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REVIEW ARTICLE

Indian Journal of Animal Research

Phytotherapeutic Control of Coccidiosis in Livestock and Poultry Animals: Harnessing Plant Heritage for Novel Drug Discovery-Ethnobotanical Insights and Modern Validation: A Review

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ABSTRACT

Coccidiosis, caused by *Eimeria* species, is a major parasitic disease affecting livestock and poultry, leading to significant economic losses. Conventional treatments are increasingly limited due to drug resistance and concerns over chemical in the livestock products. This review aims to identify and evaluate novel medicinal plants with established ethnobotanical relevance as potential alternatives for the treatment of coccidiosis. Drawing from traditional medicinal practices, plant species historically used for various are explored and assessed their phytochemical constituents, efficacy and safety profiles based on modern scientific evidence. Additionally, recent advances in drug delivery and formulation to improve bioavailability and therapeutic outcomes of plant-based are examined in the present review. The present study also addresses key challenges including standardization, formulation, validation method, mode of action and integration into current veterinary practices. The objective of this review is to provide a comprehensive, evidence-based assessment of plant-derived therapeutics for sustainable and effective coccidiosis management, bridging traditional knowledge with modern veterinary science.

Key words: Antidiarrheal, Anti-inflammatory, Anti-ulcer, Coccidia, Immunomodulatory.

Coccidiosis, caused by *Eimeria* protozoa, is a major parasitic disease impacting livestock and poultry worldwide (Saeed and Alkheraije, 2023). Infection occurs when animals ingest sporulated oocysts from contaminated feed or water, with each *Eimeria* species targeting specific intestinal epithelial cells (Haug *et al.,* 2008). After ingestion, sporozoites are released in the duodenum and invade the intestinal epithelium, starting a life cycle with asexual (merogony) and sexual (gametogony) phases (Conway and McKenzie, 2007). Asexual reproduction produces merozoites that spread the infection, while sexual reproduction forms gametes that fuse into zygotes and develop into oocysts. This rapid cycle allows a single oocyst to produce millions of parasites within 4 to 5 days (Pattison *et al.,* 2007). Pathogenic *Eimeria* infections cause hemorrhagic enteritis, bloody diarrhea, malabsorption, emaciation and stunted growth (Awais *et al.,* 2012; Ram *et al.*, 2019; Kaur *et al.*, 2019). These pathological changes reduce feed efficiency, delay sexual maturity and lower production, leading to economic losses through both mortality and subclinical effects. Disease severity depends on host, parasite and environmental factors (Reid, 1990). Its prevalence varies globally, reaching up to 98% in goats and 86% in calves and sheep in England and the USA (Keeton and Navarre, 2018), nearly 100% in Malaysian goats (Paul *et al.,* 2020), 78 to 79% in Ethiopian and Iranian goats and sheep (Dau *et al.,* 2021; Barre *et al.,* 2023; Hatam-Nahavandi *et al.,* 2023), 28.6% in India (Bangoura and Bardsley, 2020) and 39.8% in Amazonian livestock (Paul *et al.,* 2020). Management traditionally relies on synthetic anticoccidials, vaccination and plant-derived

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products. While coccidiocides and ionophores are cost-effective and widely used, rising drug resistance and regulatory restrictions, such as the Europian Union's ban on prophylactic additives (Council Directive 2011/50/EU), have prompted alternative approaches (Chapman *et al.,* 2010). Vaccination with wild-type or attenuated *Eimeria* strains offers partial protection and plant-based natural products are gaining attention as sustainable feed additives, though challenges remain in standardization, safety, mechanism elucidation and cost-effectiveness (Gokila *et al.,* 2014; Yang *et al.,* 2015; Murshed *et al*., 2023).

The rising global prevalence of coccidiosis and increased interest in natural therapies have driven research into plant-derived anticoccidial agents. Phytochemicals show promise as supplements to conventional treatments by providing immunomodulatory, anti-inflammatory and direct antimicrobial effects. The present review explores herb-based alternatives targeting coccidial infections, emphasizing anti-inflammatory, antidiarrheal, anti-ulcer and immunomodulatory actions. Evaluating traditional ethnobotanical knowledge offers valuable insights for developing plant-based strategies, potentially reducing dependence on synthetic anticoccidial drugs.

Source of literature and selection process

Coccidia infections cause diarrhea, ulcers, inflammation and immunosuppression in animals. The present study reviews plants with immunomodulatory, anti-inflammatory, anti-ulcer and anti-diarrheal properties, focusing on those cited in traditional Indian literature and validated by *in vitro* and *in vivo* studies. Although not yet tested directly against coccidiosis, these plants show potential as future phytotherapeutic agents. The present review integrates traditional Ayurvedic knowledge with modern research, considering only relevant English-language studies and excluding repetitive or unrelated data.

Sources of evidence

This review draws on both traditional and modern scientific sources. Key references include *The Ayurvedic Pharmacopoeia of India*, which details validated formulations and therapeutic uses, alongside contemporary studies sourced from Google Scholar, PubMed and Scopus. By combining traditional knowledge with evidence-based research, 48 medicinal plants were identified as promising candidates for potential treatment of coccidial infections.

Screening process and evidence mapping

After screening and removing duplicates, 48 medicinal plants from Indian traditional texts previously untested for anticoccidial activity were identified for their anti-inflammatory, antidiarrheal, anti-ulcer and immunomodulatory properties (Table 1). Their scientific relevance was verified through databases like Google Scholar, PubMed and Scopus, including only English-language articles with supportive evidence. Plants without data on these properties were excluded. Fig 1 shows the selection and screening workflow, while targeted keywords ensured retrieval of relevant literature for each activity.

Development of coccidial infection

Coccidiosis starts when animals ingest sporulated oocysts containing sporocysts and sporozoites. In the gut, the oocyst wall breaks, releasing sporozoites that invade intestinal cells. These sporozoites multiply asexually, producing many merozoites and repeat this cycle several times. Later, sexual stages form male and female gametes, which combine to create new oocysts that are passed in the feces (Saif, 2009). The life cycle of coccidia with environmental transmission and host-dependent developmental stages is shown in Fig 2.

Conceptual Framework for anti-coccidial activity of selected plants

Based on current literature, the intestinal epithelium and gut-associated immune components play a pivotal role in host–pathogen interactions during *Eimeria* infection. The major sites and mechanisms involved in the pathogenesis and potential intervention points are summarized and the mechanistic flowchart of plants exhibiting anti-coccidial effects is shown in Fig 3.

Invasion of intestinal epithelium and nutrient absorption

The intestinal epithelium, responsible for nutrient absorption, is also the site where *Eimeria* parasites invade and reproduce (Shivaramaiah *et al.,* 2014). The invasive sporozoites enter gut cells, multiply asexually (merogony) producing around 1,000 merozoites per sporozoite, repeating 2 to 4 cycles depending on the species. These merozoites then enter the sexual phase (gametogony), forming gametes that fuse into zygotes and develop into unsporulated oocysts, which are shed in feces (Lillehoj, 1998). This cycle damages the gut lining, weakens the host and aids parasite transmission.

Mucosal and cellular immune mechanisms

After *Eimeria* infection, the intestinal epithelium not only absorbs nutrients but also activates immune defenses (Shivaramaiah *et al.,* 2014). Gut-associated lymphoid tissue, including Peyer's patches, cecal tonsils and lamina propria lymphocytes, processes antigens, produces antibodies and stimulates B cells, T cells, Natural killer (NK) cells and dendritic cells (Lillehoj, 1998; Dalloul and Lillehoj, 2006). Additional defenses include mucus, antimicrobial peptides and a healthy gut microbiota (Zhao *et al.,* 2001). Cell-mediated immunity, especially $CD4^+$ and $CD8^+$ T cells producing Interferon-gamma (IFN-γ), is crucial for parasite elimination (Kim *et al.,* 2019; Arstila *et al.,* 1994; Lillehoj and Choi, 1998). Intraepithelial lymphocytes and NK cells provide early local defense, while dendritic cells bridge innate and adaptive immunity, activating T cells, reducing oocyst shedding and enhancing protection (Lillehoj, 1989; Cornelissen *et al.,* 2009; Schnitzer *et al.,*

Table 1: Potential anti-coccidial plants, major phytoconstituents and associated mechanisms.

Botanical name	Ayurvedic name	Utilized part	Key phyto-chemicals	Formulation method	Mode of action	Observed effects	Validation model	Reference
Coleus forskohlii	*Gandhira*	Root, Stem	Coleonol, coleosol, naphthopyrone and coleoforsin	Aquous, alcoholic, methanolic, ethanolic and chloroform extract	Protects cell membranes, prevents protein damage and inhibits COX-2 enzyme activity.	Anti-inflammatory	*In vivo*	(Menon and Latha, 2011; Kundur *et al.*, 2012; Philomina mary and Malarvizhi, 2022)
Erythrina indica Lam.	*Paribhadra*	Stem, bark	Apigenin and beta-sitosterol	Aquous and hydroalcoholic extract	Blocks neutrophil and monocyte recruitment and prevents protein denaturation.	Anti-inflammatory	*In vivo*	(Shah *et al.*, 2017; Wankhede *et al.*, 2009)
Nerium indicum Mill.	*Karavira*	Leaf, flower	Oleandrin	Alcoholic and hydroalcoholic extract	Inhibits nitric oxide production and PEG2	Anti-inflammatory synthesis.	*In vivo*	(Dey and Chaudhuri, 2015; and Balkan *et al.*, 2018)
Swertia chirata Buch. Ham	*Kiratatikta*	Whole plant	Xanthones, xanthone and glycoside	Ethanolic, alcoholic and hydroalcoholic extract	Inhibits tissue swelling and downregulates COX-2, NF-κB and MAPK pathways, leading to diminished serum proinflammatory responses	Anti-inflammatory	*In vivo*	(Das *et al.*, 2012; Hu *et al.*, 2019)
Cannabis sativa linn.	*Vijaya*	Leaf, stem	Cannabinols and tetrahydrocannabinol	Ointment, aquous and ethanolic extract	Suppresses NO and pro-inflammatory cytokines, blocks NF-κB/MAPK, reduces ROS and TNF-α, restores apoptosis balance, relieves edema, stabilizes mast cells, lowers oxidative stress, modulates ECS and protects epithelium.	Anti-inflammatory	*In vivo*	(Kim *et al.*, 2022; Wolińska *et al.*, 2025; Ambrose and Simmons, 2019; Cocetta *et al.*, 2021)
Angelica archangelica Linn.	Chanda	Root	Angelica lactone and linalool	Essential oil and aquous extract	Reduces IL-6, IL-4 and IL-13 levels and prevents protein denaturation.	Anti-inflammatory	*In vitro*	(Fraternale *et al.*, 2018)
Cedrus deodara	*Devadaru*	Stem bark, Heart wood	Thujopsene, α-cedrene and cedrol	Aquous extract and essential oil	Shows anti-inflammatory action by blocking COX-2, TNF-α and NF-κB, reducing swelling, stabilizing mast cells and inhibiting LOX.	Anti-inflammatory	*In vivo*	(Chen *et al.*, 2020; Rathor and Goyal, 1973; Shinde *et al.*, 1999a; Shinde *et al.*, 1999b)
Euphorbia neriifolia Linn.	*Snuhi*	Stem, leaves	Nerrifolin, Neriifoliene and euphol	Ethanolic and hydroalcoholic extract	Inhibits LPS-induced IL-6, TNF-α, IFN-γ, NO, COX and LOX production.	Anti-inflammatory	*In vitro*	(Chang *et al.*, 2023; Palit *et al.*, 2016; Palit *et al.*, 2018)
Plumbago zeylanica	*Citraka*	Root	Plumbagin	Hydroalcoholic and	Stabilizes red blood cell	Anti-	*In vitro*	(Thanigavelan *et al.*, 2014;

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linn.		bark.		dichloromethane extract	membranes and reduces paw edema by inhibiting prostaglandins.	inflammatory	and *In vivo*	Subramaniyan and Paramasivam, 2017)
Ficus lacor Buch.	*Plaksa*	Root	Beta-sitosterol and arabinose	Ethanol, petroleum ether, ethyl acetate and chloroform extract	Reduces inflammation	Anti-inflammatory	*In vivo*	(Sindhu and Arora, 2014)
Trianthema portulacastrum Linn.	*Varsabhu*	Whole plant	β-sitosterol, stigmasterol and their β-glucopyranosides, β-cyanin	Ethanolic extract	Inhibits inflammation by blocking COX and LOX, reducing prostaglandin and leukotriene production.	Anti-inflammatory	*In vivo*	(Kendri and Wari, 2015)
Saraca asoca (Roxb.) De. Willd	*Asoka*	Seeds	Apigenin, gallic acid, linolenic acid and tannins	Aqueous, methanolic and acetone extract	Reduces granuloma tissue weight.	Anti-inflammatory	*In vivo*	(Gupta *et al.*, 2013)
Achyranthes aspera Linn.	*Apamarga*	Leaves	D-Glucuronic acid	Ethanolic and alcoholic extract	Reduces edema by inhibiting inflammatory mediators in both early and late inflammation phases.	Anti-inflammatory	*In vivo*	(Semuel and Shabaraya, 2011)
Anacyclus pyrethrum DC.	*Akarakarabha*	Roots, seeds, leaves and capitula	Sarcosine, malonic acid and palmitic acid	Hydroalcoholic extract	Reduces release of serotonin, prostaglandins, bradykinin and histamine.	Anti-inflammatory	*In vivo*	(Jawhari *et al.*, 2020)
Aegle marmelos Corr.	*Bilva*	Leaves	Tannins, skimmianin and caryophyllene	Aquous, methanol acetone and chloroform extract	Reduces inflammation	Anti-inflammatory	*In vivo*	(Arul *et al.*, 2005)
Leucas cephalotes Spreng.	*Dronapuspi*	Whole plant	β-sitosterol, stigmasterol and lupeol	Methanolic, hexanes, dichloromethane, ethyl acetate methanol extract.	Reduces LPS-induced TNF-α and IL-1β production.	Anti-inflammatory	*In vivo*	(Baburao *et al.*, 2010; Patel *et al.*, 2015)
Asteracantha longifolia Nees	*Kokilaksa*	Whole plant	Myristic acid, palmitic acid and linoleic acid	Alcoholic, aquous, chloroform, and petroleum ether extract	Reduces edema	Anti-inflammatory	*In vivo*	(Muthulingam and Veerakumar, 2021)
Clitoria ternatea	*Aparajita*	Root	Rutin, quercetin	Ethanolic extract	Lowers inflammatory	Anti-	*In vivo*	(Swathi *et al.*, 2020)

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			and malvidin.		mediators.	inflammatory		
Withania somnifera Dunal	*Asvagandha*	Root	Alkaloids and withanolides	Hexane, chloroform, ethyl acetate and methanolic extract	Reduces inflammation	Anti-inflammatory	*In vivo*	(Uthirapathy, 2021)
Boerhaavia diffusa Linn	*Punarnava*	Whole plant	Punarnavine and rotenoids	Hexane, benzene, chloroform, acetone, ethanol, methanol and aquous extract.	Inhibits secretory phospholipase A2.	Anti-inflammatory	*In vitro* and *In vivo*	(Giresha *et al.,* 2017)
Symplocos racemosa Roxb	*Lodhra*	Bark	Phenols, flavonoids, saponins, tannins, glycosides and steroids	Ethylacetate, chloroform, n-butanol and aqueous extract.	Protects membranes and stabilizes cells	Anti-inflammatory	*In vitro* and *In vivo*	(Maitra *et al.,*2017; Ahmad *et al.,* 2014)
Cleome gynandra Linn.	*Ajagandha*	Leaf	Linolenic and arachidic acid	Methanolic extract	Reduces wet feces and total feces volume.	Antidiarrheal	*In vivo*	(Reddy *et al.,* 2015)
Momordica charantia Linn	*Karavallaka*	Leaf	Glycosides and momoridicine	Aquous extract	Reduces diarrheal stool count, slows gut movement and prevents intestinal fluid buildup.	Antidiarrheal	*In vivo*	(BR *et al.,* 2011)
Adhatoda zeylanica Medic	*Vasa*	Leaf	Vasicinol and vasicine	Leaf juice	Increases reabsorption of water.	Antidiarrheal	*In vivo*	(Sutare and Kareppa, 2009)
Pterocarpus marsupium	*Asana*	Stem bark, heartwood	Alkaloids, resin, gum kino and tannins	Ethanolic extract	Increases electrolyte and water absorption, preventing intestinal fluid buildup.	Antidiarrheal	*In vivo*	(Shridhar *et al.,* 2011)
Alstonia scholaris	*Saptaparna*	Aerial part, leaf	Alstoscholarisine K and ditamine	Methanolic extract	Inhibits high K-induced contractions with a spasmolytic effect similar to calcium channel blockers.	Antidiarrheal	*In vivo*	(Shah *et al.,* 2010)
Pongamia pinnata Merr	*Karanja*	Seed	Pongachromene, pinnatin and gamatin	Petroleum ether, ethanolic and aquous extract	Acts against microbial enterotoxins that cause diarrhea.	Antidiarrheal	*In vivo*	(Dahikar and Bhutada, 2017)
Butea	*Palasa*	Stem,	Coumarins, butrin	Ethanolic extract	Inhibits castor oil-induced	Antidiarrheal	*In vivo*	(Gunakkunru *et al.,* 2005)

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monosperma Kuntze		Bark	and isocoreopsin		diarrhea, PGE2-induced enteropooling and reduces gut motility.			
Cuminum cyminum Linn.	*Svetajiraka*	Seed	Eugenol and limonene	Aquous extract	Inhibits chloride channels, relaxes muscles and enhances water absorption.	Antidiarrheal	*In vivo*	(Sahoo *et al.*, 2014)
Cyperus rotundus	*Nagarmotha*	Whole plant	Dihydroatisine and heteratisine	Methanolic extract	Reduces diarrheal frequency and delays its onset.	Antidiarrheal	*In vivo*	(Uddin *et al.*, 2006)
Bauhinia variegata Blume	*Kanchanara*	Root, seed, stem, bark, leaf	Quercetin and apigenin	Alcoholic extract	Reduces gastric secretion, total acidity and free acidity.	Antiulcer	*In vivo*	(Rajkapoor *et al.*, 2003)
Indigofera tinctoria	*Nili*	Leaf	Indican	Ethanolic extract	Protects gastric mucosa from oxidative damage.	Antiulcer	*In vivo*	(Manimekalai *et al.*, 2018)
Sphaeranthus indicus Linn.	*Munditika*	Whole plant	Sterols	Ethyl acetate, alcoholic and chloroform extract	Reduces total acidity through antisecretory action.	Antiulcer	*In vivo*	(Malairajan *et al.*, 2013)
Panax ginseng Wall	*Ninjin*	Fruit, root	Panaxdiol, panaxtriole and oleanolic acid	Liquid ginseng extract	Enhances immunity by boosting neutrophil migration, phagocytosis and T-cell proliferation.	Immunomodulatory	*In vivo*	(Khare, 2008; *Panax ginseng*, 2009)
Murraya koenigii	*Surabhini-nimba, Nagalinga*	Leaf, fruit, flower	Coumarin, carbazole, alkaloids and flavonoids	Methanolic extract	Boosts phagocytic activity.	Immunomodulatory	*In vivo*	(Shah *et al.*, 2007)
Lagenaria siceraria Mol.	*Katu-tumbi*	Leaf, fruit	Cucurbitacin	Ethanolic extract	Enhances delayed-type hypersensitivity response.	Immunomodulatory	*In vivo*	(Deshpande *et al.*, 2008)
Terminalia arjuna Roxb.	*Arjuna*	Leaf, bark	Flavonoids and oligomeric proanthocyanidins	Aquous and Alcoholic extract	Boosts secondary immune response.	Immunomodulatory	*In vivo*	(Halder *et al.*, 2009)
Bauhinia variegata Linn.	*Kanchanara*	Root, bark, buds	Flavonoids, beta-sitosterol and lupeol	Ethanolic extract	Stimulates macrophages, supports neutrophil adhesion and increases antibody levels.	Immunomodulatory	*In vivo*	(Ghaisas *et al.*,2009)
Urena	*Naagabala*	Root,	Flavanoids	Methanolic extract	Enhances phagocytic	Immunomodul	*In vivo*	(Rinku *et al.*, 2009)

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lobata linn		flower			function.	atory		
Gymnema sylvestre R.Br	*Gurmaar*	Leaf	Sapogenins	Aquous extract	Stimulates phagocytosis, neutrophil movement and chemotaxis.	Immunomodulatory	*In vivo*	(Malik *et al.,* 2009)
Cordia superba Cham. and C. rufescens	*Shleshmaataka*	Leaf, fruit, bark	Alpha-amyrin	Methanolic extract	Activates nitric oxide production, IFN-γ secretion and LPS response.	Immunomodulatory	*In vivo*	(Costa *et al.,* 2008)
Picrorhiza scrophulariiflora Benth.	*Kutki*	Root	Iridoid and glycosides	Diethyl ether, ethyl acetate, Aqueous and methanolic extract	Inhibits classical complement activation, chemiluminescence and T-cell proliferation.	Immunomodulatory	*In vivo*	(Smit, 2000)
Heracleum persicum Desf. (Apiaceae)	*Golpar*	Fruit	Flavonoids and furanocoumarins	Aquous extract	Stimulates both antibody and cellular immunity and increases IL-6 secretion.	Immunomodulatory	*In vivo*	(Sharififar *et al.,* 2009)
Cissampelos pareira linn.	*Paatha*	Root	Hayatine and alkaloids	Aqueous and ethanolic extract	Enhances humoral and T-cell immunity, boosting antibody levels and immunoglobulin titers.	Immunomodulatory	*In vivo*	(Bafna *et al.,* 2009)
Abutilon indicum linn.	*Atibaala*	Whole plant	Flavonoids and triterpenoids	Aqueous and ethanolic extract	Boosts humoral immunity by increasing immunoglobulins and strengthens innate immunity *via* neutrophil adhesion.	Immunomodulatory	*In vivo*	(Dashputre and Naikwade, 2010)
Chlorophytum borivilianum Sant. F	*Safed musli*	Root	Sapogenins	Ethanolic extract	Enhances DTH response, neutrophil adhesion, phagocytosis and carbon clearance.	Immunomodulatory	*In vivo*	(Thakur *et al.,* 2006)
Hibiscus rosa sinensis linn.	*Japaa*	Flower	Cyclopropanoids	Hydroalcoholic and ethanolic extract	Stimulates both humoral and cell-mediated immunity by enhancing phagocytic activity.	Antidiarrheal, anti-inflammatory and immunomodulatory	*In vivo*	(Gaur *et al.,* 2009)
Asparagus racemosus willd	*Satavari*	Root leaf	Beta-sitosterol, glycosides and saponin	Ethanolic extract	Inhibits TNF-α production in THP-1 cells.	Antiulcer, immunomodulatory, antidiarrheal	*In vitro* and *in vivo*	(Plangsombat *et al.,* 2016; Battu and Kumar, 2010)

COX-2- Cyclooxygenase-2; NF-κB- Nuclear factor kappa-light-chain-enhancer of activated B cells; MAPK- Mitogen-activated protein kinase; ROS- Reactive oxygen species; TNF-α- Tumor necrosis factor-alpha; ECS- Endocannabinoid system; IL-6- Interleukin-6; IL-4- Interleukin-4; IL-13- Interleukin-13; TNF-α- Tumor Necrosis Factor-alpha; LOX- Lipoxygenase; LPS- Lipopolysaccharide; PGE2- Prostaglandin E2; DTH- Delayed-type Hypersensitivity; IFN-γ- Interferon-gamma; THP-1- T-helper cell leukemia/monocyte 1 (human monocytic cell line); K^+- Potassium (used in context of high K^+- Induced contractions)

2010; del Cacho *et al.,* 2012; Sheridan *et al.,* 2013; Shoaib *et al.,* 2017).

Cytokine and chemokine regulation of immune dynamics

Cytokines and chemokines are crucial for immunity against *Eimeria*, affecting both protection and disease severity (Hong *et al.,* 2006). Early responses of IFN-γ and IL-10 enhance resistance, while delayed responses increase susceptibility (Bremner *et al.,* 2021). IFN-γ inhibits parasite growth and promotes Th1 type response (Dimier *et al.,* 1998) and pro-inflammatory cytokines like IL-1β and IL-18 trigger inflammation and further IFN-γ production

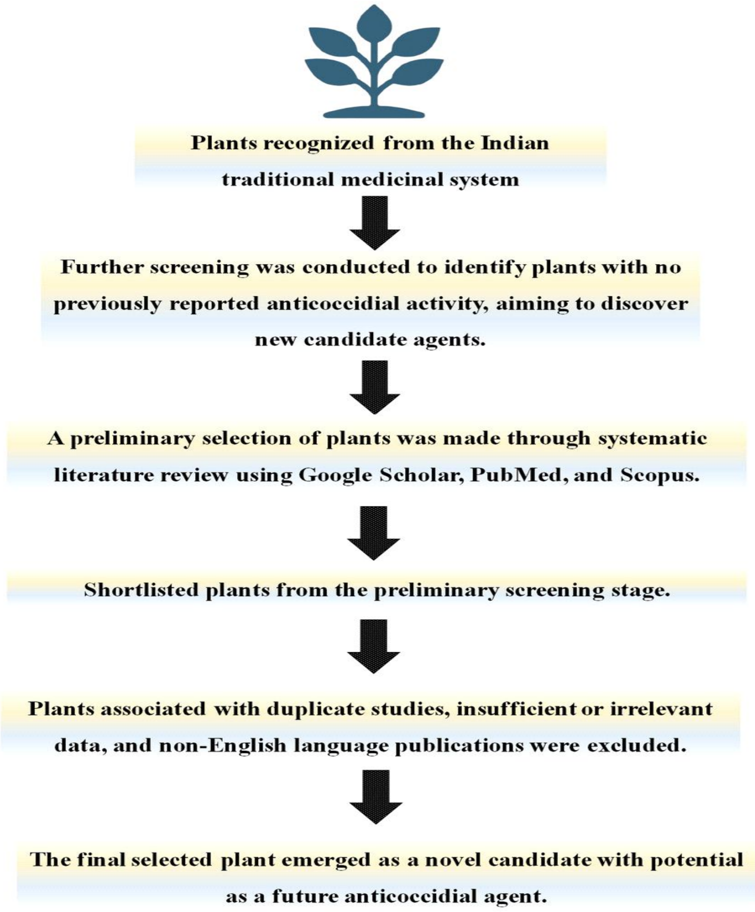

Fig 1: A stepwise flow outlining major steps of the review process.

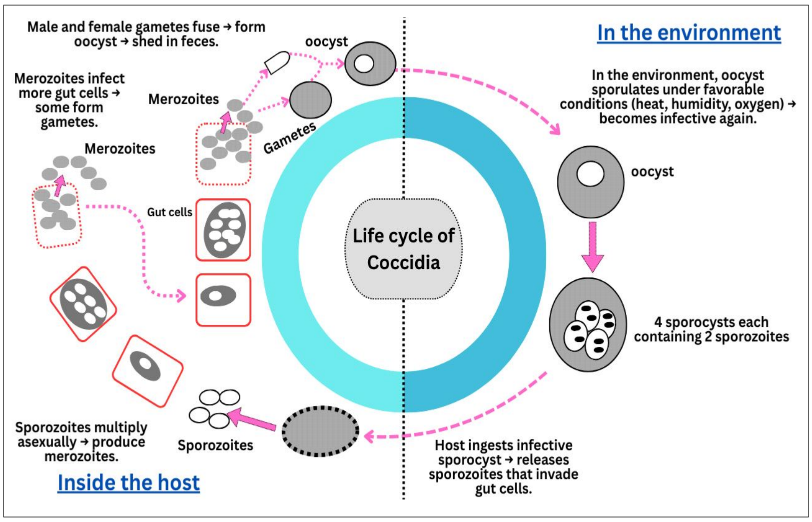

Fig 2: Life cycle of coccidia: Environmental transmission and host-dependent developmental stages.

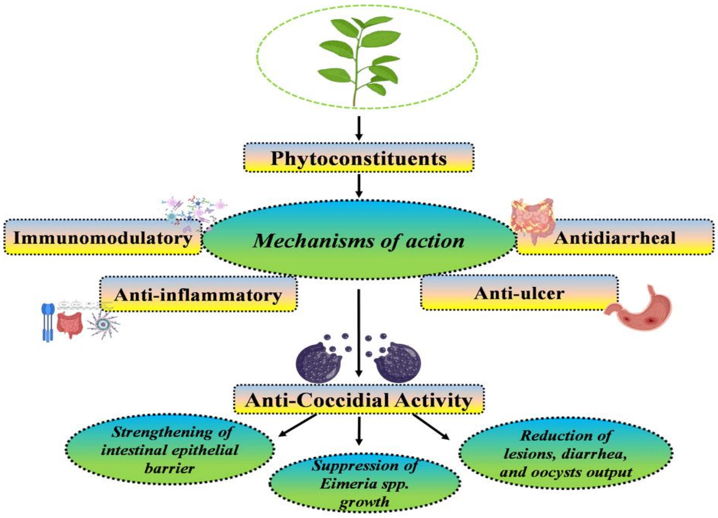

Fig 3: Mechanistic flowchart of plants exhibiting anti-coccidial effects.

(Hong *et al.,* 2006; Dalloul *et al.,* 2007). IL-2 activates T and NK cells against *E. acervulina* (Ding *et al.,* 2004) and IL-6 supports B cell differentiation and antibody production (Hong *et al.,* 2006). IL-10 controls inflammation but can suppress Th1 responses, aiding parasite survival (Rothwell *et al.,* 2004). TNF-α recruits neutrophils and drives tissue inflammation during primary infection (Zhang *et al.,* 1995), while chemokines such as MIP-1β and K203 guide immune cells to infection sites for effective gut defense (Laurent *et al.,* 2001).

Microbial dysbiosis and compromised gut integrity

The gut microbiota is vital for immune balance, metabolism and protection against pathogens. *Eimeria tenella* infection disrupts the cecal microbial community, causing dysbiosis (Huang *et al.,* 2018; Macdonald *et al.,* 2017). Beneficial bacteria like *Lactobacillus* and *Faecalibacterium* decline, while harmful bacteria such as *Clostridium and Escherichia-Shigella* increase (Chen *et al.,* 2020; Cui *et al.,* 2017). Loss of *Faecalibacterium*, a major butyrate producer, weakens mucosal integrity and anti-inflammatory responses, worsening gut damage and delaying recovery (Chen *et al.,* 2020).

CONCLUSION

The present review highlights medicinal plants with anti-inflammatory, antidiarrheal, anti-ulcer and immunomodulatory properties, emphasizing their potential as natural treatments against coccidial infections. These “phyto-coccidial” agents, noted in traditional Indian texts, may target multiple stages of the parasite’s life cycle. Although largely unexplored for anticoccidial use, they offer promising candidates for future research aimed at developing safe, plant-based therapies for coccidiosis, presenting a novel direction for drug discovery and disease management.

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Disclaimers

The views and conclusions expressed in this article are solely those of the authors and do not necessarily represent the views of their affiliated institutions. The authors are responsible for the accuracy and completeness of the information provided but do not accept any liability for any direct or indirect losses resulting from the use of this content.

Informed consent

Not applicable.

Conflict of interest

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