Coccidiosis is a dreaded disease of advanced poultry production. The disease in poultry is principally caused by intestinal protozoan parasite of Eimeria genus. All Eimeria species excepting few are intracellular parasites. These Coccidial species require a host for their development and inside the intestine of hosts they reproduce by multiple asexual (schizogony) and sexual fission. The main threat to intensive poultry production is represented by caecal and intestinal Coccidiosis.

Chickens become infected with coccidia by ingesting oocysts (eggs) from litter, soil and contaminated feed and water. The infected birds then excrete oocysts in their faeces to provide a source of infection for other birds. As coccidia can survive for long periods in infected birds and the environment, the parasite is ubiquitous wherever it is raised.

There are seven species of Eimeria that cause clinical diseases in chickens and immunity to any one species does not protect birds against infection with other species. Chickens generally are exposed to coccidia in their environment and over time develop immunity to the parasite. As long as measures are taken to keep the number of oocysts to which the birds are exposed under control, clinical diseases should not occur.

How do birds become infected?

Normally, most birds pass small numbers of oocysts in their droppings without apparent ill effects. Coccidiosis becomes important as a disease when birds are reared, under conditions that permit the build up of infective oocysts in the environment. The intensive rearing of domestic chickens may provide these conditions.

Young chickens pick up the infection from contaminated premises (soil, houses, utensils etc.). These premises may have been contaminated previously by other young infected birds or by adult birds that have recovered from the condition. Wet areas around water drinkers are a source of infection.

### Pathogenecity of Coccidia in Chickens

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Infective zone</th>
<th>Pathological lesions</th>
<th>Intestinal contents</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>E. acervulina</em></td>
<td>Duodenum</td>
<td>Characteristic white flecks or spots which in heavy infection may coalesce, leading to thickening of the gut wall</td>
<td>Mucoid</td>
<td>Slight, Reduced live weight gain and feed conversion ratio (FCR)</td>
</tr>
<tr>
<td><em>E. maxima</em></td>
<td>Jejunum</td>
<td>Thickening of mucous membrane. Punctiform red lesions.</td>
<td>Grayish brown mucous</td>
<td>Slight to moderate. Live weight gain markedly reduced, Pigmentation may be affected</td>
</tr>
<tr>
<td><em>E. necatrix</em></td>
<td>Jejunum</td>
<td>Initially small white spots leading to punctiform or coalesced haemorrhagic lesions.</td>
<td>Mucoid, blood tinged</td>
<td>Generally high, general body condition impaired, Poor performance.</td>
</tr>
<tr>
<td><em>E. tenella</em></td>
<td>Ileum, caeca</td>
<td>Haemorrhagic lesions leading to core formation.</td>
<td>Blood tinged, core formation may occur</td>
<td>Generally high, General body condition impaired, Poor performance</td>
</tr>
<tr>
<td><em>E. brunetti</em></td>
<td>Colon</td>
<td>Reddish striping. May later see necrotic white spots</td>
<td>Bloody mucoid faeces</td>
<td>Slight to moderate.</td>
</tr>
</tbody>
</table>
Oocysts remain viable in litter for many months. In this way, they can contaminate a farm from year to year. Oocysts are killed by freezing, extreme dryness and high temperatures.

Several factors influence the severity of infection. Some of these are as follows:

- The number of oocysts ingested. Generally an increase in the ingested number of oocysts is accompanied by an increase in the severity of the disease.
- Strain of Coccidia. Different strains of a species may vary in pathogenicity.
- Environment factors affecting the survival of the oocysts.
- Site of development within the host. Coccidia that develop superficially are less pathogenic than those that develop deeper.
- Age of the bird. Young birds are generally more susceptible than the older birds.
- Nutritional status of the host. Poorly fed birds are more susceptible.

Coccidiosis in chickens is generally classified as either intestinal or caecal. Most serious cases of intestinal Coccidiosis are caused by *E. necatrix*. Caecal Coccidiosis is caused due to *E. tenella*.

Coccidiosis generally occurs more frequently during warmer (April To September) than colder months (October to March) of the year.

Clinical Coccidiosis is related to grooming many birds in a limited floor space. Such husbandry allows large number of coccidial oocysts to accumulate on the floor, be infested and replicate to high levels in the gut. By contrast a few birds in a large space means that only a small number of oocysts are allowed to be picked up and parasite the gut lining. These produce no clinical disease and allow the development of immunity. A steady intake of a small number of oocysts, a “trickle infection” will produce a very strong immunity.

In the Indian context much attention has been paid to clinical coccidiosis: sub-clinical coccidiosis is often overlooked. In a less acute form (sub clinical diseases), Coccidiosis may cause unthriftness; poor feed conversion, reduced weight gain and drop in egg production. This has a great economic importance in poultry industry.

Coccidial infection may cause a watery or bloody diarrhoea and death depending on the species of *Eimeria* involved, dose of oocysts and the level of host immunity.

**Control of Coccidiosis**

The current control strategy requires a practical approach both to reduce challenge from infectious Oocysts as well as controlling sub-clinical disease. The aim must be to use drugs we have effectively and strategically. With intensive poultry production on the rise, we require more diverse fighting products and husbandry techniques.

To aid in prevention of Coccidiosis, broilers are given coccidiostats in their feed continuously from one day old until close to slaughter. There is a potential problem with parasite resistance to coccidiostat. Clinical disease is staging a comeback with the emergence of resistant and tolerant strains. Optimal control of Coccidiosis during the most critical phase of the broiler crop can only be ensured by proper selection of anticoccidials, which should include products from different chemical groups possessing different mechanisms of activity.

Often ignored is the expression of resistance as a sub-clinical infection. Sub-clinical coccidiosis is a warning sign of worse to follow and is, by itself, a major cause for economic losses in poultry production.

In order to prevent or control Coccidiosis a number of anti coccidial products are present in the market. Research into vaccination programs is ongoing, especially for breeder flocks. Now a days vaccine for commercial broilers are also available. Vaccination for Coccidiosis presents advantages like on site vaccination, avoids risk of errors in feed mills, no dry residues, no toxicity and no development of anticoccidial resistance. The cost factor is also to be considered while using the vaccines.

The commercial Coccidiosis treatment products can be divided in three main groups:

**Chemical Compounds**

These compounds are chemically synthesized and are cidal in action. The parasite is killed by interfering in one or other metabolical or physiological processes. Onset of resistance can be rapid and unpredictable.

The most important are:

- Amprolium
- Diclazuril
- Halofuginone
- Meticlorpindol or Clopidol
- Nicarbazin
- Robenidine.

**Fermentation products or Ionophores**

Manufactured by fermentation they work by interfering with the Na/K –pump of the cell. Development of resistance is generally slow and gradual.

The most important are:

- Lasolacid
- Maduramicin
- Monensin
- Narasin
- Salinomycin
- Semduramicin.
Drinking water applications
These products are also chemicals by applied in the drinking water for treatment of severe outbreaks.
The most important are:
• Quinolone derivatives
• Toltrazuril
• Amprolium

Coccidiosis control programmes

Since the parasite Eimeria can easily and quickly develop resistance, strategic and appropriate use of the available anticoccidial product is advised. For that purpose two types of programmes are applied.

Shuttle Programme
In this programme within the same flock, two different anticoccidials are used: one in starter feed, one in grower feed. Either a chemical compound is used in the first phase, or an ionophore compound. In the grower phase, most of the time an ionophore is used. This is practiced in order to reduce the development of drug resistance in coccidia and to maintain the effectiveness of individual anticoccidials.

Rotation or Mono programme
As resistance develops over time with continuous usage of the same product or programme, a rotation is implemented after a certain number of flocks. Drug rotation may be implemented after every three months, six months or every year. This mainly depends on the Coccidiosis challenge, seasonal disease pressure and on the intensivity and management of the local Farmers.

Strategy for control of Coccidial infections through use of Coccidiostats

Considering the above, there are three proposals and general practices on use of coccidiostats.
• Chemical in starter and Ionophores in growers.
• Ionophores only.
• Ionophores in starter and Chemicals in growers.

In the opinion of poultry experts, the first solution is the most efficient and effective against resistance of specific coccidiostats. In the first few weeks or days of life, a birds immune system may not be fully developed and it is usual for protection in the early stages to be provided using a chemical agent and then later switch to an ionophore. Generally ionophorous anticoccidials are static in action, so it limits losses in performance while allowing natural immunity to develop in the birds. Such shuttle programme provides an adequate balance between control of infection and the development of immunity in the older birds.

Ionophores only could be a good solution but it will involve a wide range of coccidiostats at disposal. It is recommended to use four different products in rotation to be used to avoid any development of resistance against specific anticoccidials.

Last solution is not recommended at all since some chemical coccidiostats cannot be used in birds after 20 days of age because it may lead to presence of chemical residues in meat/egg.

Conclusion
Coccidiosis control may well be achieved by a mixture of all available programmes – mono (rotation)/Shuttle and coccidiostats categories – Ionophore/Chemical. A synergic combination could be as below.

Coccidiosis Vaccine is also in vogue in many parts of the world in commercial broiler production. In India vaccination is not yet widely practiced. Coccidiosis vaccines will induce immunity in poultry specific to the Eimeria species incorporated in the vaccines. The usage of vaccines depends on ensuring minimum interference with therapeutics, feed additives and other vaccines. Also the practice of vaccination alone to a certain extent might lead to precipitation of Necrotic Enteritis, because anticoccidial drugs particularly Ionophores have activity against gram positive bacteria including Clostridia species.

Thus it can be concluded that coccidiostats when used in a structured and monitored anticoccidial programme provide a very effective tool that permits the optimum performance of the bird without compromising health status.

<table>
<thead>
<tr>
<th>Month of the year</th>
<th>1-3</th>
<th>4-6</th>
<th>7-9</th>
<th>10-12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strategy of Anticoccidial</td>
<td>Chemical + Ionophore</td>
<td>Ionophore only (Mono programme)</td>
<td>Chemical + Ionophore</td>
<td>Ionophore only</td>
</tr>
</tbody>
</table>

ERRATA In our previous newsletter, linolenic acid value (C 18:3) of soya oil was wrongly printed as 0.2, instead please read it as 6.8.

The inconvenience caused is regretted.
THE LIFE CYCLE OF EIMERIA SPP. AND ISOSPORA SPP. (THE CAUSATIVE AGENT OF COCCIDIOSIS)

The infected oocyst is ingested by a host.

The oocyst “excysts” in the small intestine, and sporozoites infect the cells lining the small intestines.

Oocysts are passed in the feces of the infected host.

The parasite reproduces asexually in the cells of the small intestine producing merozoites. The merozoites infect more intestinal cells.

After several generations of asexual reproduction, oocysts are produced.

Asexual reproduction (schizogony or merogony) is self-limiting and ceases after several generations. Oocysts are produced only as long as schizogony continues.

(Parasite and Parasitological Resources)