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INTERNAL LIPIDS AND THEIR FATTY ACIDS COMPOSITION IN A SHEEP WOOL FIBER UNDER BIODESTRUCTION WITH FLEECE MICROORGANISMS

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Microbiological destruction of fibers is a common damage to sheep's wool. Considering the defining role of internal lipids in the formation of wool fibers surface the aim of the work was to study the structure and lipid composition of the normal and damaged wool. The research was carried out on ewes of the Askanian fine-wool breed. The content of microorganisms was estimated after sowing on dense nutrient environments. Wool fibers surface was studied by scanning electron microscopy, the content of internal lipids by thin layer chromatography after preliminary alkaline hydrolysis of the fiber, and fatty acids composition by gas-liquid chromatography. Biodestructed wool was shown to contain almost three times more bacteria, as well as higher levels of actinomycetes and mushrooms compared to intact wool. The violation of the cuticular layer was detected as the result of the fleece microflora activity. In a defective wool the content of the free internal lipids and non-esterified fatty acids was increased while the content of protein-bound lipids and esterified cholesterol as well as of ceramides was decreased as compared to normal wool. The level of 18-methyleicosanoic acid in the protein-bound lipids of damaged wool was decreased, indicating the destruction of the thioester bonds by which structural lipids are covalently linked to proteins through 18-methyleicosanoic acid.

K e y w o r d s: sheep's wool fiber, biodestruction, microorganisms, internal lipids, protein-bound lipids, fatty acids, 18-methyleicosanoic acid.

Sheep wool is an ideal environment for the development of various types of microorganisms, since the simultaneous presence of air, heat and free moisture is a prerequisite for their vital activity. In the process of its vital activity, the fleece microflora uses both its environment, i.e. grease-sweat, and keratin itself as a substrate, which ultimately leads to damage to its structure, and in some cases to complete degradation of the fiber. Actually, microbiological destruction of fibers is the most widespread type of damage to woolen raw materials [1, 2].

The species composition of the microflora of the fleece is unstable and is mainly represented by species characteristic of soil, manure, and decomposing plant residues. Damage to wool by microorganisms can continue in the future when it is stored under unfavorable conditions [3, 4].

On the surface of the wool fiber, there are always specific bacteria that are peculiar only to this fiber – epiphytic microflora. Representatives of these bacteria secrete proteolytic enzymes (mainly pepsin) capable of hydrolyzing keratin to individual amino acids [5]. First, the cuticular layer is destroyed, and then the cortical layer. Biodestruction of the cuticle takes place in three stages: the first is the erosion of the fiber surface, the second is the destruction of the intercellular substance, and the third is the detachment of cortical cells. As a result of the violation of the structure of the fibers of the scales and the cells of the cortical layer, they are separated from each other and the fiber disintegrates [6].

In addition to disrupting the fiber structure, some bacteria and mushrooms reduce its quality by dyeing the wool red, blue, dirty green, and sometimes yellow, which cannot be washed off with water and detergents [7].

The biodestruction of wool is caused by both bacteria and mushrooms [8]. However, the latter play a dual role in the biodestruction of wool: firstly, using grease-sweat as a nutrient medium, they create conditions for further bacterial activity; secondly, some mushrooms, due to the growth of mycelial hyphae, can mechanically destroy the fiber structure [9].

The quality of wool largely depends on the amount and quality of wool fat (wax), because it, covering the fibers with a thin layer, contributes to the formation of staples and braids, and therefore the fleece as a whole. Under the influence of environmental factors, hydrolytic, oxidizing, and microbiological processes occur in grease-sweat, which lead to an increase in sweat pH. The higher the alkalinity of the sweat, the more oxidants it contains, the more intense the process of wax destruction. The protective function of wax and its resistance to the action of negative external factors depends on the composition and ratio of its components. Grease-sweat has the best protective properties, in which there is less than one unit of sweat per unit of wax [10, 11].

Wool fiber is a morphologically complex formation consisting of different types of chemical substances. 95% of wool fiber is made of keratin, the remaining 5% are so-called minor components, such as lipids, pigments, mineral elements, water [12, 13].

The hair structure contains a small amount (up to 3%) of lipids, which are both free and bound to protein. Since these lipids are an integral component of the fiber and most of them cannot be isolated by organic solvents without preliminary alkaline hydrolysis, they are called structural, internal or integral [14].

Hair lipids are localized not only in the cuticle, but are also components of cell-membrane complexes (CMCs) that connect the cells of the cuticle and cortex [15]. It is believed that internal lipids, which are localized in cell-membrane complexes, make up about 1.5-2% of the mass of fibers, but some amount of these lipids can exist as a component of the remnants of cell nuclei or in the remnants of cell organelles, such as mitochondria [16].

Internal lipids of fibers differ significantly from surface lipids (wax) and skin lipids, as they do not originate from sebaceous glands. They consist of cholesterol, fatty acids, cholesterol sulfate, and ceramides similar to those found in the stratum corneum of the epidermis [17].

Fatty acids in keratinized fibers are bound by thioester bonds to the proteins of the outer part of the cuticle. Special attention is paid to studies of 18-methyleicosanoic acid, which is part of the hydrophobic lipid surface of hair and covalently binds structural lipids to proteins [18]. As shown, internal lipids have a decisive influence on the formation of the surface properties of hair and its ability to protect against external negative factors [19]. They have the ability to change the structure of the fiber, reducing the moisture content and permeability, and increasing its tensile strength [20].

Materials and Methods

The object of research was the wool of ewes of the Askanian fine-wool breed of sheep belonging to the Institute of Livestock Breeding of the Steppe Regions named after M.F. Ivanova “Askania Nova”. All animals were kept and fed under the same conditions. Wool samples taken from the fleece after the spring shearing of sheep were used for research. The selected samples were divided into wool that was normal in condition and wool that had visual signs of damage and defects. Confirmation of damage or non-damage of the wool was carried out using scanning electron microscopy. After that, microorganisms were isolated from all samples. Samples of destructive wool, which had the greatest microbiological insemination, were selected for further research. Samples of undamaged wool with a low level of microbial contamination served as controls.

Study of the surface of wool fibers. The surface of wool fibers was examined using a scanning electron microscope JEOL JSM-T220A (Japan). For this, the surface of the hair was metallized by thermal spraying of a thin layer of copper (up to 10 nm). Spraying was carried out in a VUP-5 sprayer. A magnification of ×350 was used to photograph the surface of the samples, and the accelerating voltage was 20 kV.

Microbiological studies. To desorb microorganisms, 1 g of wool was ground for 5 min in a sterile porcelain mortar, adding 1 ml of a 0.3% Tween-80 solution [21]. Then the wool sample was quantitatively transferred into a sterile flask, using 99 ml of sterile water for repeated washings. Flasks with samples were shaken on a Schüttel apparatus for 30 min, after which the necessary dilutions of the bacterial suspension were prepared. The number of viable microorganisms was determined by sowing appropriate dilutions on dense nutrient media: meat-peptone agar – for bacteria, Saburo’s – for mushrooms, neurospores and molds, Chapek’s – for actinomycetes.

Colonies were counted after 4–5 days of incubation in Petri dishes at 30°C [22].

Studies of wax and sweat. In order to obtain wool fat (wax), wool was extracted in a Soxhlet extractor with carbon tetrachloride for 5 h. The total amount of wax was determined by the weight method, and its lipid composition was determined by thin-layer chromatography in the petroleum ether/diethyl ether (4:1, v/v) system using Sorbfil plates. Identification of individual classes of lipids was carried out by comparing the chromatogram of the studied samples with the chromatogram on which the witnesses were applied.

The amount of sweat was determined by extracting its salts with water, and the concentration of hydrogen ions (pH) in the extract was determined using a universal EV-74 ionometer [23].

Obtaining free and bound internal lipids. To obtain free internal lipids, after extraction of surface lipids, wool samples were re-extracted with a mixture of chloroform/methanol (2:1) in a Soxhlet extractor for 5 h. The lipid extract was dried under a vacuum and weighed. Alkaline saponification technique was used to obtain bound structural lipids [24]. Wool samples that remained after removal of free internal lipids were hydrolyzed by two-hour treatment at 60°C in 100 ml of a 1 M solution of sodium hydroxide in 90% methanol. The samples were cooled and transferred to separatory funnels. 100 ml of chloroform and 25 ml of distilled water were added to each sample. After 12 h, the lower layer of chloroform was removed, and the upper phase was acidified with a 6 M hydrochloric acid solution and re-extracted by mixing with 100 ml of chloroform. After settling, the lower layer of chloroform was removed, added to the previously obtained extract and dried by evaporation. The resulting sediment was dissolved in 10 ml of a chloroform–methanol mixture (2:1), and 3 ml of 7.5% potassium chloride was added to each sample. After 24 h, the upper phase was removed using a water jet pump, and the lower phase, which contained lipids, was used for research.

The total amount of internal lipids was determined by the weight method, and their composition was determined by the method of thin-layer chromatography. For this, we used Sorbfil plates measuring 10×10 cm with a working layer of fractionated wide-pore silica gel with a particle size of 90-120 microns, the thickness of the sorbent layer on one plate is ±5 microns. Lipids were separated in two systems: petroleum ether/diethyl ether, 4:1, v/v, (system A) and chloroform/methanol/water, 65:25:4, v/v (system B). After drying, the chromatograms were sprayed with 50% sulfuric acid and charred at 105°C. For the quantitative determination of lipids, spots were scraped from the plates into test tubes, where concentrated sulfuric acid was added and this mixture was heated to a temperature of 105°C. The optical density of each fraction was measured on a spectrophotometer at a wavelength of 400 nm in a cuvette with a thickness of 1 cm. Individual classes of lipids were identified by comparing the chromatograms of the samples with the chromatogram on which individual witnesses – cholesterol, stearic acid, lanosterol (Sigma Chemical Co., USA) were applied [25]. Other classes of lipids were compared with rf-values of classes of lipids according to literature data [26]. The content of individual lipid components was calculated mathematically and expressed as a percentage.

Determination of fatty acid composition. To determine the fatty acid composition of internal lipids, they were converted into methyl esters by direct transesterification of fatty acids [27]. The separation of methyl esters of fatty acids was carried out on a gas-liquid chromatograph Chrom-4 (Czech Republic) under the following conditions: a metal column 240 cm long and 3 mm in diameter was filled with Chromosorb 60-80 mesh, coated with 15% polyethylene glycol succinate, the temperature of the evaporator was 240°C, column thermostat – 190°C, carrier gas (nitrogen) consumption – 25 ml/min, air – 400 ml/min. For the identification of fatty acids, standard mixtures of the company Supelco were used, and the tr-Ir of all its components was measured, and the proportion of individual fatty acids was calculated using the formulas.

All the reagents used were of a qualification not lower than chemically pure. The obtained data were processed using the program Excel. The results of the research were processed statistically using the arithmetic mean and standard error ($M \pm m$) and the credible interval for assessing the degree of probability (P) using the Student's t-test. Differences were considered statistically significant at $P < 0.05$.

All procedures involving animals were conducted in accordance with the Guiding Principles for the Care and Use of the Research Animals.

Results and Discussion

According to the results of scanning electron microscopy (Fig. 1, *A*), it was established that the

cuticular, that is, the surface layer of the wool fiber, is made of keratinized scales, which are arranged in a tile-like manner from the root to the tip of the hair. Such a surface, formed with the help of the protruding edges of the cells, ensures better adhesion between them and thus protects the main part of the hair, i.e. its cortex, from the negative effects of physical and chemical factors.

From the data in Fig. 1, *B*, it can be seen that damage to the hair is manifested in destructive changes in its surface. In particular, it can be seen that in wool that has undergone biodestructive changes, the edges of the scales are not clear and almost completely smoothed, the integrity of the cuticular layer is violated, individual scales are peeled off in places, and their surface is deformed. At the same time, the cells of the cuticle of the damaged hair bend accordingly and tear unevenly. As a result, the surface of the fibers becomes even more exposed. This allows micro-organisms that are on the surface of the wool to penetrate into the deeper layers of the hair, namely into the CMC, which bind the cells of the cuticle and the cortex together, and cause their destruction.

When studying the species composition of the microflora of the fleece, it was established (Table 1) that damaged wool contains almost three times more bacteria ($P < 0.001$) compared to wool in normal condition. Damaged wool is also characterized by a probably higher content of actinomycetes ($P < 0.01$) and mushrooms ($P < 0.01$). As for mold fungi and neurospores, the likely growth of these microorganisms in damaged wool has not been recorded.

We will remind you that the fleece microflora can use wool fat as a substrate for its growth and development. In view of this, the data obtained by us during the study of grease-sweat are of great importance. In particular, it was established (Table 2) that the increase of microorganisms in damaged wool is accompanied by a probable *(*$P < 0.001$) decrease, almost by half, of wax. Instead, the amount of sweat probably increases ($P < 0.001$), as does its alkalinity ($P < 0.05$). By the way, it is known that more active development of microorganisms occurs in an alkaline environment than in an acidic one.

Highly alkaline sweat negatively affects the quality of wool, as it can lead to destructive changes in the structure of the fiber itself. Instead, the quality of wool fibers largely depends on the content of wax and, especially, its lipid composition, since wax performs a protective function. With this in mind, the wax-to-sweat ratio is an extremely important indicator that characterizes the protective properties of grease-sweat. From the data in Table 2, it can be seen that this indicator is significantly worse in damaged wool compared to wool in normal condition.

The protective property of wax is determined by its specific composition, the quality of which depends on the optimal ratio between individual classes of lipids [28]. From the data in Table 2, it can be seen that against the background of almost

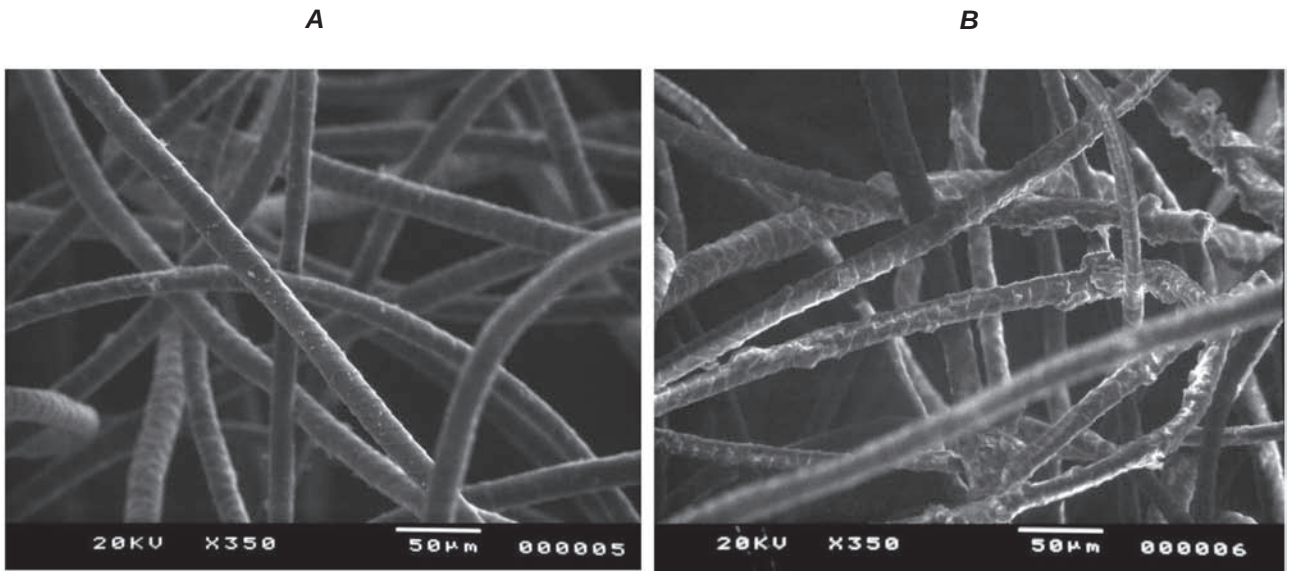

*Fig. 1. Image of the outer surface of a normal (**A**) and damaged (**B**) wool fiber*

Table 1. Quantitative and species composition of microorganisms of normal and damaged wool, CFU/g (M ± m, n = 5)

Microorganisms	Wool	
	Normal	Damaged
Bacteria $\times 10^9$	2.60 ± 0.23	7.20 ± 0.59***
Actinomycetes $\times 10^5$	2.20 ± 0.36	4.40 ± 0.23**
Mushrooms $\times 10^5$	4.20 ± 0.29	6.60 ± 0.17*
Fungi $\times 10^4$	4.80 ± 0.21	5.40 ± 0.23
Neurospores $\times 10^3$	3.60 ± 0.23	4.60 ± 0.80

Note: here and in the future statistically significant differences *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

Table 2. Content and composition of grease-sweat of normal and damaged wool (M ± m, n = 5)

Indicator	Wool	
	Normal	Damaged
Amount of wax, %	15.97 ± 0.77	9.09 ± 0.28***
Composition of wax, %:		
– polar lipids	18.57 ± 1.03	23.25 ± 0.63*
– non-esterified cholesterol	10.45 ± 0.95	10.30 ± 0.33
– lanosterol	8.23 ± 0.49	7.81 ± 0.48
– non-esterified fatty acids	5.67 ± 0.40	6.88 ± 0.23*
– dehydrocholesterol	9.45 ± 0.36	9.85 ± 0.80
–squalene	8.50 ± 0.05	7.96 ± 0.43
– esterified cholesterol	39.13 ± 1.23	33.96 ± 1.08*
Amount of sweat, %	15.39 ± 0,22	19.72 ± 0.39***
pH of sweat	8.35 ± 0.23	9.31 ± 0.26*
Wax : sweat ratio	1.00 : 0.96	1.00 : 2.17

the same amount of non-esterified cholesterol, lanosterol, dehydrocholesterol and squalene, the content of polar lipids ($P < 0.05$) and non-esterified fatty acids ($P < 0.05$) probably increases in damaged wool. As for the fraction of esterified cholesterol, its amount probably decreases in defective wool ($P < 0.05$). With this in mind, let us recall that certain types of microorganisms, possessing lipolytic activity, can hydrolyze wax lipids to non-esterified fatty acids and aldehydes [29].

Therefore, it follows from the obtained data that the increase of certain types of microorganisms in damaged wool is accompanied by the hydrolysis of some components of the wax and an increase in the alkalinity of sweat, which leads to the deterioration of the protective properties of the wax. As a result, favorable conditions are created for their influence on the very structure of wool fibers and, above all, on the cuticular layer, which ultimately leads to its damage.

As it is known, two types of structural lipids, free and bound, can be isolated from wool fiber. About 1% of them can be isolated without preliminary alkaline hydrolysis, these are the so-called free internal lipids [30]. Bound lipids are isolated only by alkaline hydrolysis, and their amount can reach 1.5-2% [31]. As a result of the conducted research, it was established (Fig. 2) that the total amount of free lipids in normal wool is 0.95%. On the other hand, in pathologically changed fibers, this indicator of changes is probably higher and amounts to 1.23% ($P < 0.05$). For bound internal lipids, the diametrically opposite picture is observed here. In normal wool, the amount of these lipids is 1.56%, and in damaged

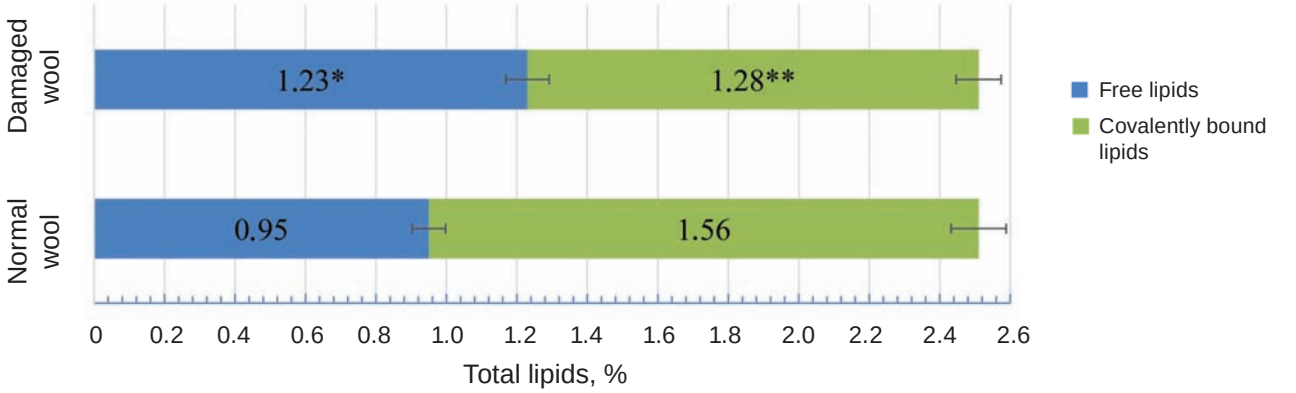

Fig. 2. Content of total internal lipids in normal and damaged wool

fibers, their amount probably decreases ($P < 0.01$). The increase in the total number of unbound internal lipids in defective wool obviously occurs due to the reduction of bound lipids that have lost their connection with the protein component of the fiber and have moved into a free state.

This distribution of total lipids is related to the structure of the fiber. In particular, free lipids are localized mainly in the cuticle, which is most negatively affected due to fiber biodestruction.

Wool damage processes are accompanied by hydrolysis of its lipid components, which is indicated by an increase in the fraction of non-esterified fatty acids and a decrease in esterified cholesterol in defective wool. Moreover, this applies to both free and bound forms of lipids (Fig. 3).

In this regard, only the mechanism of increasing cholesterol sulfate ($P < 0.01$) in the composition of bound lipids is unclear (Fig. 4). However, we remind you that this component of hair is an important component in maintaining the integrity of the fiber, that is, it serves as an intercellular "cement" in keratinized tissues. Cholesterol sulfate, which is contained in the lipids of cell membranes of wool and other keratin fibers, is an additional component with amphipathic properties, necessary for the construction of a double lipid layer [32, 33].

It is known that ceramides play an important role in the structural structure of keratins, taking part in the formation of intracellular lamellae of the stratum corneum, which are flattened vesicles pushed out of the lamellar granules into the inter-

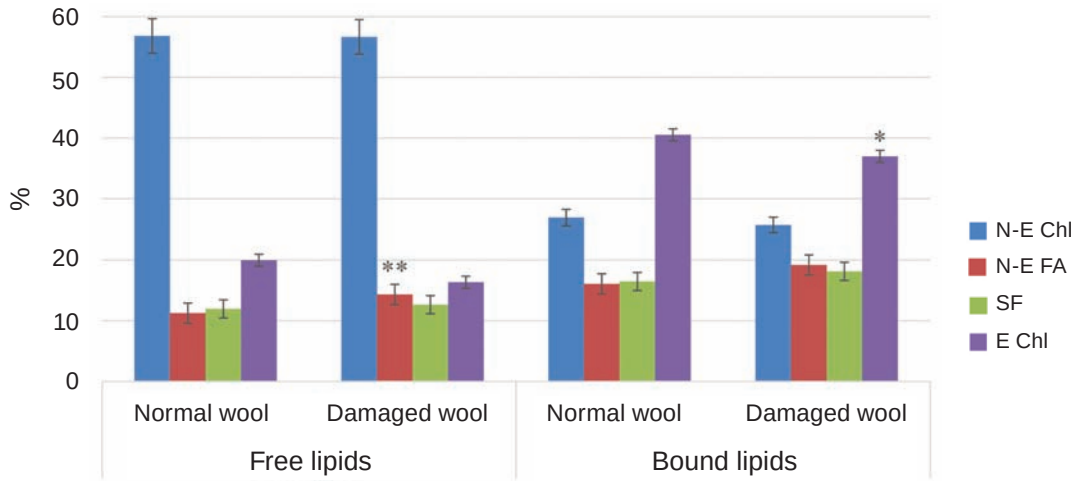

Fig. 3. The content and composition of free and bound internal lipids in normal and damaged wool, separated in system A (N-E Chl – non-esterified cholesterol, N-E FA – non-esterified fatty acids, SF – sterol fraction, E Chl – esterified cholesterol)

cellular space. These flattened vesicles fuse, thus forming a paired bilayer [34, 35]. In view of this, it is interesting to see a decrease in the fraction of ceramides in damaged wool, and this applies to both free ($P < 0.05$) and bound ($P < 0.01$) internal lipids.

When studying the fatty acid composition of internal lipids (Tables 3 and 4), we recorded characteristic differences. As we showed earlier, the fatty acid composition of free internal lipids is represented by 19 acids, two of which are unidentified, and 10 bound acids [37].

In the composition of free internal lipids, the largest amount of fatty acids is hexadecanoic, octadecanoic, and cis-9-octadecenoic (Table 3). The total content of these acids is about 67.7% of their total amount. Moreover, in wool that has undergone biodestructive changes, the amount of these acids decreases. This reduction is especially true for hexadecanoic ($P < 0.05$) and octadecanoic ($P < 0.05$) acids. Instead, the amount of 18-methyloctadecanoic ($P < 0.01$) and 18-methyleicosanoic ($P < 0.01$) acids probably increases in damaged fibers.

Regarding the fatty acid composition of bound internal lipids (Table 4), hexadecanoic acid is likely ($P < 0.05$) to increase in pathologically changed fibers. But the most significant changes are observed from the side of arachidonic and 18-methyleicosan fatty acids. The content of the first in damaged wool increases by six and a half times, the content of the second, on the contrary, decreases by almost the same amount. Thus, it can be assumed that as a result of fiber biodestruction, structural changes occur in the above-mentioned fatty acids.

In hair, fatty acids, the largest of which is 18-methyleicosanoic acid, are bound through a thioester bond to a protein on the outer surface of β-cuticular cells. This lipid F layer is decisive in the formation of the surface properties of the hair and it is the most susceptible to microbiological degradation. The decrease in the content of 18-methyleicosanoic acid in bound lipids and its increase in free lipids indicates that in the process of fiber damage, thioester bonds are destroyed, with the help of which structural lipids are covalently linked through 18-methyleicosanoic acid to protein. As a result, a significant part of the bound lipids goes into the free state [37].

However, the total amount of 18-methyleicosanoic acid in normal wool is significantly higher compared to defective wool. This suggests that a certain amount of it is lost as a result of mechanical exfoliation and subsequent peeling of a certain number of scales of the surface layer of the fiber. And this is natural, given that the main amount of this anteisoacid is localized on the outer surface of the cuticular layer, which undergoes the most significant changes.

Therefore, it follows from the obtained data that the increase of certain types of microorganisms in the fleece of sheep is accompanied by the hydrolysis of some components of the wax, and an increase in the alkalinity of the sweat, which ultimately leads

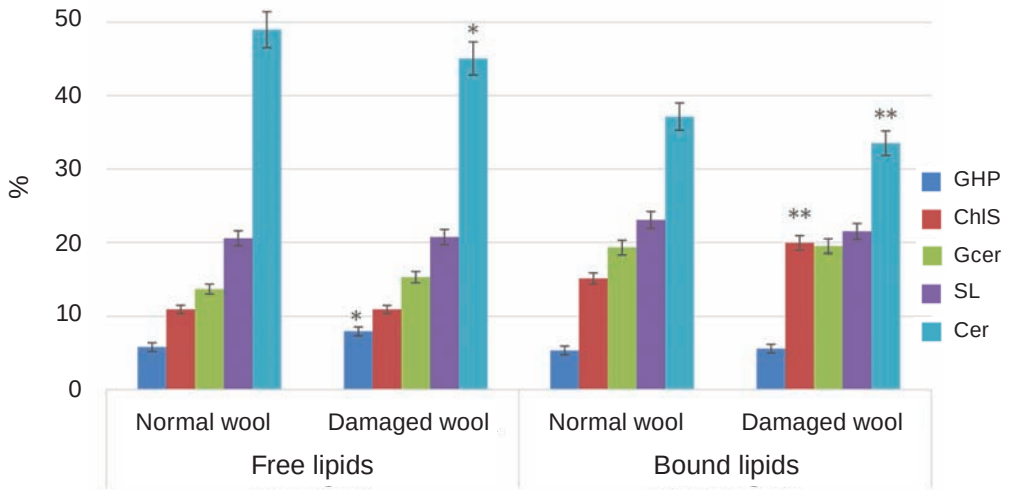

Fig. 4. The content and composition of free and bound internal lipids in normal and damaged wool, separated in system B (GHP – Glycolipids of the highest polarity, ChlS – cholesterol sulfate, GCer – glucosylceramides, SL – sulfolipids, Cer – ceramides)

Table 3. Fatty acid composition of free internal lipids of normal and damaged wool, % (M±m, n = 4)

Fatty acid	Acid code	Wool	
		Normal	Damaged
Tetradecanoic	14:0	0.86 ± 0.24	0.78 ± 0.07
Pentadecanoic	15:0	2.30 ± 1.27	1.49 ± 0.38
Hexadecanoic	16:0	23.92 ± 0.79	18.22 ± 1.17*
Cis-9-hexadecenoic	16:1 ω-7	2.57 ± 0.11	1.79 ± 0.24*
17-Methylhexadecanoic	17:0 iзo	1.89 ± 0.14	1.44 ± 0.07*
Heptadecanoic	17:0	1.31 ± 0.07	1.08 ± 0.07
Octadecanoic	18:0	24.54 ± 0.72	19.37 ± 1.56*
Cis-9-octadecanoic	18:1 ω-9	19.29 ± 0.52	16.29 ± 1.27
18-Methyloctadecanoic	19:0 iso	2.38 ± 0.18	6.50 ± 0.88**
Nonadecanoic	19:0	0.55 ± 0.11	0.27 ± 0.04
19-Methylnonadecanoic	20:0 iso	2.95 ± 0.14	2.51 ± 0.29
Eicosanoic	20:0	3.38 ± 0.29	4.61 ± 1.89
Cis-9-eicosenoic	20:1 ω-11	1.95 ± 0.31	0.99 ± 0.18
Unidentified-1	–	0.70 ± 0.16	0.79 ± 0.29
18-methyleicosanoic	21:0 ai	3.38 ± 0.03	15.75 ± 2.03**
Heneicosanoic	21:0	0.86 ± 0.09	1.25 ± 0.21
Unidentified-2	–	0.42 ± 0.13	1.37 ± 0.45
Docosanoic	22:0	2.42 ± 0.21	2.25 ± 0.56
Tetracosanoic	24:0	4.33 ± 0.44	3.25 ± 0.16
Σ saturated	–	75.07	78.77
Σ unsaturated	–	23.81	19.07
Σ unidentified	–	1.12	2.16

Table 4. Fatty acid composition of bound internal lipids of normal and damaged wool, % (M ± m, n = 3)

Fatty acid	Acid code	Wool	
		Normal	Damaged
Tetradecanoic	14:0	1.76 ± 0.71	4.13 ± 1.33
Pentadecanoic	15:0	2.39 ± 0.91	3.41 ± 0.50
Hexadecanoic	16:0	11.32 ± 0.64	15.74 ± 1.09*
Cis-9-hexadecenoic	16:1 ω-7	4.79 ± 1.38	9.11 ± 2.45
Heptadecanoic	17:0	10.29 ± 0.19	5.40 ± 2.34
Octadecanoic	18:0	15.24 ± 0.56	13.59 ± 2.11
Cis-9-octadecanoic	18:1 ω-9	7.37 ± 1.37	3.71 ± 0.24
Isooctadecanoic	18:0 iзo	2.72 ± 0.40	12.91 ± 3.66
Eicosanoic	20:0	3.87 ± 1.03	25.85 ± 2.43**
18-methyleicosanoic	21:0 ai	40.26 ± 2.13	6.15 ± 0.68***
Σ saturated		87.84	87.18
Σ unsaturated		12.16	12.82

to the deterioration of the protective properties of the wax. As a result, favorable conditions are created for their influence on the very structure of wool fibers, and above all on the cuticular layer, which ultimately leads to its damage. This occurs due to significant quantitative and qualitative changes in the internal lipids of the fiber, both from the side of free and bound fractions. As a result, fibers lose their physico-chemical and technological properties, in particular strength, elasticity, plasticity [38].

Conclusions.

1. The reason for the biodestruction of wool is intensive microbiological insemination of the fleece due to bacteria ($P < 0.001$), actinomycetes ($P < 0.01$) and mushrooms ($P < 0.01$), which use wool wax as a substrate for their vital activity under conditions of increased sweat content and its alkalinity.

2. Biodestruction processes are accompanied by hydrolysis of fiber lipid components, as indicated by an increase in the fraction of non-esterified fatty acids and a decrease in esterified cholesterol in defective wool. In such wool, the content of ceramides decreases in both free ($P < 0.05$) and bound ($P < 0.01$) lipids.

3. Biodestruction leads to significant damage to the cuticular layer of the wool fiber, resulting in the destruction of thioester bonds by which internal lipids are covalently bound to proteins through 18-methyleicosanoic acid. Such wool is characterized by worse physico-chemical and technological properties.

Conflict of interest. The authors have completed the Unified Conflicts of Interest form at http://ukrbiochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

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ВНУТРІШНІ ЛІПІДИ І ЇХ ЖИРНОКИСЛОТНИЙ СКЛАД У ВОВНІ ОВЕЦЬ ЗА БІОДЕСТРУКЦІЇ МІКРООРГАНІЗМАМИ РУНА

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Мікробіологічна деструкція волокон є поширеним пошкодженням вовни овець. Враховуючи визначальну роль внутрішніх ліпідів у формуванні поверхневих властивостей вовняних волокон, метою роботи було вивчити структуру та ліпідний склад нормальної та пошкодженої вовни. Дослідження проводили на вівцематках асканійської тонкорунної породи. Вміст мікроорганізмів оцінювали після посіву на щільні поживні середовища. Методом скануючої електронної мікроскопії досліджували поверхню вовняних волокон, методом тонкошарової хроматографії – вміст внутрішніх ліпідів після попереднього лужного гідролізу волокон, жирнокислотний склад – методом газорідинної хроматографії. Було показано, що у пошкодженій вовні міститься майже втричі більше бактерій, а також присутні вищі рівні актиноміцетів і грибів порівняно з нормальною вовною. Виявлено пошкодження кутикулярного шару внаслідок життєдіяльності мікрофлори руна. У дефектній вовні вміст вільних внутрішніх ліпідів і неетерифікованих жирних кислот був підвищений, а вміст протеїн-зв’язаних ліпідів і етерифікованого холестерину, а також церамідів був знижений порівняно з нормальною вовною. Рівень 18-метилейкозанової кислоти в протеїн-зв’язаних ліпідах пошкодженої вовни був зни-

жений, що вказує на руйнування тіоефірних зв'язків, за допомогою яких структурні ліпіди ковалентно зв'язані з протеїнами через 18-метилейкозаноєву кислоту.

Ключові слова: вовняні волокна овець, біодеструкція, мікроорганізми, внутрішні ліпіди, протеїн-зв'язані ліпіди, жирні кислоти, 18-метилейкозанова кислота.

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