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Following decapitation of the fish, the liver was immediately extracted and fixed in 5% glutaraldehyde for light and electron microscopic analyses. Samples for structural analysis were processed routinely for light microscopy, and finally embedded in paraffin. Sections were stained with hematoxylin-eosin and P.A.S.. Samples for ultrastructural study were processed according to the method of Sabatini (4), and finally embedded in Durcapan (A.C.M.). The quantitative analysis of lead in liver was determined in tenches from each batch by atomic absorption spectrophotometry (Perkin-Elmer 5100) using a dry ashing procedure.

### Results and discussion

Attention should be drawn to the absence of gross lesions in the hepatic parenchyma of tench subjected to lead nitrate poisoning. Toxic processes involving hepatic dystrophy tended to be characterised by a certain disorganisation of parenchymal architecture, particularly in the center of the lobuli, oedema and sinusoidal dilatation. Fatty degeneration and inflammatory infiltration was observed. This was more evident in the animals with longer exposure.

Ultrastructural hepatocyte analyses revealed alterations primarily in mitochondria and in the organoids belonging to the cytoplasm vacuolar system. Although intranuclear or intracytoplasmic inclusions have been reported in kidney and liver of both mammals and fish subjected to acute lead poisoning, no such findings were made in tench liver. Electron microscopy, however, revealed abundant fine

electron-dense granular material, which may constitute the initial stages of formation of inclusions (5).

It would seem to be this degenerative process which gives rise to the final formation of some residual bodies which reflect the moderate accumulation of lead but of cell debris and undigested material (5). Like other heavy metals, lead is manifestly accumulative. The lead concentration in hepatic parenchyma is much lower than in kidney and gills of the same tench. Therefore, the consumption of species which have already accumulated quantities of lead into their structures which continue to rise further up the food chain, reaching their maxima in predators.

In conclusion, it can be said that the hepatic alterations observed in tench subjected to acute experimental lead nitrate poisoning, include a moderate accumulation of lead, and moderate lesions in liver parenchyma, deducing, in this case, that liver is not a target organ in front of kidney and gills. On the other hand in kidney and gills severe necrotic-degenerative changes are established that became responsible of the death of the tenchs by a respiratory and renal failure.

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### ENZOOTIC NASAL ADENOCARCINOMA OF SHEEP IN SPAIN

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Enzootic nasal adenocarcinoma or enzootic intranasal tumor is one of the neoplastic processes which most often affects small ruminants. Generally speaking, clinical manifestation of the illness occurs in most cases in adult animals (2-5 years old), exhibiting a high mortality rate but always with a low rate of morbidity. Enzootic nasal tumours have been described as basically affecting sheep (1, 2, 3) and goats (4, 5, 6), although similar processes are also seen in cattle, horses, pigs, rabbits, cats and dogs. Tumour processes are even known to be common in the nasal cavity of man.

### Material and methods

A study was made of three adult (6-7 years old) merino sheep. These animals had been suspected of suffering from a chronic cachexia-like process and had been referred to the Pathological Anatomy Department of the Veterinary Faculty in Cáceres.

### Results and discussion

A flock of 1200 free-grazing sheep was examined, revealing a clinical history of tumour processes in 8 animals. Three of them were necropsied for subsequent histopathological study. The affected animals were, in every case, between 6 and 7 years old, and only ewes were affected.

A chronic pattern which had already been present for 3-4 months was noted in every case. From then on, severe sluggishness, babesiosis, anorexia, moderate dyspnea and progressive emaciation all started to become obvious. Clinical signs which were accompanied by copious secretion and exudate, both nasal and ocular. There was intense mucous secretion, which led to the appearance of a great deal of focal scabbing and considerable alopecia both on the muzzle and in the periorbital area.

In the anatomopathological study, a large tumourous mass was observed. It was always bilateral, spreading out to occupy a great part of the nasal cavity and thus aggravating the evident dyspnea. The mass had a distinctly polypoid, rounded morphology, with a smooth, whitish surface, and used the cribriform plate as a base for implantation and development. Examination of sections showed it to be of moderate consistency, exuding liquid. Its surface was brilliant and in the stroma there were small osseous trabeculae.

Several researches (1, 3) have achieved reproduction of the tumour process in apparently normal animals by means of intranasal inoculation of a tumor mash. This evidence strongly suggests that the tumour process has a viral etiology, although the specific taxonomy of the causative agent remains to be defined. Although some authors maintain that this intranasal tumour is caused by a herpesvirus (1), others believe that its development corresponds to the action of

retroviruses (4), as these would induce primary tumour transformation.

Microscopically, the tumour mass is covered by a hyperplastic respiratory epithelium, while on the inside of the lamina propria there is loose connective tissue rich in oedematous liquid which is P.S.A. positive. In the central part of this connective stroma, marked neoplastic glandular development is evident in the form of numerous acinotubular glandular structures of various sizes, with an irregular lumen occupied by cellular debris and an acidophilic, P.A.S. positive substance. There were neoplastic structures surrounded by intense lymphocyte and plasma cell infiltration. Other glandular structures were clearly cyst-like, with a planocuboidal epithelium, but in no case did they exhibit any cellular reaction.

Ultrastructurally, it could be seen that the tumour mass was covered by a hyperplastic respiratory epithelium, in which ciliated mucous cells with microvilli could be distinguished. Numerous plasma cells were found within the mass. These had a highly-developed granular endoplasmic reticulum, and the cisternae, whose core was fairly

electron dense, were considerably dilated. This could suggest the development of an important immune response from the neoplastic tissue. Equally evident was the presence of numerous multinucleated cells with irregular nuclei and a dilated granular endoplasmic reticulum.

Many viral particles were present, both in the intracellular and extracellular spaces. They were coated with a capsid and the core was moderately electron dense. These particles were always found in dense aggregates.

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**ATYPICAL BASAL BODIES IN THE OVIDUCTAL MUCOSA (AMPULLA) OF GILTS WITH PRIMARY CILIARY DYSKINESIA (PCD)**

*F. Roperto, P. Galati, P. Maiolino, S. Papparella*

The basal bodies/centrioles have a remarkable degree of constancy in size, shape, location and ultrastructure, so that atypical ones are very rarely seen in normal and pathological tissues.

Abnormalities of the basal bodies are more frequently observed in the «immotile cilia syndrome» in human and canine cases.

We describe atypical basal bodies in the oviductal mucosa of gilts affected by the primary ciliary dyskinesia (PCD).

In fact, we examined 500 cross-sectioned basal bodies and found roughly 3% of atypical basal bodies.

Defective basal bodies of so-called «half-centrioles» type were present in a fairly high percentage (1.8%).

Other abnormalities were represented by the distortion of geometrical configuration, the presence inside the lumen of electron-dense granular material. Only one basal body was characterized by 8 triplets and one singlet.

We discuss this report on the basis of the most recent genetic studies about the molecular composition of basal bodies/centrioles.

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**EXPERIMENTALLY INDUCED LIVER GRANULOMAS AFTER LONG-TERM INHALATION OF QUARTZ IN NON-HUMAN PRIMATES**

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Extrapulmonary lesions of silicosis are well known in human pathology. They mainly occur in the liver, spleen and bone marrow (2, 6). In experimental medicine, hepatic granulomas could be induced by intravenous injection of silica in rats and mice (3, 4). The experiments reported here were carried out to study the effect of simultaneous exposure to quartz and excess pressure on the development of interstitial lung fibrosis. Additionally to alterations of lungs and lung associated lymph nodes, quartz induced liver lesions were detectable (5).

**Material and methods**

Cynomolgus monkeys were exposed for 26 months, 5 days per week and 8 hours per day to conditions listed below:

group	n	quartz (mg/m <sup>3</sup> )	pressure (bar)
I	5	—	1.0
II	7	5.0	1.0
III	4	5.0	2.5
IV	5	—	2.5

At the end of the study, computed tomography as well as X-ray examination were undertaken. The respiratory tract was fixed by instillation. Additionally, a retrograde perfusion via the abdominal aorta was carried out. The morphologic and morphometric evaluation was done on paraffin embedded histological sections stained with haematoxylin-eosin (H&E) and after silver impregnation according to Gomori. Morphometric evaluation was carried out with the