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**AA AMYLOIDOSIS IN CAPTIVE BEIRA ANTELOPES
(DORCATRAGUS MEGALOTIS) LINKED TO MYCOPLASMAL
PLEUROPNEUMONIA**

L. Luján*, T. Crespo*, A. Deb†, A. Arif†, R. Borjal‡,
E. Salazar*, N. Álvarez*, M. Pérez*, G.M. Dorrestein‡ and
S. Hammer†

*University of Zaragoza, Spain, †Al Wabra Wildlife Preservation, Qatar and
‡NOIVBD, The Netherlands

Introduction: During 2006 and 2007, a group of captive Beira antelopes kept at the Al Wabra Wildlife Preservation (Qatar) suffered from a severe respiratory epidemic characterized by a high mortality rate.

Materials and Methods: A total of 48 Beira antelopes died within 2 years from a population of 58 alive at the beginning of 2006 with a mortality of 32.8% and 46.2% in 2006 and 2007, respectively. Morbidity in 2007 reached 100%. All animals that died were investigated by post-mortem examination and samples for histopathology and bacteriology were taken.

Results: Lesions consisted of moderate to severe fibrinous pleuropneumonia with mucoid airway obstruction. Bacteriology demonstrated the presence of *Mycoplasma ovipneumoniae* in six animals, whereas *Mycoplasma* species were detected by a generic polymerase chain reaction in 22 further animals. AA amyloid was observed in 24 antelopes and was mostly located in spleen and liver, whereas kidney, lymph nodes and gut were only mildly affected.

Conclusions: AA (systemic) amyloidosis is described for the first time in Beira antelopes following the inflammatory stimulus induced by respiratory infections linked to *Mycoplasma* spp.

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**EXPOSURE OF OVINE BLASTOCYSTS TO
POLYCHLOROBIPHENYL (AROCLOR 1254):
ULTRASTRUCTURAL MODIFICATIONS AND
EMBRYOTOXICITY**

D. Malatesta, C. Palmieri, F. Zacchini, G. Ptak, P. Loi and
L. Della Salda

Faculty of Veterinary Medicine, Teramo, Italy

Introduction: The potential embryotoxic and fetotoxic effects of polychlorobiphenyls (PCBs) have been reported only in rabbits, rats, mice, mink and gerbils. No data are available concerning the effects of PCBs (in particular, Aroclor 1254) on ovine reproduction and offspring growth rate and the ultrastructural modifications of blastocysts due to exposure to these toxic compounds. The aim of this study was to analyze ultrastructural anomalies induced in ovine blastocysts by different concentrations of PCB (Aroclor 1254).

Materials and Methods: Ovine blastocysts were fixed in 2.5% glutaraldehyde, post-fixed in OsO₄, dehydrated, rinsed in propylene oxide and embedded in epoxy resin. Semi-thin and ultrathin sections were stained with toluidine blue and uranyl acetate and lead citrate, respectively.

Results: All treated blastocysts were characterized by increased lipid droplets, severe cytoplasmic vacuolation (single membrane-bound vacuoles and empty lacunae containing whorled membrane, granular osmiophilic material or laminated membranes), marked mitochondrial swelling with loss of cristae and pyknosis, and a few autolysosomes containing mitochondrial remnants.

Conclusions: Embryos treated with PCBs show severe ultrastructural modifications not dependent on PCB concentration and correlated to the progressive increase of cell mortality rate indicated by studies *in vitro*. These results contribute to knowledge of the harmful effects of these compounds on reproduction and embryonal growth rate.

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**THE EXPRESSION OF p63 AND CALPONIN IN CANINE
MAMMARY TUMOURS: HISTOGENETIC AND
PROGNOSTIC CONSIDERATIONS**

W. Łopuszyński*, H. Kuczyńska*, Y. Millán†, S. Guil-Luna‡,
R. Sánchez-Céspedes† and J. Martín de las Mulas†

*Department of Pathological Anatomy, Veterinary Faculty, Life Sciences
University, Lublin, Poland and †Department of Anatomy and Comparative
Pathology, Veterinary Faculty, Cordoba University, Spain

Introduction: Identification of myoepithelial cells plays an important role in classification of canine mammary tumours (CMTs). The aim of this study was to compare immunoreactivity of calponin, a smooth muscle-specific protein, with expression of p63, a regulatory cell cycle element that has been demonstrated in myoepithelial cells.

Materials and Methods: Tissue samples from 10 benign and 32 malignant CMTs were evaluated. The myoepithelial phenotype of cells was confirmed by using complementary antibodies including alpha-smooth muscle actin, cytokeratin 14 and vimentin.

Results: Co-localization of p63 and calponin was demonstrated in identical cell populations, with p63 signal restricted to the nucleus and calponin to the cytoplasm. A gradual decrease of p63 immunoreactivity was observed during transformation of myoepithelial cells from pre-existing through hypertrophic and spindle-stellate to rounded cells. The antibody to p63 protein had the highest specificity for myoepithelial cells among all tested antibodies, as myofibroblasts or vascular smooth muscle cells lacked p63 expression.

Conclusions: p63 is a sensitive and specific myoepithelial cell marker and may be included in immunohistochemical panels aiming at identification of myoepithelial cells in CMT.

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**OVARIAN ARTERIOVENOUS HAEMANGIOMA IN A FEMALE
DOG**

G. Marino e, A. Tortorella, D. Tribulato and G. Montalbano

Department of Veterinary Public Health, Messina, Italy

Introduction: Haemangioma is a vascular tumour, which occasionally may affect the ovary. Vascular tumours must be differentiated from vascular abnormalities (hamartomas). The presence of normally organized arterioles and nervous structures in the lesional context may be indicative of a vascular hamartoma.

Materials and Methods: A 12-year-old female German shepherd dog, with no oestrous in the previous 2 years was admitted for abdominal distension and presence of a multicavitated structure difficult to evaluate by ultrasound. At laparotomy, the genital tract was removed including a large left ovarian neoplasm (31 × 20 × 7 cm). The mass (weight 4 kg) had a spongy and congested appearance and, on sectioning, had multiple cavernous blood-filled structures. The uterus was congested and had cystic endometrial hyperplasia. The right ovary had paraovarian cysts and an early granulosa cell tumour.

Results: The mass was characterized by newly-formed vessels, including small arterioles with scant and abnormal elastic lamina, irregularly displaced into a fibromuscular stroma. No nerves were identifiable by means of S100 and PGP9.5 immunohistochemistry. A diagnosis of arteriovenous haemangioma was made.

Conclusions: The reported case shows the differential approach, which enables the distinction between a haemangioma and a vascular hamartoma. The term arteriovenous haemangioma indicates the presence of a mixture of neoplastic arterioles with a scant elastic lamina and veins.