Ascaridia galli in Chickens: Co-Infection dynamics, effect of production, and mitigation Strategies: A review

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Abstract. In most countries where free-range and intensive farming are gaining importance, an increasing incidence of gastrointestinal nematodes is reported. Of all gastrointestinal nematodes, *Ascaridia galli*, a common gastrointestinal nematode in poultry, is a major risk to the health and performance of chickens, especially in semi-intensive and free-range systems. The complex dynamics of *A. galli* infections highlight that they predispose chickens to, or interact synergistically with, other pathogens, exacerbating clinical effects and compromising bird welfare. *A. galli* triggers innate and adaptive immune responses in the host, while chronic infections undermine the immune system and predispose the birds to secondary infections. The presence of *A. galli* negatively affects production traits such as growth rate, feed efficiency, and egg production, leading to significant economic losses. Some of the practices that have been mentioned to manage parasite infestation are rotational grazing, natural dewormers, improving biosecurity, tactical use of anthelmintics, and combined approaches to parasite control. Efficient and sustainable management strategies for modern poultry systems can only be achieved if we understand the multiple impacts of *A. galli*. This review aimed to critically assess and synthesise the latest research on *A. galli* co-infections in chickens, with emphasis on immunological mechanisms, effects on production, and the effectiveness of various control strategies. This review synthesises findings from peer-reviewed studies published between 2014 - 2024, identified through databases Scopus, Web of Science, Google Scholar, and ScienceDirect. This review highlights the current evidence and gaps, co-infection dynamics, the production effects, and the mitigation efforts, aims to support the development of improved, evidence-based practices for reducing the burden of helminth infections in commercial and smallholder poultry systems.

Keywords: *A. galli,* chicken production, co-infection, immune response, mitigation.

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1 Introduction

Chicken farming plays an important role in global agriculture, as it is a mainstay of food security and income generation, particularly in low- and middle-income countries. However, poultry farming continues to face persistent problems with parasite infestations affecting the health, welfare, and productivity of the animals. Currently, *AscaradiA. galli* infections are re-emerging in alternative poultry farming systems such as floor and free-range systems. In Manipur, India, a study found that 52.9% of chickens were positive for helminthiasis, with *Ascaridia spp*. being the most prevalent nematode. Studies have shown that egg production can decrease by 10–20% in infected chickens, with some severe cases reported to be as high as 30% [1].

To make matters worse, native chickens are often co-infected with other helminths, viruses, and bacteria in addition to *A. galli*. Ascariasis causes intestinal inflammation and mucosal erosion, which significantly impairs nutrient absorption and overall health. It is spread through contaminated feed, water, and fishing gear, and the infections tend to cause growth retardation, weight loss and even mortality, especially in immunocompromised or severely infected birds.

Our knowledge of ecology, the interactions with the immune system, and the dynamics of *A. galli* co-infection is still in a gap. There is an urgent need for integrated, sustainable, and science-driven interventions to manage the use of chemicals while maintaining the health and production of poultry. This review has attempted to summarise the current knowledge on *A. galli*, focusing on co-infections, impact on production, and new control strategies. Scientific studies were collected from well-known scientific databases such as Scopus, Web of Science, Google Scholar, and ScienceDirect. The search used precise terms such as "A. galli in poultry", "helminth infection in chickens", "gastrointestinal nematodes in chickens", "co-infection in poultry", "viral and bacterial infection", "immune response of poultry to parasites," and "anthelmintic resistance in chickens." More than 87 sources relevant to the study were reviewed and thematically categorised. The topics were mainly categorised into four broad categories: (1) co-infection patterns with *A. galli* and other poultry pathogens, (2) host immune mechanisms caused by parasitic infections, (3) impact on production and performance of affected flocks, and (4) current prevention, treatment, and control measures. The present study did not include primary data collection, such as field studies or experimental trials.

2 Co-infection dynamics *A. galli*

The effect of *A. galli* infections is generally augmented in co-infection with other pathogens, including bacteria, protozoa, viruses, or other parasitic genera, leading to more intense and complicated clinical signs. Immune system dynamics of co-infection may change the potential to mount an effective response, predisposing the chickens to secondary infections and complicating the course of already established diseases, as shown in Figure 1.

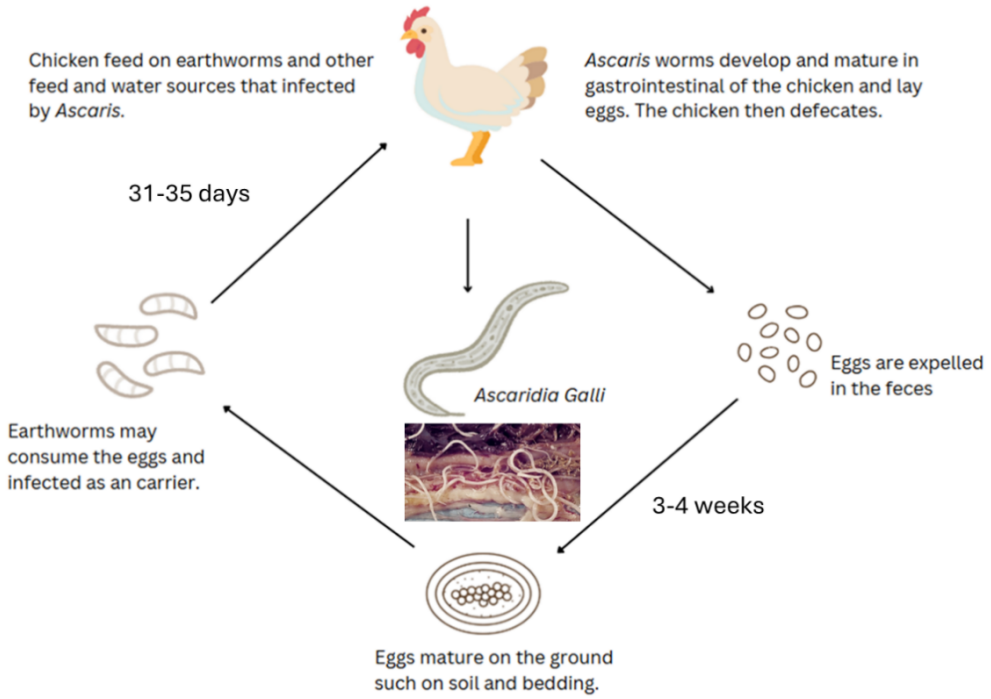

Fig. 1. Transmission and pathobiology of *A galli*. The parasitic eggs are shed in chicken faeces and become infective in 10–14 days. Chickens ingest infective eggs directly or via earthworms acting as transport hosts. Larvae hatch in the intestine, penetrate tissues, and develop into adults. Adults lay eggs in 31–35 days, continuing the cycle.

2.1 Co-infection with protozoa

A. galli infection along with *Eimeria spp. coccidiosis* (bloody faeces) in chickens may consequence from a concurrent infection, where both parasites coexist in small and large intestine in chicken. Co-infection with *A. galli* and *Eimeria spp.* can multiple intestinal injury, leading to malabsorption of nutrients, poor weight gain and mortality. A study in Romania found free-range chicken had the highest prevalence of mixed infections with *A. galli* and *Eimeria spp.* at 22.8%. The study emphasized the importance of these co-infections in their impact on poultry health. In a report from Tamil Nadu, India, on indigenous Aseel chickens, co-infection with *E. necatrix* and *A. galli* was documented. Affected chickens showed profound signs of lethargy, anorexia, brownish diarrhoea and death. [2] had earlier diagnosed co-infections with *A. galli* and coccidiosis in broilers, layers, and golden birds, evaluating them separately and together. Intestinal worms and coccidiosis caused huge losses at the 0.005 and 0.005 and 0.01 unit per bird units, respectively. The overall economic loss through treatment, prophylaxis and losses in production due to *A. galli* and coccidiosis was estimated at over $2.8 million. The huge losses were seen at farms of $0.005 and $0.01 per bird for intestinal worms and coccidiosis, respectively [2].

Both co-infections have been demonstrated to cause immunosuppression in the host. Excretory products of the larvae and adults of *A. galli* regulate the host's immune response, suppressing the bird's severe reaction to secondary infections, including coccidiosis. In addition, coccidiostats, like coccidiosis, suppress the immune system through the destruction of gut-associated lymphoid tissue (GALT) and intestinal mucosa. Destruction of the immune structures reduces immunity to *A. galli* infection. The presence of this parasite therefore supports the hypothesis that *A. galli* lives in an optimally regulated intestinal environment.

Both *Eimeria spp* and *A. galli* impose adverse effects on poultry development and growth. In a recent trial experiment carried out on 42 broiler chickens, it examined the effect of *A. galli* and *Eimeria* infections. These birds also recorded the lowest PCV levels from the 8th to the 12th day post-infection, which are indications of severe blood loss or anemia. With five out of seven birds dead, this flock also recorded the highest mortality rate. According to various studies, *A. galli* retards development by competing for nutrients and directly damaging the gut [3]. When both parasites infect a bird together, their impacts are synergistic, i.e., the overall intestinal damage exceeds the combined effects of the two parasites in isolation. Dual infection is also accountable for greater vulnerability to other secondary infections, which aggravates the adverse impact on growth and productivity [2]. Furthermore, the immune system against *A. galli* is generally ineffective, permitting chronic infestations.

2.2 Co-infection with bacteria

A. galli co-infection with bacterial pathogens is a key issue in poultry production. Chickens that are infected with *A. galli* have a heightened risk of contracting secondary bacterial infections. *A. galli* infection can devastate the intestinal wall, sever the intestinal barrier and thus allow *Salmonella spp*, *C. perfringens* or *E. coli* to travel into the bloodstream or intestines. Besides this, *A. galli* infections exert adverse impacts on chicken intestinal microbiota, thereby causing dysbiosis, which refers to a state in which the normally beneficial microorganisms that inhabit the body have instead turned pathogenic. That is, the normally beneficial microorganisms in the body have instead turned pathogenic. This has been made possible through the reality that the dysbiosis host carries unbalanced populations of gut microbes with them that are accompanied by an elevated risk of overgrowth of bacteria as well as the formation of opportunistic pathogenic bacteria. According to [3], the most notable concomitant infections in *A. galli* were bacterial co-infections with a prevalence of 59.24%, and there were significant differences regarding ages and seasons.

Examined the impact of mixed infections with *A. galli* and *E. coli* in poultry, the outcome revealed adverse interactions between the two pathogens since the co-infected groups that had *A. galli* had a very light worm burden when compared with the groups infected with *A. galli* only [4]. Furthermore, weight gain following co-infection was significantly reduced, showing an additional cost to host health and productivity. These observations stress the need for adequate integrated disease control measures in poultry farming to reduce the impact of such co-infections and intensify the secondary infection. The foundation of co infection events can be immune mechanisms that have the potential to suppress or revert the immune responses of chickens and hence increase the susceptibility to subsequent infections.

Studies show that high concentrations of *Salmonella* (*S. typhimurium*) reduce both the worm burden of *A. galli* and the egg yield in congenitally infected chickens [5]. This also means that Salmonella can suppress *A. galli* populations. On the contrary, infection with A. galli can prolong the duration of *S. typhimurium* infection in chickens, meaning that the presence of the nematode could maintain and facilitate the proliferation of the bacterium. *A. galli* has been shown to be a direct vector for the spread of *Salmonella spp*.

As [5] assessed an experiment where 32-day-old broiler chickens were further divided into three groups and maintained under control conditions to quantify the impact of *A. galli* co-infection on *S. typhimurium* colonisation. Coinfection, and T_{5000} specifically, greatly facilitated *S. typhimurium* colonisation with increased retention and secretion time in the host. In vitro, infections of *A. galli* have been found to shield *S. typhimurium* from inhibition by certain antibiotics such as cefepime, amoxicillin, and chloramphenicol [5]. These interactions are a risk to the treatment of salmonellosis in poultry because of the complexity introduced during treatment. They established five trial groups of chickens that had double infection of *A. galli* and *P. multocida*, single infection, and various orders of infections. They reported that chickens co-infected with *A. galli* and *P. multocida* exhibited the most

pronounced clinical signs, which were greater mortality, reduced weight gain and lower egg production. Mortality for these chicks was very high compared to the singly infected species. Meanwhile, chicks that were initially infected with *A. galli* and later with *P. multocida* were discovered to have a higher rate of lesions and a higher percentage of shedding of the bacteria. This is a clear indication that individuals infected with *A. galli* have a severely weakened immune system, making them susceptible to secondary infections with lethal bacterial infection.

2.3 Co-infection with virus

Co-infections with viral diseases like infectious bursal disease (IBD), avian influenza (AI) and Newcastle disease (ND) are also feasible with *A. galli* and aggravate the progression of the disease. Viral infections frequently induce immunosuppression, increasing the incidence of secondary infections or more serious manifestations of disease. Immunosuppression caused by *A. galli* can compound viral disease by devastating the host's capacity to generate protective antibodies or cell-mediated immunity [6]. The interaction of a viral infection and *A. galli* can culminate in amplified disease severity, prolonged recovery, and diminished productivity. In avian influenza, co-infection with *A. galli* can also aggravate the lesion in the respiratory and gastrointestinal tracts, causing systemic inflammation, worsening feed conversion, and resistance to secondary infection. Furthermore, *A. galli* can suppress the immune system of the host, as evidenced by decreased immune response to vaccination efficacy against Newcastle disease [7]. Free-range organic laying hens are more at risk of secondary bacterial infection and mortality because of viral immunosuppression.[5].

2.4 Co-infection with other helminths

Co-infections with *Heterakis gallinarum*, another common nematode in poultry, often occur due to shared transmission routes via contaminated litter, feed and water. Although *H. gallinarum* itself is not highly pathogenic, it acts as a vector for *Histomonas meleagridis*, the protozoan that causes blackhead disease. When chickens are infected with both *A. galli* and *H. gallinarum*, the immune response can be suppressed or diverted, leading to a more severe disease course and a higher parasite load. [4] found that in chickens co-infected with *A. galli* and *H. gallinarum*, over 80 % of the worms were expelled during the juvenile stage, primarily between 10–21 days p.i. Both local and humoral immunity were linked to the expulsion of worms where goblet cell hyperplasia and up-regulation of mucin-related genes (MUC2) occurred. Despite the high shedding rates, 10–15% of the worms matured, so that transmission is still possible.

In an experiment trial on 139 chickens conducted by [3], about 20.1% of the chickens were infected with *A. galli* and H. gallinarum indicating a high probability of co infection. The co-infection with a single nematode raised the chances of harbouring the other by 2.7 times. The positive correlation is explained by the fact that both parasites induce similar immune responses. The study also found an inverse correlation between *H. gallinarum* and *H. meleagridis*, a protozoan parasite transmitted by *H. gallinarum*, showing intricate correlations between co-infected parasites. Another common co-infecting pair of helminths are *Capillaria obsignata* and *Capillaria annulata*. Depending on the species, these parasites infest the crop, esophagus or small intestine and cause lesions, thickened mucous membranes and bleeding.

Cestodes such as *Raillietina tetragona* can also co-occur with *A. galli*, especially in extensive housing systems. Although *Raillietina* infections are less common, their presence can increase intestinal constipation and hinder the movement and colonisation of nematodes such as *A. galli*. Although the resulting co-infection does not directly increase mortality, it does contribute to chronic poor performance and reduced immunity. However, [6] reported co-infection of *Raillietina* and *A. galli* in one-month-old Aseel birds. The affected birds

showed decreased appetite, weight loss and sudden death. Routine microscopic examination of the intestinal contents and postmortem findings revealed gross lesions, including intestinal obstruction caused by *Ascaridia* worms and mucosal damage caused by *Raillietina,* together with liver necrosis, an edematous and hemorrhagic bursa, and congested lungs. The study concluded that this co-infection probably contributed to the observed mortality and severe organ pathology.

3 Performance of chicken on *A. galli* infection

Birds afflicted with ascaridiosis experience diarrhoea and anaemia. Hypoglycemia, elevated urates, thymus atrophy, stunted development, and decreased egg production are all commonly brought on by the worm. The main factor causing the atrophy of the breast and thigh muscles, which leads to weight loss, is heavy infection. Adult parasites, particularly helminths, can indeed cause intestinal blockages in severe infections, leading to decreased nutrient absorption and depletion of fat stores [6].

Studies showed that the higher the *A.galli* population in chickens would bring obvious changes than in lesser population of parasites on infected chicken [6]. Lighter birds have been found to have higher infection intensities and worm burdens than bigger birds. Furthermore, *A. galli* infestation is most common in young poultry whereby the time they are 2-3 months old, birds will usually become immune to low-level exposure. Because of the damage to the intestinal lining, birds that are not immune and are infested with many nematodes become less able to digest and absorb food. In cases where the infestation is severe, a high number of worms may potentially cause intestinal blockage.

A. galli infection has an impact on the welfare, behaviour as well as social status of chickens. Infections with *A. galli* have been shown to cause behavioural abnormalities and raise the risk of severe feather pecking and cannibalism, endangering the welfare of birds. In comparison to the control birds, ground pecking activity decreased compared to infected birds. After regular deworming, the infected birds slightly had the same behaviour as that control group of birds. Compared to the controls, chickens infected with *A. galli* spent more time in their nests during the prepatent and patent periods. This behaviour diminished with regular deworming, but it remained considerably higher than that of the control group [8].

Due to the short lifespan of broilers, determining the true prevalence of *A. galli* infection in this group is often challenging. However, in layers and broiler breeders, where birds are reared for longer periods, the parasite is more likely to establish chronic infections, leading to significant negative impacts on performance parameters such as body weight, feed efficiency, egg production, and overall health [8]. Performance impact due to *A. galli* infection in chicken is illustrated and summarised in Table 1. The feed conversion ratio worsens dramatically, rising from 2.0 to 3.5, indicating that infected chickens require more feed to gain the same weight. When *A. galli* infection precedes *Eimeria* infection, it can lead to severe outcomes, including reduced weight gain, low feed intake, and increased mortality [6]. Egg production decreased from 84 to 60% after 6 weeks of concurrent infection of *A. galli* with *P. multocida*. [5], Others have demonstrated no significant difference in egg production between non-infected hens and hens with a high prevalence of *A. galli* infection. The egg production also declines with infected hens laying only 2 eggs per week instead of 5, marking a 60% reduction. Additionally, the mortality rate increases from 2% in healthy birds to 15% in infected ones. Therefore, overall health scores drop from 9 to 4 on a scale of 1 to 10, reflecting a significant decline in the well-being of affected chickens [6, 9] as presented in Table 1.

Table 1. Estimated impact of A galli infection on key production parameters in layers and broiler breeders based on experimental and field studies.

Parameter	Layers chicken	Broiler Breeders chicken	References
Body Weight Loss	5–10% average body weight loss	5–8% decrease in body weight gain	[8]
Feed Conversion Ratio (FCR)	Worsens by 0.1–0.2	Worsens by 0.05–0.15	[10]
Egg Production	Reduced by 5–20% (daily egg production)	5–15% lower (hatching egg production)	[9]
Eggshell Quality	Thinner shells, increased breakage	Effects of Poor-quality Hatchability	[8]
Yolk Pigmentation	Malabsorption Can lead to pale yolks	Poor quality yolks: reduced chances of fertility	[8]
Nutrient Absorption Efficiency	10%–30% decreased for major nutrients (protein, fat)	10–25% reduced absorption of amino acids, vitamins	[8]
Mortality (indirect)	Slight increase (Due to secondary infections)	Mild increase (in compromised immunity only)	[9]
Growth Retardation (pullets)	Delayed maturity by 1–2 weeks	Delayed peak production onset	[8]

4 Mitigation efforts against *A. galli*

4.1 Farming system

Different housing systems for poultry, such as deep litter, floor, and cage systems, influence the risk of infection with *A. galli*. If litter moisture is maintained at 25–35%, parasite survival can be prevented. As ingestion of contaminated bedding is an important route of transmission, practices such as regular turning, drying, and replacement of bedding reduce the infection pressure. Moisture and humidity are critical factors for the development of parasites. Warm, humid conditions favour the embryonic development of *A. galli* eggs, while good ventilation and controlled humidity inhibit their life cycle. Stress due to high stocking density can also affect immunity and lead to a higher number of parasite eggs in the faeces.

A lower stocking density or adequate space per bird can therefore reduce the severity of infection. Free-range or pasture systems pose greater challenges due to constant exposure to contaminated soil and manure. However, rotational grazing can interrupt the life cycle of the parasite and reduce reinfection. A rest period of several months on the pastures reduces the burden of infective eggs, as the eggs of *A. galli* do not survive indefinitely [4]. Other preventative measures include drying runs in the sun, improving drainage, administering natural anthelmintics, routine faecal egg counts, and strategic deworming programs to improve bird health.

4.2 Effectiveness of different treatments and rotational drug classes

The treatment of *A. galli* infections in poultry relies on a variety of strategies, including chemical, probiotic, and herbal agents, each with different mechanisms, benefits, and limitations. A comparative evaluation of these approaches is essential to develop sustainable management plans that protect poultry health while reducing reliance on synthetic chemicals. In the past, chemotherapy in the form of insecticides, anthelmintics, and anticoccidials formed the backbone of parasite control. These substances were initially inexpensive and highly effective [6] and enabled large-scale poultry production systems. However, the high cost of developing and approving new drugs has limited the available options for helminth control.

A major problem is the increasing incidence of anthelmintic resistance (AR), caused by the repeated use of the same class of drugs and poor deworming practices. Resistant worm populations pass on survival traits, making future treatments less effective. This practice reduces selection pressure and delays the development of resistance by switching between drugs with different mechanisms of action. For example, switching between benzimidazoles (e.g., fenbendazole), imidazothiazoles (e.g., levamisole), and piperazine can help to avoid cross-resistance. A practical sequence could include fenbendazole (month 1), levamisole (month 3), and piperazine (month 5), followed by re-evaluation with the FECRT (Faecal Egg Count Reduction Test). If fenbendazole has an efficacy of ≥95% at month 7, it can be used again; otherwise, a switch to another class is essential. Therefore, while drugs remain the foundation of *A. galli* control, resistance risks require careful switching of drug classes, monitoring, and integration of non-chemical strategies to maintain long-term efficacy as discussed in Table 2.

Table 2. Comparison of Chemical, Probiotic, and Herbal Treatments for Parasite Management in Poultry

Comparison criteria	**Treatments**		
	Chemical [11]	**Probiotic** [12]	**Herbs** [13]
Mechanisms of action	Ivermectin and fenbendazole target the nervous system and metabolic process of parasites, leading to their rapid elimination.	*Lactobacillus* and *Bacillus* species help to maintain a healthy gut microbiome, which can inhibit the growth of *A. galli*.	Contain compounds with anthelmintic properties, such as tannins, saponins, and essential oils, which can disrupt the life cycle of parasites and promote gut health.
Effectiveness	These treatments are highly effective and provide quick results, often reducing parasite loads within days.	While probiotics may not eliminate parasites, it reduces the severity of infections and improves overall health.	Herbal remedies can vary significantly depending on the specific herbs and their preparation. It can reduce parasite loads, though they may not match the rapid efficacy of chemical treatments.

Cost	Low-cost dose but for repeated treatments, can accumulate expenses.	Generally, it is more expensive over time due to the need for continuous administration, but it may provide long-term benefits that offset the initial cost.	Cost-effective, especially if locally sourced. However, the variability in quality can affect their overall cost-effectiveness.
Resistance	A significant risk associated with chemical treatments is the potential development of resistance, which can undermine their efficacy over time.	Probiotics do not contribute to issues; thus, it is a sustainable option for ongoing treatments.	Herbal treatments are less likely to lead to resistance due to their diverse active compounds, providing a more holistic approach to parasite management.
Health impact	This can lead to adverse effects on liver function and disrupt the gut microbiome.	Contribute to better digestion and nutrient absorption while enhancing the immune system.	It can act as an inflammatory and antioxidant.

4.3 Improve detection and monitoring methods

Effective detection of *A. galli* infection is critical to maintaining poultry health and preventing disease transmission. Early diagnosis provides the opportunity to intervene in time, minimize production losses, and reduce environmental contamination for future flocks [2]. To achieve this, reliable and sensitive diagnostic tools are needed for long-term parasite control. Conventional surveillance often relies on faecal egg counts (FEC) to determine infection levels and guide interventions. Routine egg counts using the flotation or McMaster method remain popular as they are simple and inexpensive. These methods assume that faecal egg shedding correlates with adult worm burden [2]. However, without frequent monitoring, infections can go undetected until an advanced stage, making treatment difficult and costly [3]. A major disadvantage of conventional FEC methods is their low sensitivity during early infection, when the worms are not yet mature and not yet producing eggs. In addition, FEC cannot reliably distinguish *A. galli* from morphologically similar species such as *Heterakis gallinarum* [6]. This limitation poses a particular challenge in cases of mixed infections or low parasite loads, where egg counts may underestimate actual infection rates.

To overcome these challenges, molecular diagnostic methods have been developed. Polymerase chain reaction (PCR) and quantitative PCR (qPCR) allow the detection of parasite DNA in faeces or intestinal tissue and identify infections even before eggs are shed. These techniques offer higher sensitivity and specificity and allow for more accurate quantification of parasite load and better tracking of infection dynamics over time. The integration of advanced molecular tools into routine monitoring provides a more comprehensive approach that supports selective treatment strategies. This not only helps to reduce the unnecessary use of drugs but also prolongs the efficacy of available anthelmintics, thereby promoting sustainable parasite control.

4.4 Improve biosecurity and management

Biosecurity measures are crucial to prevent the spread of infectious diseases, including

parasitic infections, by reducing the use of antimicrobials and reducing the risk of resistance. Basic practices such as pest control, disinfection, and strict hygiene are essential for effective farm management. Structural measures such as fencing and regulated vehicle or passenger traffic also help to prevent the introduction of pathogens.

Parasite eggs can easily adhere to equipment, clothing, or surfaces, facilitating spread within and between farms [15]. Therefore, agricultural practices such as proper disposal of dead birds, exclusion of rodents and wild birds, provision of clean feed and water, and restricted access for outsiders are effective in reducing infection pressure. Ecological approaches, such as vector control and habitat management, complement these measures by addressing environmental conditions that favour the persistence of parasites. Interruption of the life cycle remains crucial, either by isolating birds from intermediate hosts or by reducing exposure to contaminated faeces. Regular hygiene, including removal of dead birds and decontamination of litter, further reduces parasite survival.

Modern improvements in aeration and irrigation systems have also improved litter conditions, limiting the sporulation and survival of *A. galli* eggs. As a result, exposure to intensive farming has decreased significantly in recent years. Preventing access to earthworm or roundworm eggs remains an additional protective measure, especially in floor housing, where control of intermediate hosts can significantly reduce the risk of ascariasis. Therefore, a combination of strict biosecurity, environmental management, and modern housing technologies offers the most sustainable protection against *A. galli* in poultry production as presented in Table 3.

Table 3. Coinfection Dynamics of A galli in Chickens with Other Pathogens

Pathogen Type	Example Pathogen (s)	Interaction with *A. galli*	Mode of Action / Immune Impact	Clinical/ Production Consequences	Ref.
Virus	IBDV NDV	Viral-induced immunosuppression enhances *A. galli* establishment and survival.	IBDV damages B-cells, lowering humoral immunity; NDV affects respiratory and systemic immunity.	Higher worm burdens, reduced response to dewormers, poor vaccine efficacy.	[7, 6]
Bacteria	*S. enterica* *C. perfringens* *E coli* *P. multocida*	Gut damage by bacteria may facilitate A. galli larval penetration and chronic colonization.	Synergistic gut damage and dysbiosis, mucosal barrier compromise.	Enteritis, septicemia risk, reduced weight gain, and increased mortality.	[5]
Protozoa	*E. tenella,* *E. acervulina* *H.meleagridis*	Eimeria induced intestinal damage promoters easier larval migration and reduced	Mucosal erosion by coccidia enhances *A. galli* larval establishment mutual aggravation.	Severe intestinal inflammation, diarrhea, feed inefficiency.	[3]

		immunity.			
Helminths	*H. gallinarum Capillaria spp.*	Competitive or synergistic Effects on the gut environment: *H. gallinarum* carries H. meleagridis.	Mixed helminth infections increase immune evasion, prolong infection periods.	Egg production drops, nutrient malabsorption , increased parasite persistence.	[4]
Mycoplasma	*M. gallisepticum*	No direct gut interaction, but systemic stress may enhance *A. galli* pathogenicity.	Chronic immune exhaustion leads to susceptibility to helminth colonization.	Poor growth rate, immune suppression, increased disease complex risk.	[1]

5 Conclusion

A. galli continues to be a serious problem for poultry health, especially on farms with higher exposure of birds to environmental parasites, such as free-range and semi-intensive farms. Its chronic infection potential, its interaction with co-infecting pathogens, and its ability to evade the immune system are evidence of its multifaceted role in the disease process in poultry. The resulting effects on growth, feed conversion, and egg production are associated with considerable economic consequences. Controlling *A. galli* infections requires a comprehensive and holistic solution - one that considers increased biosecurity, strategic administration of anthelmintics, rotational grazing, and sustainable alternatives such as natural dewormers. Prevention and long-term control must also focus on host immunity and parasite surveillance at the farm level. Comprehensive knowledge of the biology of *A. galli*, its interaction with the host, and environmental risk factors is crucial for the development of effective, sustainable control strategies tailored to modern poultry production systems.

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References

1. Chen, H., Cao, Z., Liu, M., Diamond, MS., & Jin, X. (2023). The impact of helminth induced immunity on infection with bacteria or viruses. Veterinary Research, 54(1), 87.
2. Rashid, M., Akbar, H., Bakhsh, A., Rashid, MI., Hassan, MA., Ullah, R., Hussain, T., Manzoor, S., Yin, H. (2019). Assessing the prevalence and economic significance of

coccidiosis individually and in combination with concurrent infections in Pakistani commercial poultry farms. Poultry Science, 98(3),1167-1175.

3. Mousa, MR., Attia, MM., Salem, HM., Al-Hoshani, N., Thabit, H., Ibrahim, M.A., & El Saied, MA. (2024). Coinfection of the gut with protozoal and metazoal parasites in broiler and laying chickens. Poultry science, 103(1),103227.
4. Stehr, M., Sciascia, Q., Metges, CC., Gauly, M., & Daş, G. (2018). Co-expulsion of *Ascaridia galli* and Heterakis gallinarum by chickens. International Journal for Parasitology, 48(13), 1003–1016.
5. Foka, ES., Yondo, J., Katte, B., Anangmo, CN., & Mbida, M. (2019). *Ascaridia galli* protects Salmonella typhimurium from antibiotics. South Asian Journal Parasitol (3),1-5.
6. Sharma, N., Hunt, PW., Hine, BC., & Ruhnke, I. (2019). The impacts of *Ascaridia galli* on performance, health, and immune responses of laying hens: New insights into an old problem. Poultry Science, 98(12), 6517–6526.
7. Pleidrup, J., Dalgaard, TS., Norup, LR., Permin, A., Schou, TW., Skovgaard, K., & Juul Madsen, HR. (2014). *Ascaridia galli* infection influences the development of both humoral and cell-mediated immunity after Newcastle Disease vaccination in chickens. Vaccine, 32(3), 383–392.
8. Tarbiat, B., Jansson, DS., & Höglund, J. (2022). Implementation of a targeted treatment strategy for the sustainable control of *Ascaridia galli* infections in laying hens. Veterinary Record Open 9(1), e37.
9. Tarbiat, B., Enweji, N., Baltrusis, P., Halvarsson, P., Osterman-Lind, E., Jansson, DS., & Höglund, J. (2021). A novel duplex ddPCR assay for detection and differential diagnosis of *Ascaridia galli* and Heterakis gallinarum eggs from chicken's feces. Veterinary Parasitology, 296,109499.
10. Schou, C., Filippova, M., Quattrocchi, A., & Karanis, P. (2020). The current status of protozoan parasitic diseases in Cyprus: A narrative literature review. Environmental Sciences Proceedings, 2(1), 61.
11. Rodenbücher, AL., Walkenhorst, M., Holinger, M., Perler, E., Amsler-Kepalaite, Z., Frey, CF., Mevissen, M., & Maurer, V. (2023). Pumpkin seeds, lemongrass essential oil and ripleaf leaves as feed additives for *Ascaridia galli* infected laying hens. Veterinary Research Communications, 47(2), 817–832.
12. Zhang, R., Yang, J., Wang, Q., Hu, D., Zhao, Q., Zhu, S., Qiao, Y., Zhao, F., Wang, Z., Wang, J., Yu, Y., Han, H., Hao, L., & Dong, H. (2024). Comparative Efficacy of Plant Extracts and Probiotics on Growth and Gut Health in Chickens with Necrotic Enteritis. Animals, 14(22), 3312.
13. Jamil, M., Aleem, MT., Shaukat, A., Khan, A., Mohsin, M., Rehman, TU., Abbas, RZ., Saleemi, MK., Khatoon, A., Babar, W., Yan, R., & Li, K. (2022). Medicinal Plants as an Alternative to Control Poultry Parasitic Diseases, 12(3), 449.
14. Khangembam, R., Czeglédi, L., & Vass, N. (2023). Routine microscopy examination of faecal samples as a tool for detection of common gastrointestinal parasites: A preliminary report from two Hungarian farms. Acta Agraria Debreceniensis, 1, 63–66.

15. Al-Alwani, SI., & Abid, H. (2023). Comparative Effect of *Ascaridia galli* Infection on the Two Laying Hen Lines on the Liver Function and Immune Response. Arch Razi Inst 78(3), 1065–1070.