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ABSTRACT

This study covers various aspects of caprine coccidiosis involving both natural and experimental infections. Epidemiological studies using faecal samples from goats on nine farms in south-eastern Queensland and from areas near Melbourne, Victoria revealed mixed infections with up to eight species of Eimeria. Only five were found in a small number of samples from Kenya. The two most frequently seen species were Eninakohlyakimovae and Earloingi. A ninth species was found, in very small numbers, in a separate study using goats from a University farm in south-eastern Queesland. The nine species were as follows (listed in the order of the most frequently seen to the least): Eninakohlyakimovae, Earloingi, Ealijevi, Eapsheronica, E.christenseni, E.hirci, E.jolchijevi and E.pallida.

The numbers of parasites, as measured by the oocysts per gram (OPG) of faeces, decreased with the age of the goats, but the few goats seven years and older reversed this trend. Management factors which resulted in a greater chance of goats ingesting infective oocysts from contaminated food and water, as well as the age structure of the herd, affected the level of faecal oocyst shedding on the various farms. The following did not have any apparent effect; concurrent infections with helminths, caprine arthritis and encephalitis (CAE), the sex of the goat, pregnancy, or milking. Breed differences were noted, with the Anglonubians shedding significantly more oocysts than the Saanen, Angora and British Alpine goats.

Studies on the effect of corticosteroids on the level of oocyst shedding by goats with natural infections or by naive

kids infected experimentally, revealed increased and sometimes prolonged parasite reproductivity as reflected in the high faecal OPG values. This phenomenon was seen in goat groups with multispecific, as well as with monospecific infections with E.apsheronica. Gains in body weight were reduced, but the packed cell volume levels were not altered by the levels of coccidial infections used.

The histopathology of the goat intestines following natural and experimental infections consisted of lesions typical of coccidiosis. There was generalised haemorrhage and oedema, and some greyish-white nodules (1-4 mm in diameter) were seen in the jejunum. Microscopic changes were; erosion of the villi epithelium, especially in the jejunum, and the development of crypt abscesses comprised of necrotic host cells, degenerating parasite stages and inflammatory mononuclear and polymorphonuclear leukocytes. The nodules were composed of schizonts and masses of parasites:there were a few microgametocytes, and many macrogametes in various stages of maturation. The macrogametes were P.A.S. positive, and this reaction intensified with their maturation so that young oocysts stained very intensely. The merozoites and microgametes were negative but unlike the macrogametocytes and young oocysts, they stained positively with Feulgen DNA stain .

A study of the life cycle of <u>E.apsheronica</u> was carried out and the accompanying pathological changes noted. Development of first generation, giant schizonts took place in the small intestine, the mesenteric lymph nodes and in the caecum, a location from which they have not been described before with any species of <u>Eimeria</u>

of goats or sheep. Most of the schizonts matured within 16 days. Second generation schizonts and the gametogenous stages were mature at 20 days post-infection and they were also found in the small intestine and the caecum. Generalised haemorrhage, congestion, and oedema were seen in the small and large intestines. The first generation schizonts caused little inflammatory reaction except with a few of those in the submucosa of the jejunum where they sometimes caused pronounced cellular responses characterised by neutrophils, macrophages and lymphocytes.

In another study, intravenous inoculation of intact sporulated oocysts of <u>E.apsheronica</u> and of <u>E.christenseni</u> with or without concurrent administration of corticosteroids resulted in infections in the small intestines. Weekly weight gains were temporarily checked in the <u>E.christenseni</u> infected kids around the period when the infections became patent.

In a study of <u>in vitro</u> excystation of <u>E.apsheronica</u> oocysts, the best results were obtained with pre-treatment with carbon dioxide followed by incubation at $37^{\circ}C$ in 5 % bile then treating with 0.25 % trypsin. The optimum temperature for excystation was $37^{\circ}C$.

The relative susceptibility of Anglonubian and Saanen goats to experimental coccidiosis was investigated using a mixture of six Eimeria species (about 50 % of the oocysts in the infecting dose was E.christenseni). This confirmed the earlier epidemiological observation that Anglonubian goats shed more oocysts than the Saanens ($P \le 0.01$), and the Anglonubians also lost more weight. In both goat breeds, post-infection serum antibody levels,

measured by ELISA, increased significantly above the pre-infection levels. The Anglonubians had somewhat higher pre-infection antibody levels, but this did not seem to have any effect in controlling the infections.

Electron microscope observations on features of the endogenous stages of E.apsheronica, E.arloingi and E.christenseni generally agreed with earlier reports on other Eimeria spp. Between individual species, however, differences were noted. In E.apsheronica, merogony was by endodyogeny and endopolygeny whereby merozoites budded exogenically and endogenically. E.arloingi meronts formed cytomeres with peripheral concentration of nuclei, some with a spindle apparatus. No formed merozoites of E.christenseni were seen, but the meronts had peripheral aggregations of potential merozoite nuclei indicating that maturation was by external budding.

Macrogametogenesis was basically similar in all three species. However, the endoplasmic reticulum and mitochondria in the macrogametocytes of <u>E.christenseni</u> were not seen, and there was much more glycogen than in the other two species. The two commonly described types of wall forming bodies (WFBs 1 & 2) were seen in all three species, but the the type 2 bodies of <u>E.apsheronica</u> were unusual because they sometimes occurred in groups of up to four bound by a common membrane of endoplasmic reticulum. The WFBs type 1 were seen to contribute to the formation of the oocyst wall by what was seen in T.E.M. to be a disaggregation into smaller bodies, but these may have been oblique sections of the bud-like processes, observed by S.E.M., of the WFB 1. Observation by SEM of fusion of the WFB 1 confirmed

a commonly believed but not observed process of fusion or coalescence of these bodies to form the oocyst wall.

The nucleus of the macrogametyocyte was found to decrease in size as the macrogametocyte matured, and its margins became irregular with indentations in which were glycogen vacuoles, lipid inclusions and, in <a href="Example:Example:Example: Example: Examp

Microgametogenesis was found to occur at the surface of the microgametocytes. The microgametes of E.apsheronica and of E.christenseni had a mitochondrial groove on the nucleus, and a thick stalk connected the microgametes of the latter species to the microgametocyte prior to full maturation. In E.apsheronica, an atypical microgametocyte filled with mature microgametes was seen. This was unlike the other two species, which normally released the gametes as they matured. In E.arloingi, a few large microgametocytes were seen which were filled with microgametes in the centre of which was a mass of dark material, within which were dark round granules. The residual body in the microgametocyte of E.apsheronica and of E.christenseni contained endoplasmic reticulum, while that of E.arloingi contained groups of glycogen vacuoles.