

COCCIDIOSIS

T. V. Susila, R. Amutha and S.C. Edwin
Department of Poultry Science, Veterinary College and
Research Institute, Namakkal-637001.

INTRODUCTION

Coccidiosis is an economically important disease in poultry production and even during mild infections, losses in feed conversion efficiency and decreased weight gains may lead to the difference between profit and loss in modern, intensive production. Globally, the poultry industry invests over US\$800 million annually to control coccidiosis and still the disease is associated with 6% to 10% of total mortality in the industry. Coccidial infections are known to be a predisposing factor to other syndromes, in particular necrotic enteritis. The reduction of prophylactic antibiotics in feed has led to increasing problems with necrotic enteritis, as it is more difficult to control the causative organism, *Clostridia perfringens*. It is now widely known that the pathogenicity of *Clostridia perfringens* is related to a previous infection with *Eimeria* species, which damages the cells lining the gut, allowing colonisation of the *Clostridia*.

Step - wise life cycle

Nine species of *Eimeria* are known to infect chickens. Each has a specific site of infection, and this is often used as a rough diagnostic tool to determine the species of parasite present. The life cycle has both intra and extra intestinal phases, with intra intestinal phase being responsible for the pathology associated with the disease.

The infective form is the sporulated oocyst which contains sporozoites, enters the host via contamination of food/water. The sporozoites are liberated from the oocyst. The factors responsible for excystment are CO₂, trypsin and bile (In birds it is mainly by mechanical rupture of oocyst in the gizzard). The liberated sporozoites pass through the penetration tube. They are engulfed by the macrophage in the lamina propria and are transported in to the glands of Lieberkuhn. Here they leave

the macrophages and enter the epithelial cells (Intra epithelial leucocytes-IEL), lining the glands and the developmental forms being found distal to the host cell.

Schizogony /Merogony

Then the sporozoite round to form a Trophozoite, then it starts its schizogony/ Merogony (A sexual life cycle). The trophozoite divide to become a schizont/ meront, which contains numerous first generation schizonts. The first generation merozoites again enter the cell and form second generation merozoites. Some of the second generation merozoites enter next generation i.e. third generation. Rest of the second generation merozoites / third generation merozoites enter gamogony / gametogony, the sexual reproduction.

Gamogony/Gametogony

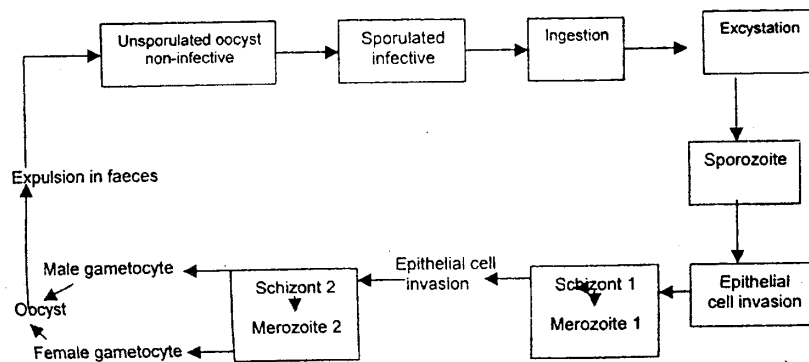
Some merozoites enter the cell; divide by

binary fission to produce numerous nuclei called as **"Microgamonts"**. The microgamonts become mature, motile and acquire 2-3 flagella called 'Microgametocytes/ Microgametes' (analogous to sperm). The rupture of the host cell releases them. Most of the merozoite enter the cell and become rounded. They gradually increase in size, which become spherical/oval called as **"Macrogamonts"**, which mature to form **"Macrogametocytes/ Macrogametes"**. The microgametes enter the cells of macrogametes and syngamy takes place. The resultant **zygote** gets a thick resistant wall by wall forming bodies with plastic

granule called as an **Oocyst**, they are diploid. They have sporont mass and it is excreted out in faeces as an unsporulated oocyst.

Sporogony

The unsporulated oocyst expelled in the faeces, in the external environment, sporogony (a sexual multiplication) takes place. In this, sporont divides into sporoblasts and ultimately 4 sporozoites develop. This sporulated oocysts found in the environment is infective to other host, via contamination through **water/feed**.



In most cases the number of oocysts ingested is small, leading to a sub-clinical infection with no obvious diagnostic symptoms, and subsequent immunity to re-infection. However, as a result of the intensive farming techniques practiced by most poultry producers the number of oocysts in the litter rapidly build up to very large numbers, which are then ingested leading to a severe infection with clinical symptoms. These are usually seen as a reduction in feed intake and feed conversion efficiency, with subsequent weight loss, a disheveled looking bird, diarrhoea and blood in the faeces. In very severe infections mortality occurs. Pathology is largely associated with the destruction of the epithelial lining of the infected part of the intestine, which results in a reduced ability for the digestion and absorption of nutrients by the bird.