Acta Veterinaria Hungarica 50 (2), pp. 133–142 (2002)

STRESS-INDUCED CUSHING'S SYNDROME IN FUR-CHEWING CHINCHILLAS

Marina TIŠLJAR[1*], D. JANIĆ[2], Ž. GRABAREVIĆ[3], Borka ŠIMPRAGA[1], A. MARINCULIĆ[4], Ljiljana PINTER[5], Z. JANICKI[6] and Ankica NEMANIČ[1]

[1]Poultry Center, Croatian Veterinary Institute, 10000 Zagreb, Heinzelova 55, Croatia; [2]'PLIVA' d.d., Zagreb, Croatia; [3]Department of General Pathology and Pathological Morphology, and [4]Department of Parasitology; [5]Department of Microbiology and Infectious Diseases, and [6]Chair for Game Biology and Pathology, Veterinary Faculty, University of Zagreb, Croatia

(Received September 17, 2001; accepted December 13, 2001)

One of the most serious problems in the chinchilla industry is 'fur-chewing', when the chinchilla bites off areas of its own or some other animal's fur. The condition generally develops in both genders at the age of 6–8 months. In chinchilla farms in Croatia an incidence of 15–20% has been observed. A pathomorphological, microbiological and parasitological investigation was conducted on eleven 6- to 11-month-old chinchillas of both sexes with clinical symptoms of 'fur-chewing' and three chinchillas without such signs. Histopathology of the adrenal glands and of the chewed skin revealed changes typical of Cushing's syndrome in 'fur-chewed' chinchillas, such as hyperkeratinisation of the epidermis, epidermal atrophy, pronounced follicular and sebaceous gland atrophy, hyperkeratinisation of the follicles with comedo formations and the presence of calcium salts in subcutis.

Key words: Chinchilla, 'fur-chewing', histopathology, skin, hyperadrenocorticism, hypercortisolism, Cushing's syndrome

According to the general classification the chinchilla belongs to the order of *Rodentia* and the family of *Chinchillidae* (species *Chinchilla laniger* and *Chinchilla brevicaudata).* They are native species of South America (Andes), and raised world-wide as ranched animals for the production of fur. Through many generations of selective breeding for fur quality, colour and growth rate, the ranch-raised chinchilla has vastly improved in quality over its wild counterpart (Tapscott, 1998).

Rees (1962) estimated that as much as 30% of chinchillas have chewing habit in which the animals bite off their fur. Therefore 'fur-chewing' ('fur-biting') is not only a great problem because of large damaged areas of the body, from which patches of fur may be chewed (so the shorter fur has a dead appear-

*Corresponding author; E-mail: czp-hvi@zg.tel.hr; Fax: +385 (0) 1 2441 396

0236-6290/2002/$ 5.00

ance and often looks sheared), but fur recovery is generally uncompleted and the chewers have to be eliminated. In most advanced cases, a 'lion's mane' appearance is often seen when all the fur within reach on the lower body has been chewed short (Merry, 1990).

Many theories concerning the cause of 'fur-chewing' have included the vice induced by boredom or stress (loud noises, proximity to crowded streets, visits by guests or other animals), improper diet (without hay or with just pressed hay cubes), too small and/or dirty cages, or inappropriate micro-environmental conditions (temperature and relative humidity different from 12 °C and 60%, respectively) (Mösslacher, 1986; Grauvogl, 1990*a*, *b*; Merry, 1990; Jenkins, 1992). In most cases humans are the main cause of fear and anger, which are well-known stress factors in chinchillas. Thus, correct and gentle handling of these extremely hypersensitive animals, as well as providing enough place for playing to avoid boredom, will reduce the social stress factors to a minimum. These guidelines for raising animals for fur production were also recommended by the Council of Europe (Grauvogl, 1990*a*, *b*). Although fur-chewing attacks all age categories, Mösslacher (1986) emphasised that the most frequent cases occur during pregnancy in young breeding females when another young female is added to a polygamous breeding group. Sometimes it also depends on the buck's incapability of mating. Some chinchilla owners believe that the problem is inherited along a certain line of chinchilla (Harris, 1987).

Owners et al. (1975) described fur-chewing as the only clinical manifestation in some chinchillas suffering from *Histoplasma capsulatum* infection. While Shaull (1988) suggested that the syndrome was caused by a 'fur-breakage' fungus that had not yet been isolated, Merry (1990) quoted that fungal cultures and microscopic examination were invariably negative. The fur-chewers might have increased thyroid and adrenocortical activity (Vanjonack and Johnson, 1973) as well as abnormal adrenal or pituitary glands (Kraft, 1987). Eidmann (1992) has suggested that as an infectious cause is unlikely, the affected animals suffer from malnutrition and chew their fur in an effort to meet their dietary requirements.

A recent theory suggests that 'fur-chewing' is a behavioural disorder because the vice can be 'transmitted' from the mother to the offspring and because it usually appears in the commercial herds with a maladapted displacement behaviour (Williams, 1976; Donnelly and Schaeffer, 1997). Experiments have shown that up to 50% of the progeny of such females also become fur biters (Mösslacher, 1986). Kraft (1994) found that the clinical signs seen in 'fur-chewing' chinchillas and the clinical manifestations occurring in domestic poultry suffering from 'feather-picking' are very similar.

Vanjonack and Johnson (1973) showed increased thyroid activity and adrenocortical hormone activity confirmed by the results of histopathological examination of the thyroid and adrenal glands in fur-chewing chinchillas. However, no data describing skin histopathological findings in affected animals could be

found in the available literature. As fur-chewing is a significant problem in Croatian chinchilla farms, in this work we present the results of clinical, pathomorphological, microbiological and parasitological investigations.

Materials and methods

Eleven 6- to 11-month-old chinchillas of both sexes with clinical symptoms of 'fur-chewing', and 3 clinically healthy animals, were obtained from a chinchilla buying-up station.

Pathomorphological examination

The animals were sacrificed under chloroform anaesthesia and necropsied. Selected tissues (liver, brain, spleen, thymus, heart, kidney, adrenal gland and skin taken from the chewed area) were fixed in 10% neutral formalin, embedded in histoplast and cut into 5-µm thick sections. The slices of all organs were stained with haematoxylin and eosin (HE) while the skin was additionally stained by the methods of van Gieson, von Kossa and Gomori.

Bacteriological and mycological examination

Samples of the liver and fur-chewed skin were taken for bacteriological and mycological analysis. A 5% sheep blood agar (Tryptic Soy Agar, Biolife) and blood agar (Gas-Pack) were used for cultivation of aerobes and anaerobes. The specimens were incubated at 37 °C for 24 h. For detection of *Salmonella* the liver was inoculated into a selective enrichment broth (Selenite Broth, Biolife), and incubated at 41–42 °C for 24–48 h. A few drops of broth culture were inoculated into the solid medium xylose lysine deoxycholate agar (XLD agar, Biolife). After the incubation at 37 °C for 24 h, the suspect colonies were inoculated into triple sugar iron agar (TSI agar, Biolife).

Mycological analysis was based on direct microscopy of the hair and the skin scrapings in lactophenol blue and the culture of the sampled material on Sabouraud's dextrose agar (Biolife) supplemented with chloramphenicol (0.5 mg/ml) and Sabouraud's dextrose agar with chloramphenicol (0.5 ml/mg) and cycloheximide (0.5 mg/ml). All the specimens were incubated at 27 °C for 5 days and 3–4 weeks and suspected cultures were identified by the morphology of the thallus and the microscopic appearance (Campbell et al., 1996).

Parasitological examination

For parasitological investigation the scrapings taken from the skin surface were mixed with 1% potassium alkali and incubated in water-bath for 30 sec. After that they were examined under light microscope.

Results

Clinical features

In almost all animals fur-chewed areas occurred along the midspinal area from the lumbar part to the tail, spreading symmetrically to the inguinal regions. The complete fur cover resembled a moth-eaten coat because the tips and bar portion of the fur were generally missing (Fig. 1). The chewed areas were not hairless but covered with very short fur. Two of eleven chinchillas had a typical 'lion's mane' appearance (Fig. 2). Bilaterally symmetric areas of alopecia were seen in the lumbar regions in only one case. In all cases the head and distal extremities were spared and the chewed area was covered with the short fur of a darker shade compared to the basic fur colour (Figs 1 and 2).

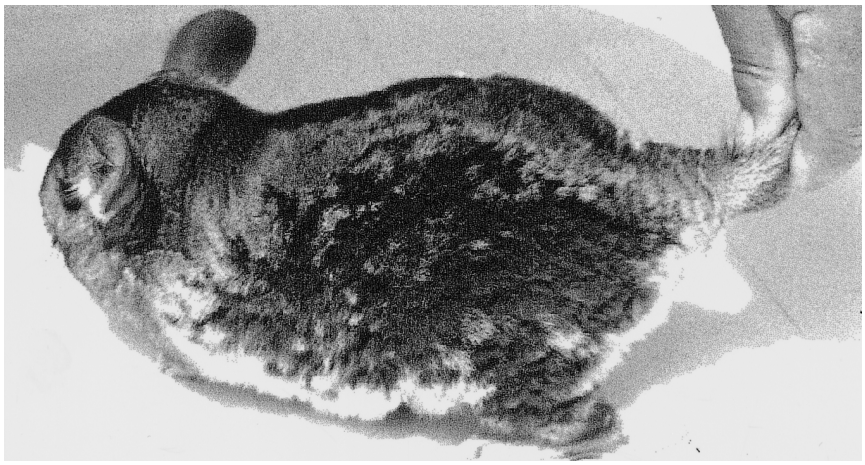

Fig. 1. Chinchilla (11-month-old female). Chewed fur resembles a moth-eaten coat

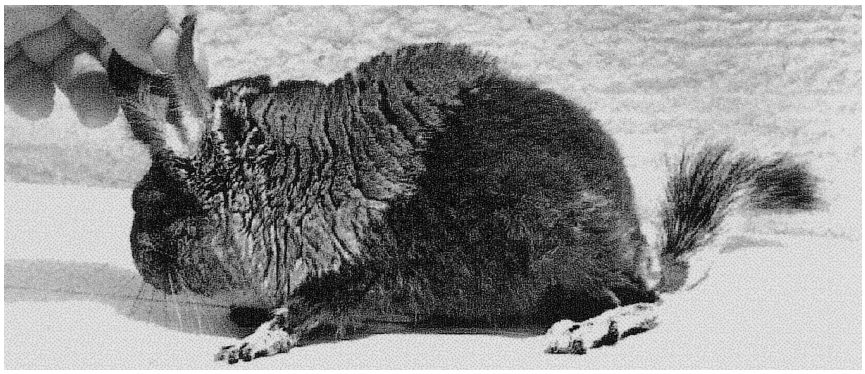

Fig. 2. Chinchilla (7-month-old female). A typical 'lion's mane' appearance. The head, the neck and the distal extremities are spared

Pathomorphological investigation

Gross lesions. Necropsy did not reveal notable differences between the animals. The chewed hair was not present in the gastrointestinal tract. While adrenal hyperplasia was confirmed in all fur-chewing chinchillas (Fig. 3), in the clinically healthy animals no macroscopically visible changes were found in the adrenal glands.

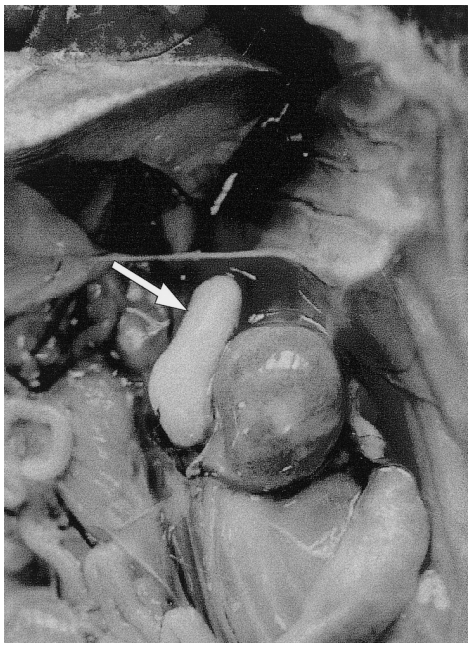

Fig. 3. Adrenal hyperplasia (arrow) in a 'fur-chewer' chinchilla (7-month-old female)

Histopathological examination

There were no significant changes except in the adrenal glands and skin. The adrenal glands generally showed a diffuse cortical hyperplasia (Fig. 4).

The epidermis was hyperkeratinised (orthokeratotic or anuclear hyperkeratosis), which was associated with simultaneous epithelial atrophy (1–2 cell thickness) as well as a pronounced follicular and sebaceous gland atrophy (Fig. 5). In some cases a telogen-type atrophy of the hair follicles was present. Most of the thin-walled follicles contained keratin material (comedo formations) (Fig. 6), and some of them looked like cystic formations (HE, van Gieson) (Fig. 7). The presence of calcium salts was confirmed in three out of eleven samples (von Kossa). In chinchillas without clinical manifestations of 'fur-chewing', mild adrenocortical hyperplasia was followed by epidermal hyperkeratinisation but without follicular keratinisation or follicular and sebaceous gland atrophy.

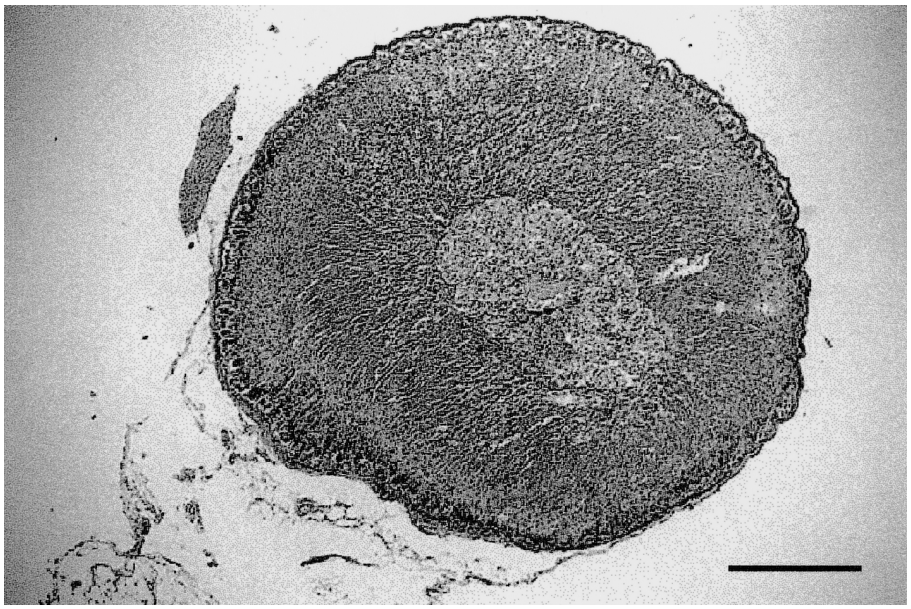

Fig. 4. Adrenal gland in a 7-month-old female 'fur-chewer' chinchilla. Note diffuse cortical hyperplasia. Haematoxylin and eosin (HE). Bar = 50 μm

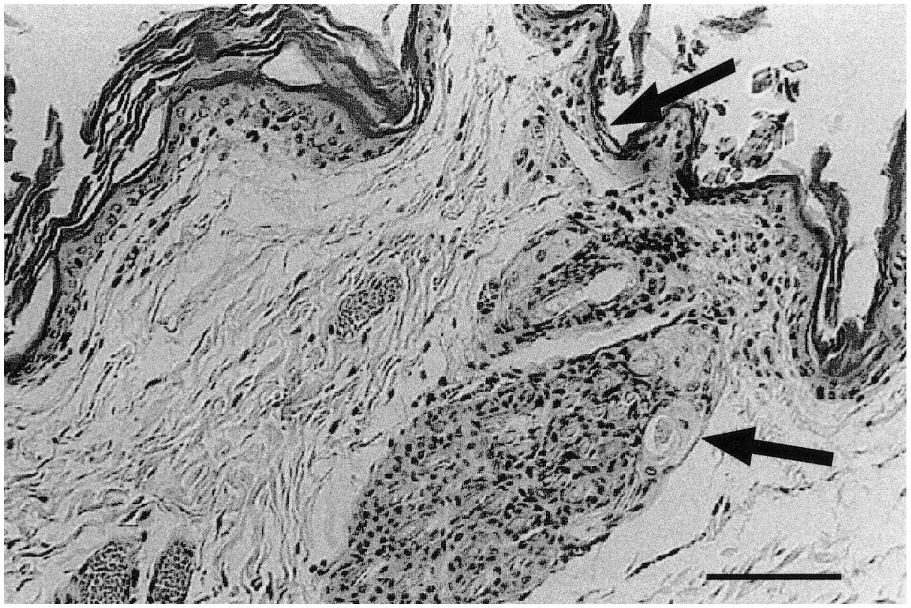

Fig. 5. Skin under the chewed fur. Note hyperkeratinisation of the epidermis and atrophy of the surface epithelium and the sebaceous glands, associated with small accumulations of keratic material (arrows). HE. Bar = 10 μm

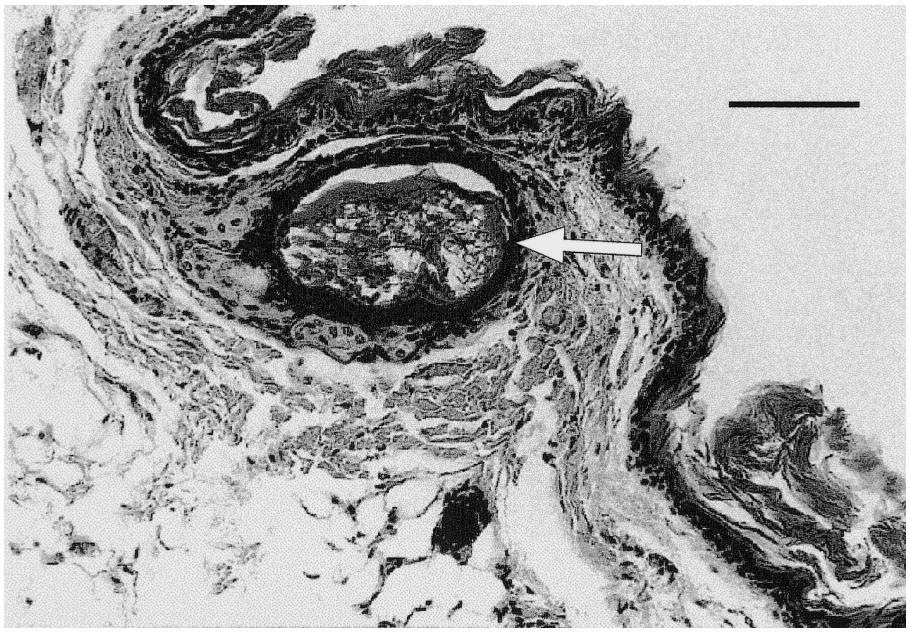

Fig. 6. Skin under the chewed fur. Note the keratin material (comedo) in the thin-walled follicle (arrow). HE. Bar = 10 μm

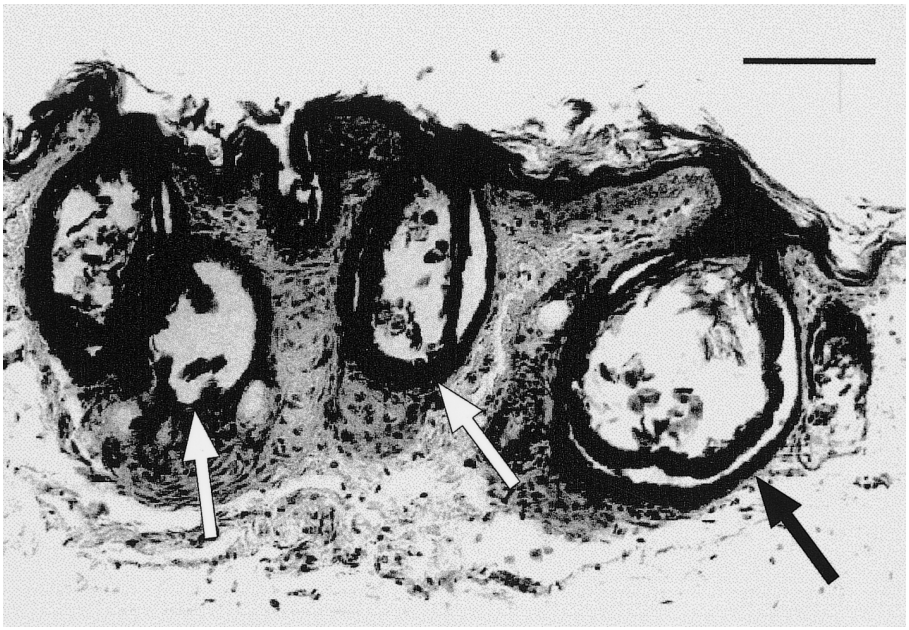

Fig. 7. The skin under the chewed fur. Note cystic-like follicle formations (arrows). HE. Bar = 10 μm

Bacteriological and mycological analysis

Bacteriological analysis did not confirm the presence of any pathogenic bacteria, either aerobes or anaerobes, in the liver or the skin.

Mycological examination confirmed the presence of *Mucor* sp. in two skin samples. In no examined animals was there any evidence of dermatophytes.

Parasitological investigation

Light-microscopic examination of the skin scrapings did not confirm the presence of parasites.

Discussion

According to the case history and data of the literature, the animals examined in this study were at the age (6 to 11 months) when fur-chewing is most severely manifested (Kraft, 1984; Mösslacher, 1986; Donnelly and Schaeffer, 1997).

Clinically, fur-coat changes typical of 'fur-chewing' were observed, but the chewed areas resembled the lesions distinctive for canine hyperadrenocorticism (bilaterally symmetric alopecia, often sparing the head and the distal extremities; a decreased elasticity of the skin, alterations in pigmentation) (Gross et al., 1992). The absence of chewed fur in the gastrointestinal tract of 'fur-chewing' chinchillas confirmed the findings of Kraft (1984). Bacteriological, mycological and parasitological examinations did not reveal significant findings. The most important findings were histopathological changes in the skin and in the adrenal glands.

Adrenocortical hyperplasia followed by the histopathological skin changes described earlier indicated that all examined 'fur-chewing' chinchillas suffered from hyperadrenocorticism accompanied by fully developed Cushing's syndrome. The same histopathological findings in the adrenal glands of 'fur-chewing' chinchillas were shown by Vanjonack and Johnson (1973) and by Kraft (1987, 1994), but histopathological examination of the skin of fur-chewing chinchillas was not performed.

Although follicular keratosis is a nonspecific feature of many atrophic (as well as dysplastic) follicular skin diseases, comedo or keratic cyst formation within a thin-walled follicular infundibulum is generally restricted to Cushing's syndrome.

Cutaneous calcinosis, followed by comedo formations, also supports this diagnosis (Gross et al., 1992).

Gross et al. (1992) have reported that behavioural changes are associated with canine hyperadrenocorticism. Two such changes, self-mutilation and aggressiveness, characterise 'fur-chewing' behaviour in the chinchilla.

In this study it was interesting to note that furred skin taken from 'non-chewers' showed a mild epidermal hyperkeratosis but without follicular atrophy or comedo formations and cutaneous calcinosis. However, the adrenal glands displayed identical, although milder, microscopic presentation as in 'fur-chewing' chinchillas.

Our findings corroborate the hereditary, congenital or behavioural nature of the syndrome (Vanjonack and Johnson, 1973; Mösslacher, 1986; Harris, 1987; Kraft, 1987; Donnelly and Schaeffer, 1997). Namely, affected animals could be stress-sensitive or stress-prone variants which could not adequately cope with stress.

This work provides new morphological evidence that hyperadrenocorticism can be demonstrated in the background of fur-chewing in chinchillas. Our findings cannot confirm Zeinert's hypothesis (Zeinert, 1986) that 'fur-chewing' is not a threat to the animals' health. For a definitive and complete diagnosis a great number of clinical and laboratory tests are required, especially if the presumptive diagnosis includes hormonal disorders. On the basis of the present studies it cannot be determined whether this phenomenon is due to primary stress susceptibility or to some other cause.

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