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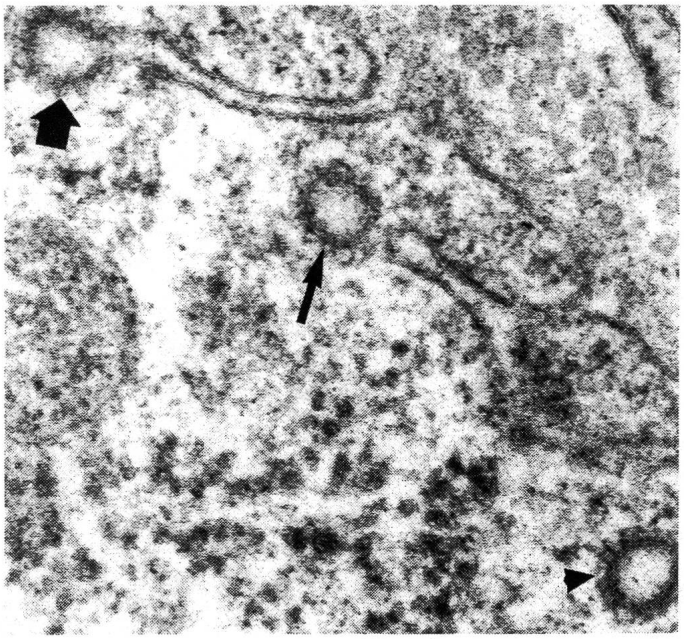

Fig. 1: ECL-cell: invagination of the basal plasma membrane with a coated segmentation (big arrow); coated vesicle in the immediate vicinity of a plasma membrane invagination (small arrow); coated vesicle near the basal plasma membrane (arrow head). 136 000X

nally towards the basal lamina and seldom towards parietal cells. The most interesting observation was the occurrence of smooth invaginations of the plasma membrane with coated segmentation and coated vesicles in the vicinity (fig. 1).

Discussion

Neuroendocrine cells release their products by exocytosis (2, 3). From their findings on endocrine cells of the pituitary gland and adrenal medulla Douglas et al (2) and Nagasawa (3) postulated the exocytosis-vesiculation-sequence (EVS). According to the EVS, exocytosis proper is followed by the sementation of coated vescles from the fusionated granule. The vesicles move into the cytoplasm where they lose their coats and become smooth vesicles that are partly taken up by lysosomes for degradation.
The appearance ot exocytotic figures of the rat fundic ECL cells and the enhanced proportion of lysosomes after treatment with B831-78 is in accordance with the EVS.

Conclusion

Under conditions of potent acid suppression the ECL cells in the rat gastric fundus show a massive degranulation and an increased number of exocytotic figures. The occurrence of smooth invaginations with coated segmentation of vesicles for the first time gives evidence that the EVS applies to the rat fundic ECL cells.

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EXPERIMENTAL INTOXICATION WITH LEAD NITRATE: HISTOLOGICAL STUDIES OF HEPATIC CELLS FROM TENCH

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Lead, one of the highly toxic heavy metals, is found in increasing amount in living matter, in both marine and terrestrial ecosystems, largely due to the continuous and increasing pollution of the encironment in industrialised regions all over the world.
The abundant sources of environmental pollution involving lead include fumes from combustion engines, the dumping of industrial waste, the use of lead piping for water supplies and the use in the past of lead-based paints. This pollution gives rise to the progressive accumulastion of lead compounds in the aquatic environment (1, 2). Because they are in direct and continuous contact with water, teleost act as ideal primary biological indicators of the degree of pollution present in an aquatic ecosystem (3). This article describes hepatic lesions in tench following acute experimental lead nitrate poisoning – due to the consumption of large amounts of lead over a short period of time – and also considers the extrapolation of the results to other animals.

Material and methods

The adult tench (*Tinca tinca, L.*) used in this experiment were sacrificed as shown in Table 1.

Table 1

Batch	No. Tench	Duration of Exposure
I	3	24 h.
II	3	48 h.
III	3	4 days
IV	3	7 days
V	3	9 days
VI	3	12 days
VII	3	15 days
Control	22	same as principals
Total	43	

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 absortion 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 electrondense 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 same 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 ist one of the neoplastic processes which most often affects small ruminants. Generally speaking, clinical manifestation of the illnes 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 Caceres.

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 an 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 exudence, both nasal and ocular. There was intense mucous secretion, which led to the appearance of a great deal of focal scabbing and condiserable 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 osseus trabeculae.

Several researches (1, 3) have achieved reproduction of the tumour process in apparently normal animals by means of intranasal innoculation 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