

Coccidiosis and Its Prevalence in Broiler Poultry

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Abstract

Despite the dense and industrial poultry industry today, coccidiosis has also been recognized as one of the most economically important diseases. Despite many advances in the prevention and treatment of coccidiosis over the past 5 years, the disease is still one of the major causes of injury and injury in the poultry industry. As the poultry industry expands and the number of primary medicines is eliminated, newer methods of prevention and the use of more effective medicines to treat and ultimately improve poultry management and the use of new management tools to control coccidiosis and reduce inevitable waste are needed. Therefore, in this study, we have thoroughly investigated this disease in broiler poultry.

Keywords: Coccidiosis, Broiler poultry, Eimeria

INTRODUCTION

The increasing human population and the scarcity of natural resources, on the one hand, and human attention to quality of life, on the other hand, have called for deep thinking and a great challenge to nature. The issue of nutrition and nutrition needs is of particular importance, since nutrition needs have been one of the primary but fundamental goals of humans for many years. A society that cannot meet the majority of its food needs will never be able to succeed in other scientific and industrial areas, and even in the importance of nutrition needs, as long as the independence and territorial integrity of a given country and its food independence will remain ^[1].

For this reason, in order to meet the nutritional needs of the global population, today certain scientific techniques and techniques have replaced the traditional methods of agriculture and livestock, and in fact we are witnessing the replacement of industry with tradition in agriculture and livestock. An important feature of the livestock industry is the increasing livestock and poultry density in new farms as well as the efficient and reasonable use of agricultural inputs. Applying specific scientific management to these farms and striving to improve the feed conversion rate and accelerate the growth of livestock and livestock are other characteristics of the industry. But this rapid increase in production efficiency and change in the natural system of livestock breeding will never be without cost, the rapid spread of diseases and the emergence of metabolic disorders, and the observation of a variety of syndromes from the effects of industrial livestock breeding. Some diseases, such as coccidiosis, are of particular importance because reflecting the disease annually causes millions of dollars in damage to the livestock sector worldwide. According to the above descriptions, it is important to identify and prevent, treat and combat animal diseases and diseases, and the management of

livestock farms should be sufficiently aware of these diseases ^[2]. Since dense and industrial poultry farming has been considered, there has been an increase in coccidiosis damages in poultry flocks. Worldwide, the damage caused by this damage is estimated at about \$ 900 million to \$ 1 billion ^[3]. The disease is densely dependent on how the poultry is raised so that the rate of disease increases when a large number of susceptible young birds are placed in an environment conducive to the growth and reproduction of coccidia ^[4]. Coccidiosis is produced by the protozoa of the apical complex of the protozoa, which have a direct evolutionary pathway, without an intermediate host, which is transmitted by resistant oocysts and propagates in the host body of the parasite within the intestinal epithelial cells. Most of the bird coccidia belong to the genus *Imria*.

Imrimas are abundant in nature, but the disease occurs when too many birds of the same species are kept locally, and this causes a large number of imimara to be capable of causing disease, so coccidiosis is a major concern in the widespread and intensive poultry farming ^[5].

In dense poultry breeding, oocytes rapidly proliferate in the litter, and contact of the chicken and the introduction of large numbers of oocytes into the gastrointestinal tract and intestine

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cause clinical coccidiosis in poultry. The number of oocytes per unit area is low, so introducing fewer oocytes into the gastrointestinal tract not only causes clinical coccidiosis but also creates immunity against the disease. So by applying or using effective management tools that Reduce the number of oocytes in the bed in addition to preventing clinical disease The herd will become infected, but the flock will gradually become immune to disease ^[6].

Imria is a protozoan intracellular parasite and the causative agent of coccidiosis. Coccidiosis is an important disease in the poultry industry, which mainly has two forms (clinical) and subclinical (subclinical). Clinical signs, severity, and lesions due to coccidiosis vary, and are often undetectable because many infections are subclinical and are not addressed by ranchers and veterinarians ^[7]. The clinical form of coccidiosis causes severe lesions in broiler, laying, laying mother, broiling mother, broiling ancestors and laying ancestors. These protozoa proliferate in the intestinal tract and cause tissue damage resulting in malfunctioning of nutrition, digestive and food absorption processes, dehydration, blood loss and increased readiness to receive other pathogens. Also in broiler chickens, even its subclinical form reduces feed conversion and weight gain of poultry and chickens and the important sign is entrite and the parasite is almost due to young animals disease.

Economic importance of coccidiosis:

It is one of the most important growth restricting factors in the poultry industry. Only after the sulfanamide family drug was manufactured did it become possible to grow poultry on a more commercially viable basis. Until the early 1940s, poultry farmers had to keep the poultry on a wired floor, or they had to suffer the damage caused by heavy mortality following poultry farming. Half a century later, it has been partially controlled using a variety of chemical drugs and the proper management of coccidiosis ^[8]. In industrialized countries, disease-related mortality is not a major issue, but rather the negative impact of the disease on food conversion efficiency, pigment reduction, and reduced profits ^[5]. In developing countries with limited access to resources for disease diagnosis, access to antioxidants and new management techniques, coccidiosis is the leading cause of mortality and a negative impact on poultry growth.

Factors determining the economic importance of coccidiosis are:

- 1- Increased drug resistance especially to anti-coccidiosis chemicals
- 2- The rapid growth of the broiler industry in tropical and humid climates, which is a good environment for coccidian bodies.
- 3- Increased incidence of immune-system-mediated diseases such as Mark and Gambio's that are contributing factors to the disease.
- 4- The widespread use of abnormal components in the diet such as wheat, barley and local products that affect intestinal function

Coccidiosis

The disease is caused by protozoa that are single-celled parasites of the genus *Eimeria*, also called diarrhea. *Aimeria* has a worldwide reach and is dedicated to hosting and special building. Most vertebrate animals, including birds and mammals, are susceptible to coccidiosis. Hosts of this parasite include birds, cows, goats, pigs, horses, camels and rabbits ^[9]. The first classic study of bird parasitic coccidia was performed by Tyzzer ^[5].

Eimeria parasite

Eimeria parasites are in the Protozoa dynasty, the apical branch of Complexa, Sporozoa, sub-species *Coccidia*, *Eococcidia*, *Aimrina* and *Eimeridae*.

The parasites of the subfamily *Amrina* are intracellular parasites of the intestinal tissue and have two asexual (schizogonic) and sexual (gametogonious) reproductions, which, for *Eimeria*, occur both stages in the host body, and therefore *Eimeria*, It is considered to be monogenic, but other genera of the *Eimeridae* family that include isospora are heterogeneous ^[7].

The following indices are used to determine *Eimeria* species:

1. Number of sporocysts and sporozoites after hatching
2. The time required for equitable hogging.
3. The period of incubation or the period after the oocysts have been experimentally tested in the host to detect them in the faeces.
4. Characteristics of the oocyst in terms of dimensions, color, wood or absence of valve, wall thickness, presence or absence of residual objects ^[10].

- **Biological Properties of *Eimeria*:**

Parasitic diseases can be divided into different dimensions. One of these divisions refers to the location of the parasite inside or outside the body. According to this view, parasites or parasites are divided into two groups of internal parasites and external parasites. Due to the location of the adult form of *Coccidia* parasite, it can be said that it is one of the internal parasitic diseases.

The majority of the parasites in this branch are host, and the development of the parasite takes place in specific host cells; *Eimeria* has all parts of a living cell such as the mitochondria, the endoplasmic reticulum, the nucleus, the membrane, and the ribosome. The *Eimeria* oocyst contains four sporocysts and each sporocyst has two sporozoites .

- ***Aimeria* oocyst building**

The oocyst is a zygote and is in fact a parasitic stage in the life cycle of the parasite that resides in the host tissues and is excreted through the stool. It has four sporocysts each of which have two sporozoites. The characteristics of the *Eimeria* oocyst can be summarized as follows: It has two transparent layers that may be membrane-coated, some oocysts show a clear valve when they are complete, and through which the inner membrane is protruded and they show the polar cap.

Near the crystal clear valve, called the polar grain, is found within the sporocysts, two oval or circular sporozoites^[1, 2]. The oocysts may contain the oocyst residual and the sporocyst residual, respectively, the former being the sporocystic residue and the latter being the residual material of the sporozoite formation^[11]. The sporocyst has a button object called a stand object at one end and another object called a stand object below it. Sporozoites are usually lying, having a rounded end and anterior, conical, or sausage-like ends. They have one or more protein-rich or refractive cells (Figure 1). The precise role of the refractory body within *Eimeria* sporozoites is not known, but the anterior refractory body disappears 24 hours after infection, and it has been determined that diffusion of the refractory body into the schizont cytoplasm is required for continued parasite development^[12].

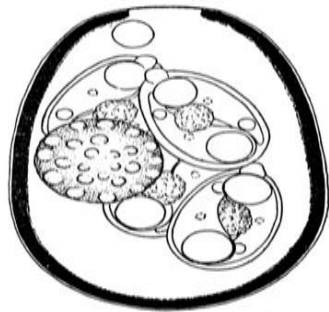


Figure 1: Schematic view of *Eimeria* oocysts

• Species diversity of *Eimeria* parasite birds:

So far nine *Eimeria* species have been identified in poultry including

1. *Eimeria* Necatrix
2. *Aimeria* Magzima
3. *Aimeria* Astrolina
4. *Aimeria* Bronti
5. *Aimeria* Tenela
6. *Aimeria* Mittis
7. *Eimeria* Perry Cox
8. *Aimria* Miyati
9. *Eimeria* Hagani

These species are separated on the basis of microscopic characteristics and site of injury.

Of these 9 species, only the first 5 species are *Eimeria* pathogenic and all species except *Eimeria* bronchi have been reported from Iran.

Eimeria tenella is the most virulent and most important species of poultry coccidia that is controlled by coccidiostats to increase coccidiosis caused by *Eimeria nectatrix*. After *Eimeria* Neclatrix, *Eimeria* Bronti is highly pathogenic but rarely seen^[4].

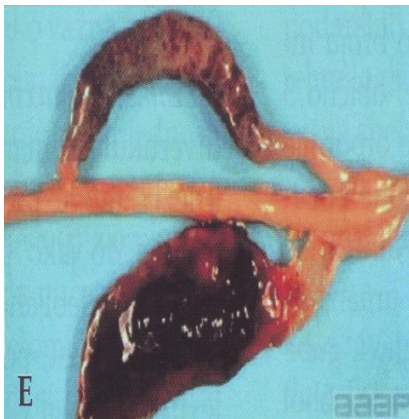


Figure 4: Swollen, intestinal blind intestine in poultry infected with *Eimeria tenella*

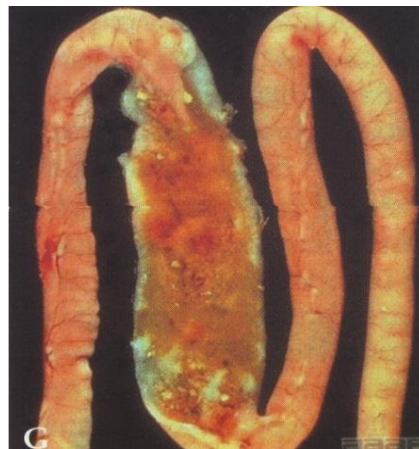


Figure 3: Ballooning and thickening of the mucosal layer by *Eimeria* Necatrix



Figure 2: A sample of broiler poultry suffering from coccidiosis Clinical

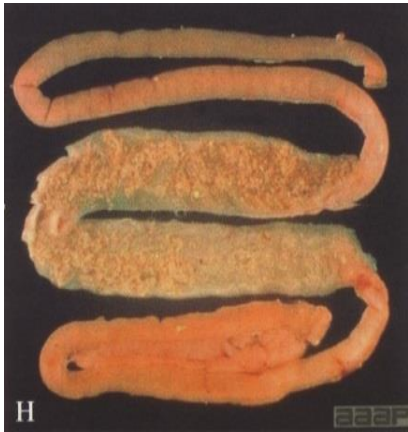


Figure 7: Intestine with intestinal enteritis and intestinal tract with fluid, blood and orange mucus Posted by Aimeria Maxima



Figure 6: Dry caspase necrotic membrane in *Eimeria bronchii* poultry

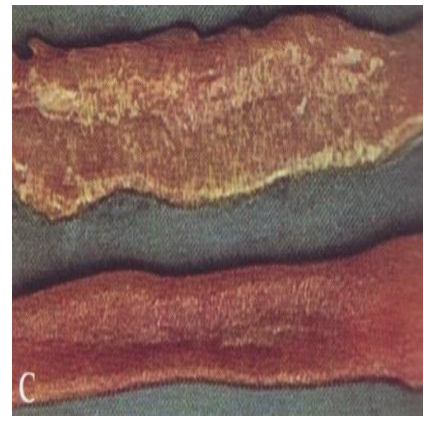


Figure 5: White plates transversely and ladder-shaped due to *Eimeria ascortina* infection



Figure 9: Epithelial cell involvement of the duodenum bending area due to *Eimeria peroxidase*



Figure 8: Serum intestinal surface due to *Eimeria miwati* can be seen as colonies

• **Life Cycle:**

Eimeria's life cycle is a self-limiting cycle. This means that after the developmental period, the parasite is excreted by the stool.

Each oocyte excreted through the stool contains a cell called the espront, and under appropriate environmental conditions for heat, moisture and oxygen, the oocyst undergoes a sporogonous or haggasic stage and reaches the infection stage. The diploid sporon undergoes a descending division and forms a refracted polar body [12], the spur directly divides into four sporoblasts, each of which subsequently becomes a sporocyst. Within each sporocyst, two sporozoites are formed [5]. The duration of these changes depends on the temperature, humidity and oxygen content, and in optimum conditions,

this period is 2-4 days. After this stage the oocysts become infected and can resume the life cycle if swallowed by the host. When the infected oocyst is swallowed by the animal, the wall of the oocyst is eroded and torn by gastrointestinal motions, especially the small intestines, and sporocysts exit the mature oocysts. The wall of sporocysts is destroyed by bile salts and chymotrypsin in the small intestine and the sporozoites are released and the released sporozoites enter the epithelial cells of the small intestine (lamina propria cells) and become trophozoites [10]. In other words, sporozoites either enter the epithelial cell directly or are located between the epithelial cells and are transmitted between cells by the lymphocytes. Sexual reproduction is called gametogeny and asexual reproduction is called schizogony. After replacement of the sporozoites, the schizological or asexual division begins [13].

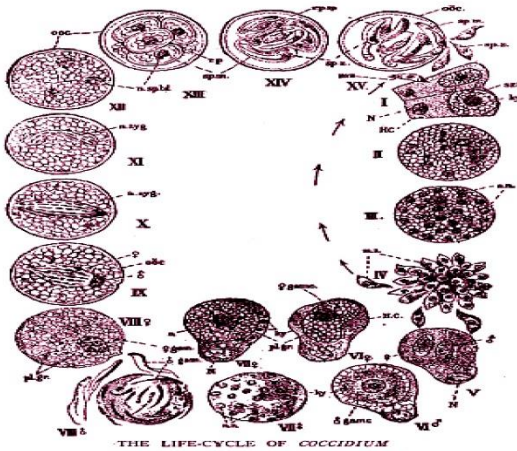


Figure 11: Eimeria sex and asexual life cycle and different stages of Eimeria life cycle

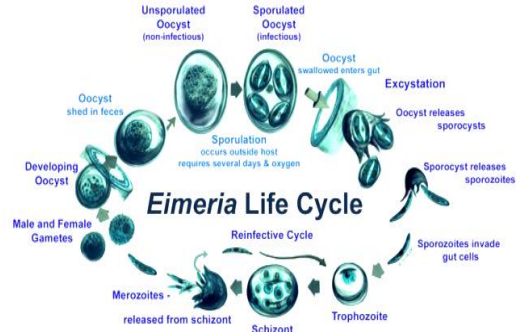


Figure 10: Different stages of Eimeria's life cycle

• **Eimeria parasitic infections in the host**

Eimerias are predominantly intestinal parasites of the gastrointestinal tract and intestines, but are also found in other organs such as the kidneys. Each type of imria is replaced in a specific location by the intestinal tract such as cecum, duodenum, ileum, etc. Areas attacked by different species of poultry are listed in Table 2-1. Some of these sites invade different areas of the intestine, such as villus epithelial cells, Liebknohen caves, and even mucosal cells. The location of the parasites within the host cell also varies, with some species located above the host cell nucleus, some below it, and even in some species some parasites within the host cell nucleus. Some species contribute to the size of the host cell to some extent, and some enlarge it, as well as the size of the host cell. Even if they are not attacked, the location of the parasites' replacement depends on the genetics of each species [3].

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Table 1: Distribution of Different Eimeria Species in Poultry [3]

The location of the parasite in the tissue sections	Eimeria species
Eimeria necatrix E.necatrix	second generation schizont layer below the epithelium
Eimeria maxima E.maxima	gametocyte sub-epithelium
Eimeria Asherolina E.acervulina	Epithelium
Eimeria Bronti E.brunetti	Second Generation Schizont Sub-Epithelium
Eimeria tenella E.tenella	Second generation schizont layer below the epithelium
Emeritus E.mitis	
Eimriaprea Cox E.praecox	Epithelium
Eimeria miwati E.mivati	Epithelium
Eimeria Hagani E.hagani	epithelium

Animals susceptible to coccidiosis and natural and experimental hosts of the disease

In addition to poultry and roosts are also found in turkeys, goats, ducks and pheasants, Quebec, parrots, pigeons, quails, and other birds, as well as in mammals such as cows, sheep, goats, pigs, rabbits, and other domestic animals. Poultry are the only natural host for their own coccidia species and they never infect other birds. There is no crossover between Eimeria species from poultry to other host species except in birds where there is a safety agreement. All poultry breeds are susceptible to infection at all ages but are restricted to reinfection due to the rapid development of immunity. Coccidiosis in poultry is most commonly seen in 3-6 weeks of age and rarely in birds less than 3 weeks old. Due to the short period of earlier exposure and the high bio-potency of the parasite, the oocyst in the bed initially increased rapidly, but gradually the bird was immunized by mild infections (no clinical signs). Like:

The high degree of environmental contamination in the early stages, the existence of suitable conditions for oocyst spores such as high bed moisture and high heat, the high number of swallowed infectious oocysts lead to the onset of disease and death in birds. Because under these conditions the bird will not be able to gain immunity against the parasite .

Breeding sheep and laying offsets are at greatest risk of being infected with *Coccidia* because they are kept in bed for 20 weeks or more.

Coccidiosis rarely occurs in laying flocks, but if the herd has not previously been exposed to coccidia species, the risk of coccidiosis is high ^[4].

Epidemiology

Coccidiosis is found almost everywhere in chicken and breeding grounds, so Coccidiosis exists in all countries around the world. However, herd depletion, lack of improvement and hygiene of the environment, and bed moisture increase the likelihood of an outbreak.

A very high percentage of positive herds from Europe have been reported, in addition to surveys conducted in North and South America indicate that coccidia are present in almost all broiler farms ^[2].

Although coccidiosis occurs in all domestic and wild birds, it is caused by any specific bird that does not cause disease in the other species, so wild birds are eliminated as sources of contamination ^[13] and thus most intermediates for the propagation of coccidia. These are mechanical factors ^[3] that are driven by people moving between farms.

The disease is most commonly seen in 3-6 weeks of age and rarely occurs in herds less than 3 weeks old. The self-limiting phenomenon of coccidial infections among chickens and other birds is well known. There is no cross-immunity phenomenon among different species of *Coccidia* and according to this there is a possibility of Coccidiosis by different species in several stages. There is an interaction between herd immunity acquired by mild infections (without clinical signs) and an increase in the parasite population. Immunogenicity usually occurs without clinical signs and oocyte production is reduced and the oocyst population in the substrate declines rapidly ^[8]. Obviously, before immunity in birds, the number of oocysts in the substrate increases rapidly due to the short pre-emergence and bioavailability of parasites.

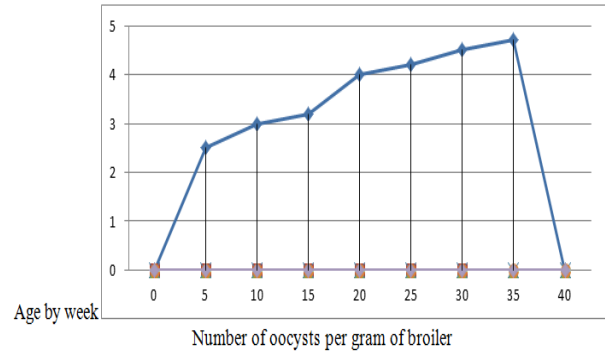


Chart 1: Number of oocysts per gram of broiler chickens during growth ^[8]

The number of casualties in winter coccidiosis below 2 months that have not previously been treated with *Eimeria* and have been suddenly exposed to severe infection is low. Most coccidiosis occurs in poultry in less than three weeks.

Sources of pollution:

The sources of contamination are the feces of the diseased or vector animals, and the infection enters the body by eating contaminated food and water or oocyst-impregnated litter. Fecal oocysts are non-infectious and have no virulence. These primary oocysts should be subjected to appropriate conditions, including temperature, oxygen and moisture, which will result in infectious oocysts. In fact, the primary oocyst becomes sporulated and becomes an infectious oocyst. Suitable temperatures for growth are 12-23 degrees Celsius and temperatures above 40 degrees Celsius destroy oocysts in a relatively short period of time. The oocysts usually die at temperatures of about 7-8 ° C. High and low dry and warm conditions, the sun's ultraviolet rays, and the presence of ammonia gas accumulated by the activity of bacteria and yeasts in the feces, kill oocysts. Eating infectious oocysts can lead to contamination but must be eaten in large numbers to cause disease and this is usually provided by continued re-infection and increased environmental contamination such as crowding in small environments such as denser halls.

Prone birds in a herd may enter the oocysts by tapping the bed, which is a normal and clear task for them. Although there is no intermediate host for *Eimeria* species, oocysts can be mechanically dispersed by various animals, such as insects and tools, and dust and poultry beetles are one of the mechanical agents of oocyst transmission in broiler chickens ^[8].

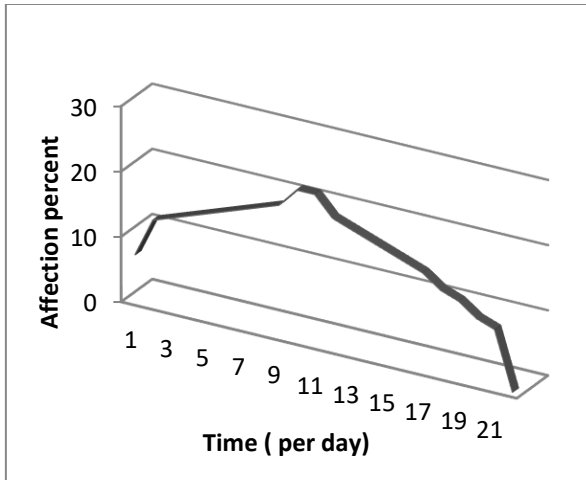


Chart 12: The incidence of broilers in chronic forms of coccidiosis.

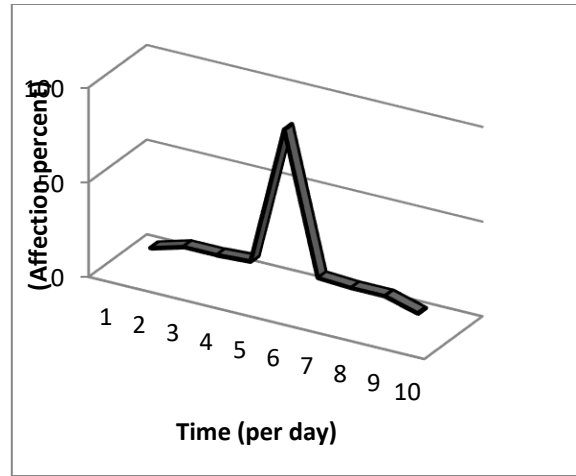


Figure 11: Chicken Broilers Acute Forms of Coccidiosis.

Effect of subclinical coccidiosis on growth rate and feed conversion ratio:

The damages caused by subclinical coccidiosis include:

- Overweight reduction
- Increase food conversion
- Reduced pigmentation

This disease increases the feed conversion ratio by 5 to 12%, equivalent to a loss of 60 to 120 g of body weight.

In some cases it has been observed that infected poultry may be 100 g lighter than the control (healthy poultry) at slaughter. With a bit of reflection and mathematical calculations we can see the extent of weight loss created. For example, in a 20,000-poultry farm in the event of a disease, at least 30% of the 6,000,000 birds will be affected. If we lose an average weight of 100 g per bird, we will have a 600 kg reduction in production over a period of time. It is about 2% of total production.

The extent of the lesions caused by this disease depends on the time it occurs, ie if the chickens become infected during the critical period of life (5 - 3 weeks), in the post-disease period until slaughter, production losses, non Will be compensated. Causes of disease impact on growth process and reduced feed conversion efficiency in broiler chickens, invasion of Eimeria in intestinal epithelium and extensive destruction of intestinal wall and bleeding and loss of body fluid and protein, which impact disease on growth and conversion efficiency. Food is listed in Figure 13 [13].

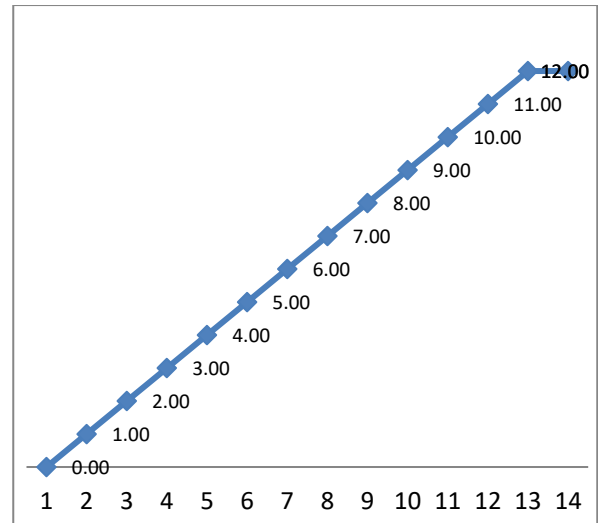


Chart 13: Diagram of the impact of disease on growth and food conversion efficiency [3]

CONCLUSION

It is much easier to prevent coccidiosis than to treat it. Certain chemicals called coccidiostats disrupt the protozoan life cycle. Coccidiostats are usually added to a certain percentage but not all coccidiostats have the same effect on all Eimeria species. Some drugs prevent the oocysts from excreting with the stool and thus preventing the contamination of the floor of the salon.

The most effective way to prevent coccidiosis is to prevent oocyst ingestion, especially adult or sporadic animal disease. Concerning the control of coccidiosis, it should be noted that only interruption of the life cycle of Eimeria species can prevent disease.

Today, the control and prevention of coccidiosis is based on three principles:

- Health and disinfection

- drug prevention
- Immunization and vaccination.

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