Pathological findings in 'wet belly' in young black sables (*Martes zebellina*)

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Abstract

'Wet belly' in industrial sables is an economically significant problem of modern industrial fur farming in Russia due to defects in the skin and fur of animals, which are associated with the damaging effect of constantly excreted urine. The incidence in young sables in different years can range from 0.1 to 6% in this age group. In this regard, in case of 'wet belly' disease in young sables, the regulations of veterinary intervention provide only local aerosol treatment with antimicrobials. In this regard, there are practically no studies devoted to the search for the causes of wet belly disease in young sables, including from the point of view of analyzing the results of autopsy. The causes and pathophysiological mechanisms of 'wet belly' development remain unknown, in particular, the mechanisms of urinary disorders and urinary incontinence. The present article is the first attempt to analyze the autopsy data of 76 young sables, which showed that quite a large number of animals had concomitant pathology of the digestive and respiratory system, as well as clinical and histological signs of cystitis and probable urinary tract infection (UTI).

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Introduction

'Wet belly' is a disease of some caged furbearer species, including mink, which is associated with urinary incontinence and consequent damage to the abdominal wall and fur.[1] The pathology of 'wet belly' in young, caged sables is largely unknown to Western readers. The successful commercial domestication of sables in Russia and the establishment of farms for their breeding has been associated with the commercial destruction of natural populations of these animals and, consequently, a general decline in the numbers of the species. The most significant accomplishment in the history of sable breeding in Russia is the development of two distinctive breeds: the Black Sable in 1969 and the Saltykovskaya-1 in 2007.[2] In the context of industrial sable rearing in Russia, a range of sheds with varying designs are utilized, wherein the animals are confined to frameless cages crafted from galvanised metal mesh. The cages are arranged in two or four rows, with a technological passageway for service personnel with a concreted floor situated between the rows of cages. The animals observed during the course of this study were housed in standard barns (project 806-32) in frameless cages constructed from galvanised metal mesh, measuring 0.9 x 0.9 x 0.9 x 0.6 meters in size (Figure 1a). The mating season for sables occurs during the summer months, spanning from mid-June to mid-August. The gestation period lasts for 273 to 300 days. The majority of female sables reach sexual maturity at 15-16 months of age and give birth for the first time at two to three years of age. The average litter size is one to eight cubs, with an average fecundity of three pups. The typical lifespan of the animal is 15-18 years, with females typically engaging in breeding activities until the age of 10-12 years.[3] In accordance with the technological cycle, the females of the sable are gestated in April, the pups are weaned at the age of 45 days, and the animals are placed in cages, with two of each sex. Towards the end of July and during the first ten days of August, the young sables are transferred to individual cages. From this point onwards, the animals begin to display the initial symptoms of wet belly, which manifest as urinary incontinence with wetting of the fur in the abdomen and hind limbs (Figure 1b). The entire rearing period of sablefish pups, from birth to euthanasia, lasts approximately seven months. The prevalence of disease in young sable pups, as reported by Severnaya Pushnina, ranges from 0.1 to 6% in this age group across different years. The disease is observed in both male and female animals. The objective of the present study was to investigate pathological alterations in internal organs in diseased animals and to develop a straightforward and practical clinical classification of the disease based on the stages of damage to the skin and fur of animals.

Materials and Methods

The study was conducted in the company of the fur farm Severnaya Pushnina in the Leningrad region. A random selection method was used to autopsy 97 young sables, including 21 healthy animals and 76 animals (64 males and 12 females) with a lifetime clinical diagnosis of 'wet belly'. After planned technological euthanasia, the area of skin and fur damage by urine was measured with a caliper and the area of damage was calculated in square centimetres. Animals were weighed on a Mertech 122 ACF-3000.1 Accurate LSD laboratory scale (Mertech, China).

Autopsy of sables was carried out in the dorsal position, internal organs were examined and then extracted as a single organ-complex. The results of pathological autopsy were recorded in our modified protocol.[4] Bladder tissue samples were fixed in 10% buffered formaldehyde solution. Histological sections of the bladder 7 microns thick were obtained on a Slee Cut 5062 rotary microtome, stained with hematoxylin-eosin, toluidine blue to detect mast cells and Hale's method to identify mucopolysaccharides. Histological sections were viewed using a Zeiss Axio Observer A1 microscope; microphotographs were obtained using a Pixera Penguin 150 CL image capture device. The normality of the distribution was evaluated through the implementation of the Shapiro-Wilk test. The data were evaluated using descriptive statistical methods, with mean values and standard deviations determined in M±S format. The statistical significance of the differences between the indicators of the compared groups was determined by single-factor analysis of variance. Differences of the compared indicators were considered statistically significant at the level of $p<0.05$.

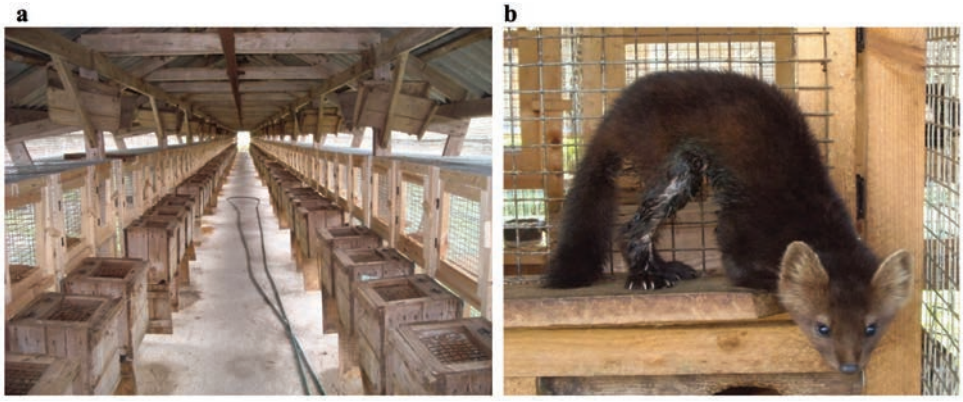

Figure 1. a) Interior view of the animal housing shed. **b**) Black sable with clinical signs of 'wet belly'.

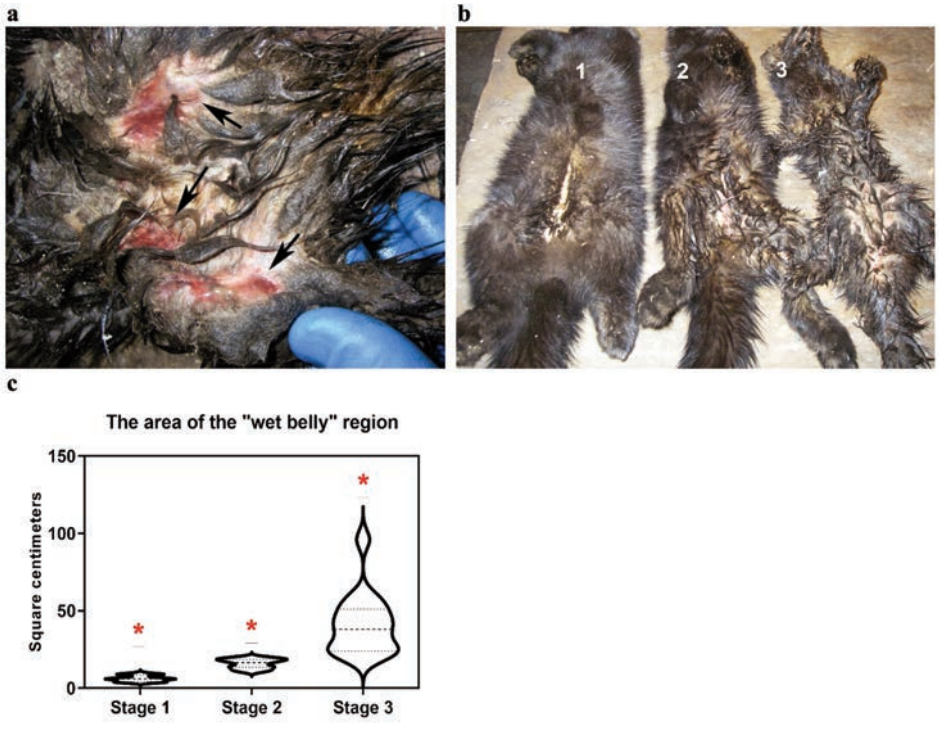

Figure 2. a) Ulcerative dermatitis (black arrows). **b**) Three stages of wet belly in sables: first, second third (1-3, left to right). **c**) Statistical analysis of wet belly area at different stages of the disease (M±S). *$p\leq0.01$.

Results

The main clinical symptom of 'wet belly' in young sables is damage of fur and skin in the lower abdomen area by constantly excreted urine, which may be accompanied by ulcerated skin defects (Figure 2a). In this case, depending on the severity of the disease, the area of the damaged region varies widely from 2 to >20 cm². The classification of 'wet belly' in young sables is proposed to be dependent on the area of fur and skin damage (cm²) and to comprise three stages: the first stage (1, mild) comprising 2-10 cm², the second stage (2, medium severity) comprising 10-20 cm², and the third stage (3, severe form) comprising >20 cm². (Figure 2 b,c). Other sometimes observed symptoms of the disease include anaemic mucous membranes (Figure 3 a,b), diarrhoea, dehydration and dystrophy of skeletal muscles (Figure 3c).

The affected animals are stunted, have a decreased appetite and reduced physical activity. Behavioural signs of discomfort in movement and urination have been observed in some sick sables,

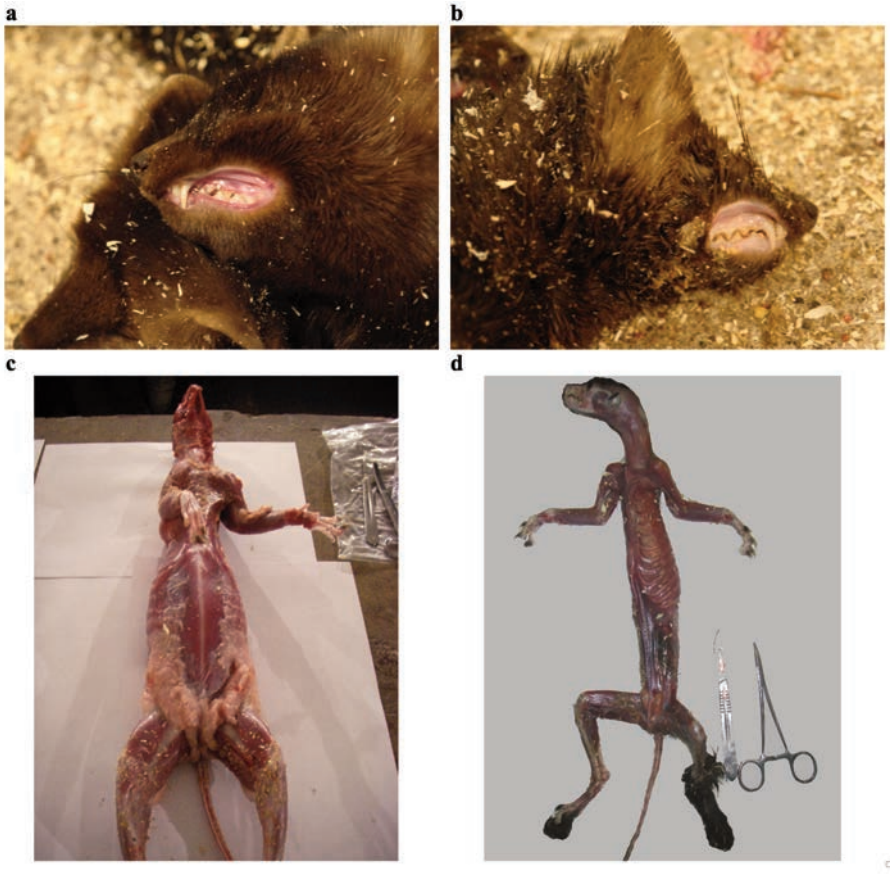

Figure 3. Color of labial mucosa and gums of healthy (**a**) and sick (**b**) sable. **c**) Carcass of a healthy sable. **d**) Sable carcass on stage 3 of "wet belly": emaciation, muscular dystrophy, lack of subcutaneous fat.

Table 1. The results of a selective autopsy of young sables diagnosed with 'wet belly' (n=76).

	Pathological diagnosis	Males (n=64)	Females (n=12)
Respiratory pathology	Bronchopneumonia	14 (22%)	6 (50%)
Stomach and bowel pathology	Gastric dilatation	4 (6%)	-
	Gastritis	8 (12%)	1 (8%)
	Gastroenteritis/enteritis	28 (44%)	3 (25%)
	Enterocolitis	14 (22%)	4 (33%)
Liver pathology	Hepatitis	5 (8%)	2 (16%)
	Hepatic lipidosis	18 (28%)	2 (16%)
Urinary pathology	Cystitis?		
Bladder hemorrhages		18 (28%)	5 (42%)
No pathological changes		17 (26%)	4 (33%)

probably indicating dysuria. Sables with stage 2 and 3 ‘wet belly’ develop extensive dermatitis, ulcerated skin lesions (Figure 2a).

A post-slaughter autopsy was conducted on a randomly selected cohort of seventy-six sable carcasses (sixty-four males and twelve females) diagnosed with ‘wet belly’. The results indicated that 10% of the animals exhibited signs of diarrhea, while 27% displayed evidence of anemia of mucous membranes (Figure 3b). Dystrophic changes in skeletal muscles were present in twenty-six males (37%) and three females (10%) (Figure 3d). Signs of cadaveric dehydration were present in twelve males (17.1%) and two females (6.6%). Associated internal organ pathology was identified in forty-seven (73%) males and eight (67%) females. Pathology of digestive system organs, including liver pathology and bronchopneumonia, was most frequently recorded in males and females (Figure 4 a-f). Pathological changes in kidneys were not detected. It should be noted that seventeen males (26 %) and four females (33 %) of the sample size showed no pathological changes in internal organs (Table 1).

Hemorrhagic areas in the bladder were observed in 28% of males and 42% of females (Figure 5 a-d). Regarding hemorrhagic foci in the bladder, an iatrogenic origin of hemorrhagic foci cannot be excluded due to potentially iatrogenic trauma caused by lethal injection during euthanasia. However, some sables showed extensive, non-localized edema and hyperemia of the bladder mucosa with infiltration of the submucosal layer with erythrocytes. (Figure 5e). Other histological findings in the bladder of diseased animals

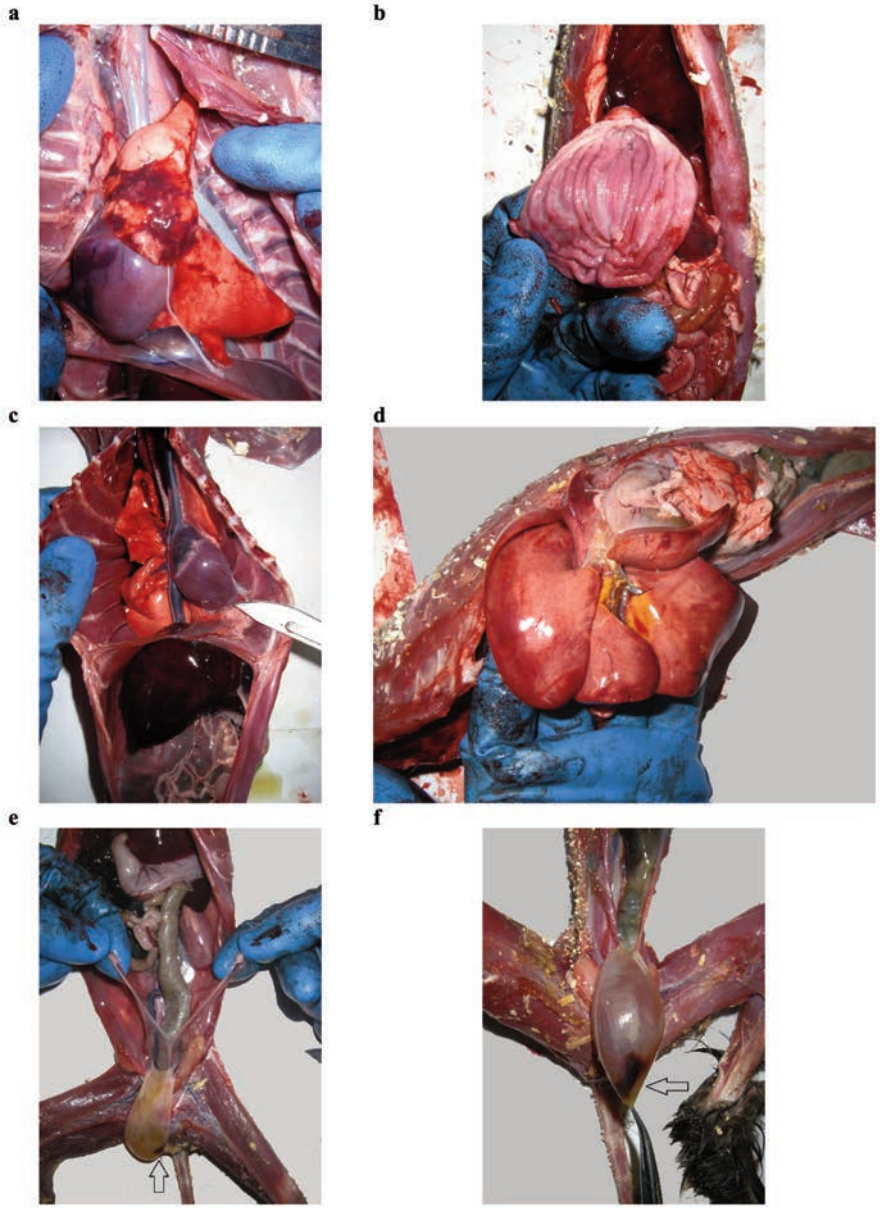

Figure 4. Associated pathology of internal organs in young sables at “wet belly”: bronchopneumonia (**a**); gastritis (**b**); hepatitis and liver necrosis (**c**); hepatic lipidosis (**d**); haemorrhagic area in the female bladder (arrow) (**e**); haemorrhagic area in the male bladder (arrow) (**f**).

include local areas of cell metaplasia in the submucosal layer resembling Von Brunn's nest (Figure 5 b,c). Such a histological picture partly resembles non-Hunner bladder pain syndrome.[5] In some sables, papillary-polypoid elements were present on histological sections of the bladder (Figure 5d), which are similar in some respects to the pattern seen in polypoid cystitis.[6] However, it should be noted that we did not detect infiltration of the submucosal base by immunocompetent cells and/or neutrophils, which would clearly indicate the development of inflammation of the classical type. An increase in the presence of mast cells in the bladder tissue was also not detected.

Discussion

The aetiology of wet belly in caged fur-bearing animals remains unknown. The primary causes of the disease, as identified by Russian researchers, are believed to be malnutrition and a lack of essential nutrients. These include an excess of fat in the diet, a lack of carbohydrates, an imbalance in the ratio of calcium and phosphorus, deficiencies of vitamins A and E, and choline, as well as metabolic disorders. Additionally, the feeding of low-quality by-products and the ingestion of exogenous poisons have been linked to the development of the disease. [6,7] The majority of publications

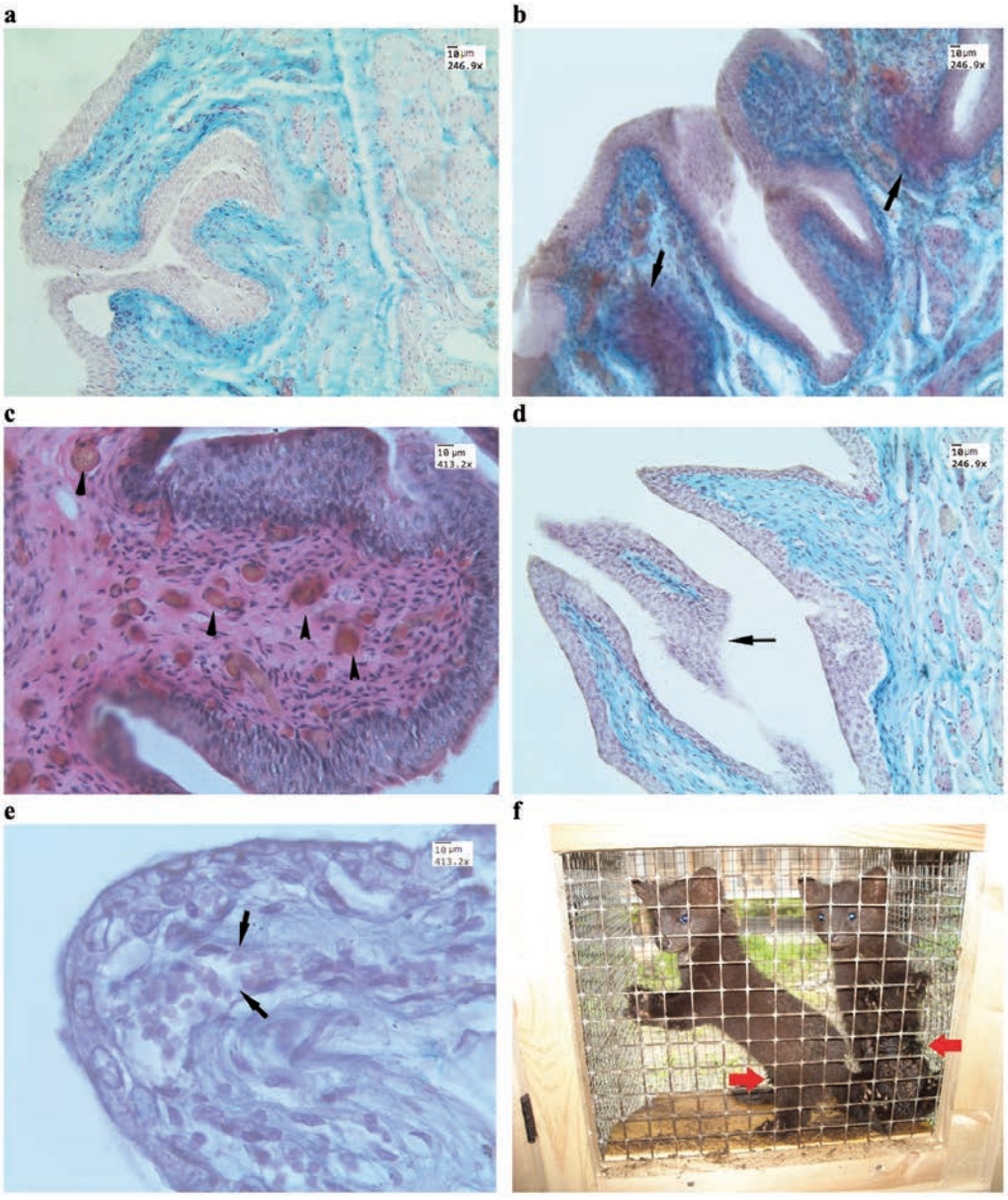

Figure 5. Histology of the urinary bladder in sables: in a healthy animal (staining with toluidine blue) (**a**); in a sable with stage 2 wet belly (staining with toluidine blue), areas of focal cell metaplasia (black arrows) are seen in the submucosal layer (**b**); in a sable with stage 2 wet belly (staining with hematoxylin and eosin), areas of focal cell metaplasia (black arrowheads) are seen in the submucosal layer (**c**); in a sable with stage 2 wet belly (staining with toluidine blue), papillary-polypoid pattern (black arrow) (**d**); in a sable with stage 3 wet belly and hemorrhagic parts in the bladder (Hale's staining), desquamation of the epithelium to the basal layer, infiltration of the submucosal layer with erythrocytes (black arrows) (**e**); two young sables with clinical signs of wet belly (red arrows) **f**).

dedicated to the investigation of the aetiology and pathogenesis of wet belly in fur-bearing animals originate from the 1960s and 1970s. The disease has been the subject of the most detailed study in a representative of the marten family, the American mink (*Mustela lutreola*). In minks, a number of factors have been identified as probable causes of wet belly, including feeding conditions, climatic factors, heredity, breeding techniques, and the influence of pathogenic microorganisms.[8-13] The greatest amount of research has been dedicated to investigating the impact of feeding conditions on animal morbidity. In particular, an increased incidence of morbidity was observed in minks whose diets contained an elevated calcium level of 1.03% and a calcium-phosphorus ratio of 2:1.[14] An increase in the fat content of the diet of minks from 4.6% to 8.6% has been observed to result in a more than fivefold increase in animal morbidity.[10] The guidelines for the feeding and breeding of mink indicate that the incidence of wet belly disease is increased under the influence of unfavourable climatic conditions, as well as when the ambient temperature.[11]

Some authors have noted that "wet belly" presents with clinical features that are similar to those observed in urinary tract infections (UTIs).[15] (Budd *et al*, 1966). Bacterial infection may be a contributing factor in the development of the disease.[16] On the other hand, there are studies indicating complete absence of histological changes in kidneys, ureters, bladder and prepuce in wet belly minks, which refutes the hypothesis of infectious origin of the disease.[17]

Russian researchers have postulated that the pathophysiological mechanisms underlying the phenomenon of 'wet belly' are attributable to the formation of hard-to-soluble lipoid compounds as a consequence of excessive calcium and fat intake. These compounds, when excreted with urine, reduce its surface tension, thereby facilitating the downward flow of urine along the abdominal wall and into the interior of the limbs during the act of urination.[7] Urine with a lower surface tension has the capacity to penetrate fur more effectively, resulting in the phenomenon of "soaking" of the fur. The presence of dicarboxylic acids, a product of fat metabolism, in urine could theoretically act as an agent to reduce surface tension.[18,19] This hypothesis was tested through the addition of dicarboxylic acid to the diet of minks, yet the results demonstrated that this did not lead to the development of the disease, thus disproving the hypothesis.[20]

The observations of Severnaya Pushnina specialists indicate that the incidence of wet belly disease in young sables significantly increases following the placement of animals in individual cages in early August. This is typically done with male and female animals that were previously kept in pairs. It is hypothesised that the housing of animals in pairs following weaning from their mothers contributes to their optimal development and the prevention of self-chewing.[21] However, in accordance with the technological cycle, from 1 August of each year all animals are housed in individual cages. The placement of young animals in individual cages is done, among other things, to prevent fights between animals, which can lead to fur damage. In this regard, it is theoretically possible that technological stress may influence the occurrence of the disease. Nevertheless, the disease is also recorded in cohabitating sable pups prior to breeding (Figure 5f).

To prove the connection between technological stress and the occurrence of "wet belly" in young sables, it is advisable to determine the so-called "stress biomarkers", which currently number more than ten.[22,23] These include the concentration of cortisol in saliva, blood serum and urine, the content of adrenaline and noradrenaline in plasma and urine, the content of albumin and glycosylated hemoglobin in blood serum; the concentration of some cytokines such as IL-6 and IGF-1 in blood plasma, as well as the levels of systolic and diastolic blood pressure. Determination of these parameters in the conditions of industrial animal breeding is practically unfeasible due to the high cost of research work, as well as additional stress for animals during sampling and manipulation.

During pathological examination of internal organs in mink with wet belly, unlike sables in our study, the authors observed good or even very good condition of animal carcasses. The lower part of the skin had a white-cream color, and the fur on the affected areas was colored dark blue due to a high level of melanin. Changes in internal organs were insignificant and manifested as circulatory disorders. Quite rarely degenerative microfoci were found in the liver, spleen or lungs. Sometimes kidneys in minks were enlarged in size and with small areas of yellow-gray tissue edema.[24]

The autopsy of the sables revealed no macroscopic indications of renal pathology. Concurrently, our study revealed the presence of pathological alterations in the digestive and respiratory system organs within a considerable proportion of the examined sample in young sables. It is likely that these findings represent 'associated pathology', although it is unclear whether they can be linked to urinary disorders and the presence of a wet belly. The identification of such a considerable number of pathological alterations within the digestive and respiratory organs offers a compelling rationale for investigating the quality and sufficiency of the nutritional and housing conditions of the animals in question. Of greater interest in the context of identifying the etiology of disease in sables are the hemorrhages in the bladder and the histological evidence of cystitis that we have observed. Further investigation is required to elucidate the etiology of these findings, including the utilization of comprehensive urine analysis and microbiological examination to identify potential pathogens. The next stage of the study will focus on investigating the quality of animal nutrition and urine analysis in sables with "wet bellies". It is noteworthy that 26% of males and 33% of females exhibited no pathological signs of the internal organs or urinary system at autopsy. In light of the established nosology and classification of the condition known as "wet belly," it is reasonable to conclude that this group of sables should be considered as exhibiting the classic diagnosis of "wet belly." However, the role and influence of concomitant pathology in the other animals remains to be elucidated.

Conclusions

Pathological autopsy of young sables with the clinical diagnosis of "wet belly" revealed the presence of "concomitant pathology" of the digestive and respiratory system organs in a rather large volume - 73% in males and 67% in females. At the same time, hemorrhagic areas in the urinary bladder were detected in 28% of males and 42% of females, and only 26% of males and 33% of females had no pathology of internal and urinary organs. An interesting finding is localized areas of cell metaplasia in the submucosal layer of the bladder in diseased sables resembling Von Brunn's nest and papillary-polypoid elements, which partly correlates with idiopathic[5] and polypoid[6] cystitis. Thus, in the context of the search for the causes of the disease, more questions are raised than answered, which provides a logical prerequisite for further research. To the best of the author's knowledge, this is the first study to perform such an analysis from the perspective of evaluating wet belly pathology in young sables of commercially kept sables. An attempt has been made to develop a simple and conve-

nient classification of wet belly disease degrees in young sables, which can be further improved and supplemented, for example, with information on histological correlates of the degree of skin and fur damage at each stage.

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