was also depleted by formaldehyde in a dose-dependent manner. At higher formaldehyde concentrations,

lipid peroxidation ensued followed by cell death. Cytotoxicity was also prevented when cyclosporine or carnitine was added to prevent the opening of the mitochondrial permeability transition pore which further suggests that formaldehyde targets the mitochondria [27].

Formaldehyde may also exert these oxidative stress effects in tissues indirectly, mediated by an inflammatory response [18, 20]. The reaction of formaldehyde with amino groups of proteins is critical in inducing an immune response *in vivo* [13]. Li and co-workers (2007) studied the formation of antibodies against formaldehyde-protein conjugates in Sprague-Dawley rats for their possible use as biological markers of formaldehyde exposure. A greater response of highly specific antibody on formaldehyde with exposure period (for up to 6 months) was observed [13]. Lino dos Santos Franco and co-workers (2006) have used a pharmacological approach to study the mechanisms underlying the rat lung injury and the airway reactivity changes induced by inhalation of formaldehyde (1 % formalin solution, 90 min once a day, 4 days). Formaldehyde exposure may affect lung resident cells, including macrophages and mast cells that could mediate the lung inflammatory response and the systemic release of inflammatory mediators. The inflammatory mediators may trigger systemic immune responses [14, 15].

Yildiz and co-workers (2009) also found that non-specific immune parameters of rainbow trout after exposure to formalin have undergone alterations in general. The increase in hematocrit, leucocrit, and serum glucose levels in fish exposed to formalin was noted [30]. Im and co-workers (2006) investigated the effects of formaldehyde on rat plasma proteins. Rats were exposed to three different concentrations of formaldehyde (0, 5, 10 ppm) for 2 weeks at 6 hours per day and 5 days per week in an inhalation chamber. Level of MDA, carbonyl insertion and DNA damage in plasma, livers and in the lymphocytes of rats exposed to formaldehyde was found to be increasingly dependent of the dose. Proteins involved in apoptosis, transportation, signaling, energy metabolism, cell structure,and motility were found to be up- or down-regulated associated with formaldehyde exposure [10]. Cytotoxic effects of formaldehyde in rat lung tissues exposed to ambient air and two different concentrations of formaldehyde (0, 5, 10 ppm) for 2 weeks at 6 h per day and 5 days per week in an inhalation chamber were confirmed by Sul and co-workers (2007) [24].

Conclusions. In the present study, we demonstrated that the lipid peroxidation biomarker was significantly increased only in the cardiac tissue of formalin-disinfected group. Thus, it might be concluded that the formalin disinfection can increase oxidative stress in the heart of rainbow trout which in turn will lead to cardiac problems. Recognizing the role of biochemical changes in the tissues of formalin-exposed trout has important implications for understanding the complexity of the physiological changes that occur during disinfection but also for improving aquaculture practices to maximize tissues growth and health of treated trout.

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ПЕРЕКИСНОЕ ОКИСЛЕНИЕ ЛИПИДОВ В СЕРДЦЕ И ПЕЧЕНИ РАДУЖНОЙ ФОРЕЛИ (ONCORHYNCHUS MYKISS) ПОСЛЕ ДЕЗИНФЕКЦИОННЫХ МЕРОПРИЯТИЙ С ФОРМАЛИНОМ

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Проведена оценка перекисного окисления липидов в сердце и печени радужной форели (Oncorhynchusmykiss Walbaum) после профилактических дезинфицирующих мероприятий с формалином. Радужная форель была разделена на две группы (контрольную и опытную). Рыбу опытной группы (n=10) дезинфицировали формалином в концентрации 200 мл на 1 м^3. Погружение рыбы в раствор формалина длилось 20 минут; процедуру повторяли трижды каждые 3 дня. Через два дня после последней процедуры рыбы были отобраны из бассейнов для дальнейших исследований. Печень и сердце были выделены после декапитации рыб. Как показали результаты наших исследований, действие формалина как дезинфицирующего агента существенно не влияет на интенсивность липопероксидации в печеночной ткани и вызывает увеличение содержания ТБК-продуктов в сердце радужной форели (на 37 %, p=0,020). Результаты исследований согласуются с другими исследованиями, которые показывают, что формальдегид может вызывать окислительный стресс, увеличивая образование АФК. Формальдегид производит АФК, которая вызывает изменения структуры ДНК, разрыв нитей ДНК, в свою очередь, способствует мутагенезу и другим патологическим процессам. Известно, что неспецифические иммунные параметры радужной форели после воздействия формалина претерпевают изменения в целом, в частности, отмечается повышение уровней гематокрита, лейкокрита и глюкозы в сыворотке крови у рыб, подвергшихся воздействию формалина.

Таким образом, дезинфекция формалином может усилить окислительный стресс в сердце радужной форели, что в свою очередь приведет к проблемам с сердцем. Признание роли биохимических изменений в тканях форели, подвергающейся воздействию формалина, имеет важное значение для понимания сложности физиологических изменений, происходящих во время дезинфекции, а также для улучшения практики аквакультуры для максимального роста тканей и здоровья обработанной форели.

Ключевые слова: радужная форель Oncorhynchus mykiss, дезинфекция, формалин, окислительный стресс, печень, сердце

ПЕРЕКИСНЕ ОКИСНЕННЯ ЛІПІДІВ У СЕРЦІ І ПЕЧІНЦІ РАЙДУЖНОЇ ФОРЕЛІ (ONCORHYNCHUS MYKISS) ПІСЛЯ ДЕЗИНФЕКЦІЙНИХ ЗАХОДІВ З ФОРМАЛІНОМ

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Грудневська Й., Відділ досліджень лососевих риб, Інститут прісноводного рибного господарства ім. Станіслава Саковича, Рутки, Жуково, Польща.

Проведено оцінку перекисного окиснення ліпідів в серці і печінці райдужної форелі (Oncorhynchus mykiss Walbaum) після профілактичних дезінфекційних заходів з формаліном. Райдужна форель була розділена на дві групи (контрольну і дослідну). Рибу дослідної групи (n=10) дезинфікували формаліном в концентрації 200 мл на 1 м^3. Занурення риби в розчин формаліну тривало 20 хвилин; процедуру повторювали тричі кожні 3 дні. За два дні після останньої процедури риби були

відібрані з басейнів для подальших досліджень. Печінка і серце були виділені після декапітації риб. Як показали результати досліджень, дія формаліну як дезинфікуючого агента суттєво не впливає на інтенсивність ліпопероксидації в печінці і викликає збільшення вмісту ТБК-продуктів в серці райдужної форелі (на 37 %, p=0,020). Результати досліджень узгоджуються з іншими дослідженнями, які показують, що формальдегід може викликати окислювальний стрес, збільшуючи утворення АФК. Формальдегід виробляє АФК, яка викликає зміни структури ДНК, розрив ниток ДНК, в свою чергу, сприяє мутагенезу та інших патологічних процесів. Відомо, що неспецифічні імунні параметри райдужної форелі після дії формаліну зазнають зміни в цілому, зокрема, відзначається підвищення рівнів гематокриту, лейкокриту і глюкози в сироватці крові у риб, які зазнали впливу формаліну.

Таким чином, дезінфекція формаліном може посилити окислювальний стрес в серці райдужної форелі, що в свою чергу призведе до проблем із серцем. Визнання ролі біохімічних змін в тканинах форелі, що піддається впливу формаліну, має важливе значення для розуміння складності фізіологічних змін, що відбуваються під час дезінфекції, а також для вдосконалення практики аквакультури для максимального зростання тканин і здоров'я обробленої форелі.

Ключові слова: райдужна форель Oncorhynchus mykiss, дезинфекція, формалін, окиснювальний стрес, печінка, серце.

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ВІКОВА ДИНАМІКА ЯКОСТІ М'ЯСА МОЛОДНЯКУ КРОЛІВ

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Кролівництво – одна з найприбутковіших галузей тваринництва, яка відіграє значну роль в забезпеченні людства продовольством та хутровими виробами.Завдання кролівництва як галузі полягає у розведенні кролів для отримання цінного хутра, пуху та м'яса.

М'ясо кроля, як дієтичний продукт, використовується у харчуванні людей будь-якого віку. Воно соковите, нежирне, містить значну кількість повноцінного білку та дуже мало холестерину, дрібно волокнисте, відрізняється високою перетравністю та не має протипоказань для вживання при різних захворюваннях.

Несприятливі екологічні зміни в природі обумовили необхідність використання у годівлі екологічно чистих мінеральних компонентів і природних мінералів, визначення норм потреби та введення їх у раціон.

У статті наведено результати досліджень із визначення у м'ясі кролів 30 до 150-добового віку м'ясо-шкуркового напряму продуктивності за використання комбінованого типу годівлі хімічних (вологість, суха речовина), фізико-технологічних (вологоутримуюча здатність, площа плями, кислотність) властивостей м'яса та найдовшого м'язу спини, калорійної і біологічної цінності. Вста-

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