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Review Article

Development of immunization trials against *Eimeria* spp.

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ABSTRACT

Coccidiosis is a major intestinal disease affecting economically valuable livestock animals such as chickens and turkeys. Economic losses are associated with decreased productivity in afflicted animals. The different *Eimeria* spp. are the main etiologic agents for that virulent disease. The usefulness of prophylactic and therapeutic anticoccidial compounds has decreased in recent years due to the emergence of drug resistance in *Eimeria*, together with their possible toxic effect to the human consumers. Despite that, biosecurity and disinfection measures are the cornerstone to control the emergence of the pathogen, the immunization methods proved to be more practical and promising to prevent outbreaks due to coccidia. Since the early 1950s, several attempts were followed to formulate commercial immunotherapies, but up till now none proved to be sufficient. This review summarizes, classifies, and evaluates the trials performed to prevent avian coccidiosis, thereafter introduces an out of frame scientific strategy to find a solution for that emerging parasite.

Contents

1. Introduction

Animals as humans, suffer from diseases. Coccidiosis is a disease that affects a variety of wild type and domesticated vertebrates including chickens, turkeys, rabbits, cattle, sheep, goats, pigs, fish, and reptiles. The most virulent and economically important types of that disease are those infecting poultry and mammalian livestock. In poultry, the disease usually provokes severe intestinal disorders, diarrhea, dehydration, loss of weight, anorexia, and weakness. Therefore, obviously affects the animals husbandry, due to the reduced production it causes and the remarked mortality rate [1]. The main etiologic agents of coccidiosis in poultry are members of the genus *Eimeria*; which

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Table 1
The most important farm animal hosts of *Eimeria* species.

Host	*Eimeria* spp.	References
Chickens	*E. acervulina*, *E. brunette*, *E. necatrix*, *E. tenella*, *E. maxima*, *E. mitis*, *E. praecox*	[2,3]
Turkeys	*E. adenoids*, *E. meleagrimitis*, *E. dispersa*, *E. meleagridis*, *E. gallopavonis*, *E. innocua*, *E. subrotunda*	[2]
Rabbits	*E. stiedae*, *E. flavescens*, *E. intestinalis*	[6]
Cattle	*E. bovis*, *E. zuernii*, *E. alabamensis*, *E. auburnensis*, *E. brasiliensis*, *E. bukidnonensis*, *E. canadensis*, *E. cylindrica*, *E. ellipsoidalis*, *E. pellita*, *E. subspherica*, *E. wyomingensis*	[7]
Sheep	*E. crandallis*, *E. ovinoidalis*, *E. faurei*, *E. granulosa*, *E. intricata*, *E. pallida*, *E. parva*, *E. weybridgensis*	[7]
Goats	*E. arloingi*, *E. christenseni*, *E. caprina*, *E. hirci*, *E. ninakohlyakimovae*, *E. alijevi*, *E. aspheronica*, *E. caprovina*, *E. hirci*	[7]
Pigs	*E. debliecki*, *E. polita*, *E. scabra*, *E. spinosa*, *E. porci*, *E. neodebliecki*, *E. perminuta*, *E. suis*	[7]

are obligatory intracellular apicomplexan protozoan parasites that belong to family Eimeriidae [2]. The clinical disease depends on the ingested dose of the sporulated oocysts of *Eimeria* by susceptible fowl [3]. The different in-host stages of *Eimeria* spp. invade the cells of the intestine and duodenum (enterocytes), and replicate resulting in variable pathological changes. These changes range from local destruction of the mucosal barrier and underlying tissue, to systemic effects such as blood loss, and death [4]. Collectively those symptoms are associated with drop in egg production, impaired growth rate due to nutrients malabsorption in adults, necrotic enteritis due to *Clostridium perfringens*, and high mortality rates especially in young's [5]. Table 1 clusters the different species of *Eimeria* in groups according to their domesticated hosts. The table orders each group from the most virulent to least to that specific host.

The infection of poultry with *Eimeria* spp. (Fig. 1) begins when the host swallows the sporulated oocysts that excyst in the intestine and each releases 8 sporozoites (SZ). Once free within the intestine, the sporozoites penetrate the host's enterocyte in very short time, encapsulate themselves safely within a parasitophorous vacuole, and replicate. Merozoites (MZ) are then released in tremendous numbers, they rupture the host cell, and invade new ones. It has been proven that each *Eimeria* sp. is programmed genetically for a specific number of merogonic divisions. As much the parasite reinvades the cell, as much it damages the intestine and causes severe manifestations. After the last species-specific merogonic generation, merozoites invade the enterocytes to differentiate into male and female gametocytes (GAM). After fertilization the oocyst ruptures from the host's enterocytes and leaves the host with feces [2,8,9].

Experienced farmers noticed that lactating or previously infected animals show increased survival against the parasite for several months [10,11]. Despite the loss of an adequate full perception of immunity against coccidiosis, many studies underlined the immunological mechanisms that are active during primary and secondary infection of *Eimeria* spp. [12]. Hosts usually develop both immunological specific and nonspecific responses against the intracellular developmental stages of *Eimeria* spp. Specific responses include mucosal and circulating humoral response that initiates against the sporozoite, the merozoite, and the late sexual stages [4,13]. On the other hand, the gut-associated lymphoid

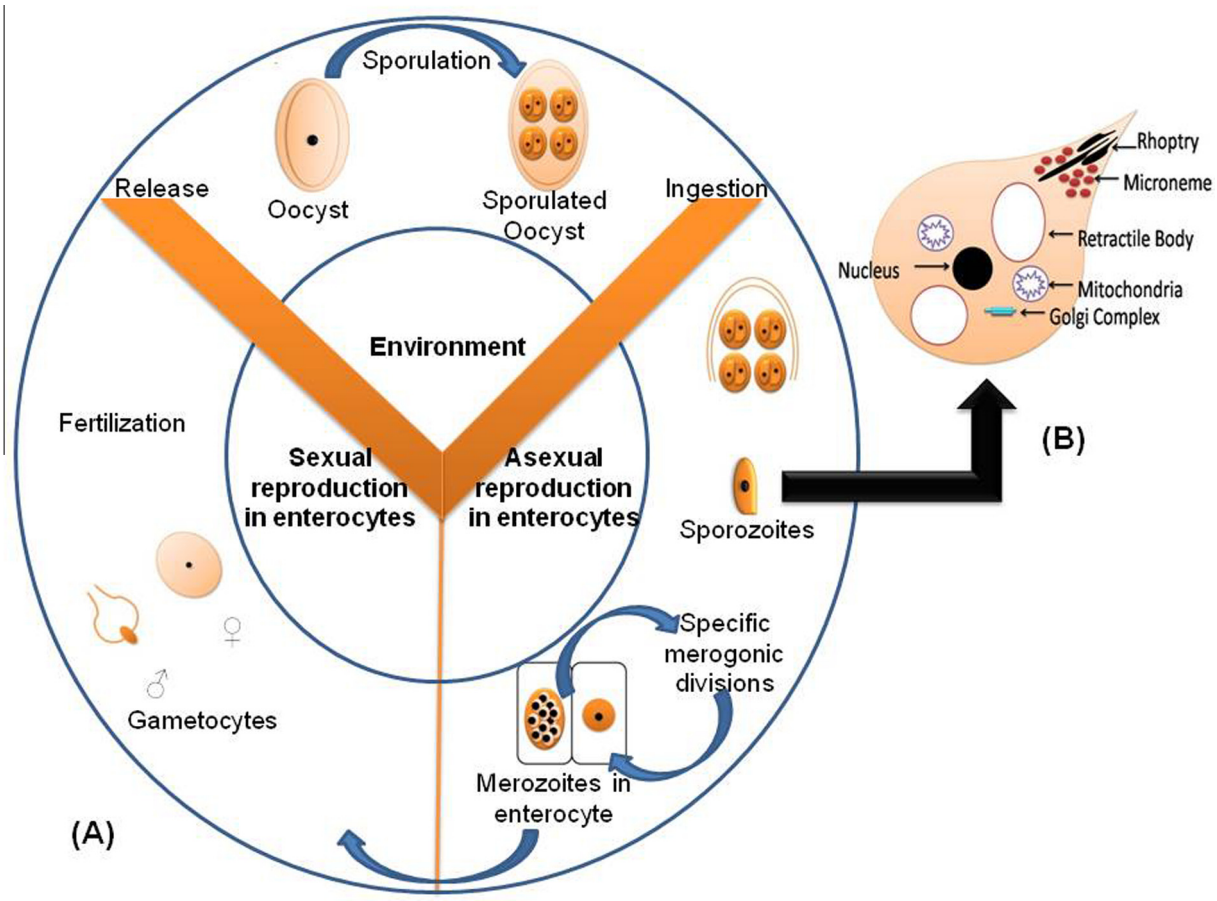

Fig. 1. (A) Diagrammatic life cycle of *Eimeria* and (B) simplified structure of *Eimeria* spp. sporozoite.