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Microbiome Dysbiosis in *Balantidium coli* Zoonotic Infections: Emerging Mechanisms and Host Implications

Azhar Abdulsada Neamah[1] , Khilood Hamdan Fahad[2] , Jenan Nadhim Sadeq[3]
https://orcid.org/0000-0001-9481-6972[1], https://orcid.org/0000-0002-2614-2387[2]
https://orcid.org/0000-0002-6350-6311[3]
Department of Microbiology, College of Veterinary Medicine, University of Al-Qadisiyah, Iraq

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Correspondence:
AzharA.Neamah
azhar.neamah@qu.edu.iq
Jenan Nadhim Sadeq
jinan.sadeq@qu.edu.iq

Abstract *Balantidium coli* is a rare protozoan that infects humans. It is the only known ciliate parasite in people. The infection often comes from pigs or primates. It spreads through food or water that has been contaminated. Some people have no symptoms. Others may get diarrhea, dysentery, or long-term bowel problems. Studies show that B. coli can change the gut microbiome. Helpful bacteria like Bacteroidota and short-chain fatty acid producers may decrease. Harmful bacteria such as Campylobacterota and Brachyspira may increase. These shifts can harm the gut lining and trigger inflammation. The parasite eats bacteria, damages te gut wall, and changes nutrient levels. The immune system's reaction can also promote harmful bacteria. These changes can remain after the infection clears, raising the chance of future illness. This imbalance can weaken the gut barrier. Bacteria may then pass into the blood. The balance of short-chain fatty acids may also change, affecting colon health and metabolism. Chronic cases may cause poor nutrient absorption. Checking the gut microbiome could help diagnose the infection. It could also guide treatment. Using antiparasitic drugs with probiotics or prebiotics may help restore balance. Understanding how B. coli affects gut bacteria could lead to better ways to prevent and treat the disease in people and animals.

Keywords: *Balantidium coli*, dysbiosis, gut microbiome, pathogenesis, zoonosis

Introduction *Balantidium coli* is a zoonotic protozoan of medical and veterinary concern. It is the only ciliated protozoan known to infect humans. It also infects pigs, camels, ruminants, and horses (1). The parasite lives mainly in the large intestine (2-5). There, it can cause a disease called balantidiasis. Human infections are uncommon but can occur in tropical and subtropical areas. Cases have also been found in cooler climates, showing the infection is not limited to warm regions. The main source is the domestic pig. Other animals, such as non-human primates, can also transmit the parasite. Poor hygiene, unsafe water, and close contact with infected animals increase the risk (6).

Infection may cause no symptoms or lead to diarrhea, dysentery, and abdominal pain. Severe disease can result in ulcers in the colon. Rarely, the parasite spreads beyond the intestine. Studies show that living near pigs greatly increases the chance of infection. In some groups, more than 16% may be infected. The parasite is also found in camels, where it may add to other parasitic infections. It is often spread through contaminated water. Its cysts can survive in the environment for long periods, making it a persistent waterborne threat (7,8).

In many regions, B. coli remains an overlooked pathogen. In pigs, it can cause diarrhea and worsen co-infections. In humans, rare deaths have been reported, usually due to late diagnosis or other illnesses. Learning more about its biology, spread, and health effects is important for prevention and treatment. This is especially true in areas with high human–animal contact and poor sanitation (9).

In both humans and animals, infection with B. coli can disrupt the normal gut microbiome. Studies show that this protozoan feeds on intestinal bacteria, which changes the microbial community (10). Loss of beneficial microbes such as Akkermansia muciniphila may weaken mucosal barriers and immune regulation (10). This bacterium is linked with gut health, metabolic stability, and resistance to inflammation. Reduced levels have been reported in several intestinal and systemic diseases, including parasitic infections.

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Evidence from other parasites supports this link between infection and microbiota disruption. Chronic Opisthorchis viverrini infection alters gut bacterial composition and may contribute to cholangiocarcinoma risk (11). Helminth infections in livestock, such as Trichuris suis, can shift microbiome communities and metabolic pathways, affecting nutrient absorption and immune balance (12). These changes can also influence other co-infections and disease outcomes (13, 14). Such findings suggest that B. coli, like other intestinal parasites, may trigger dysbiosis through both direct and immune-mediated effects.

Waterborne protozoa, including B. coli, are known to survive in contaminated sources and interact with diverse microbial species (13). Infections with Trypanosoma cruzi in Chagas disease have shown microbiota alterations in both human and animal hosts (15). These cross-kingdom interactions can promote pro-inflammatory bacterial growth and reduce beneficial taxa. In B. coli infection, dysbiosis may increase gut permeability, enable pathogen translocation, and worsen symptoms. This link between microbiome shifts and pathogenesis highlights the need for targeted studies to clarify mechanisms and guide therapy.

Epidemiology and Life Cycle

Balantidium coli is the only ciliate protozoan that infects humans and several domestic and wild animals. Pigs are the main reservoir, but infection also occurs in camels, cattle, and non-human primates (1,3,6). The parasite has been reported worldwide, especially in tropical and subtropical areas where sanitation is poor (1,2,3). Outbreaks have been linked to rural communities, areas with high pig density, and natural disasters that disrupt water systems (3,7). Though rare in humans, cases continue to emerge, including in non-endemic or cooler regions (2). These patterns highlight its persistence and adaptability in diverse climates.

Transmission occurs mainly by the fecal–oral route. Infective cysts are shed in the feces of reservoir hosts and contaminate water, food, or hands (1,7). Consumption of unwashed vegetables irrigated with contaminated water is a common source (7). Direct contact with pigs or other infected animals also plays a role in zoonotic spread (6). Populations living in close proximity to livestock and using untreated water sources are at higher risk (5). High infection rates, exceeding 16%, have been reported in some rural areas with frequent pig contact (5). Such settings favor continuous circulation of the parasite between animals and humans.

After ingestion, cysts pass through the stomach and excyst in the small intestine. The released trophozoites migrate to the large intestine, especially the cecum and colon, where they multiply by binary fission (3,8). They feed on bacteria, debris, and intestinal mucosa, causing tissue damage and inflammation (8). In severe cases, trophozoites invade the mucosa, leading to ulcer formation, hemorrhage, and sometimes systemic spread (9). Extraintestinal invasion is rare but can be fatal (9). The parasite's ability to survive in the environment and colonize multiple hosts makes it a continuing threat to both public and veterinary health.

Hosts: Pigs, Primates, Humans

Balantidium coli infects different mammals, but pigs are the main reservoir (1). Domestic pigs often carry the parasite without showing signs. This carrier state supports long-term environmental contamination. The infection is common in pig-farming areas, where human contact with pig feces is more likely (1).

Non-human primates are also natural hosts (3). They can maintain the parasite in forest and captive settings. Contact between humans and primates can lead to transmission, especially in rural or wildlife-adjacent communities. Cases have been found in both endemic tropical regions and cooler climates, showing the adaptability of the parasite (2,4).

Human infection is uncommon but still important. It is usually linked to water or food contamination. In rural regions near pig farms, prevalence in humans can be over 16% (5). In camels, B. coli is one of several neglected zoonotic parasites in dry areas (6). Its ability to infect many hosts raises the risk of cross-species transmission. This also makes control harder. Knowing the role of each host in the life cycle is vital for prevention and public health planning

Transmission through Food and Water

Balantidium coli is spread mainly by the fecal–oral route (1). The cyst is the infective stage. It is shed in the feces of pigs, primates, and infected people. Cysts can survive for weeks in moist soil or water (7). This durability supports ongoing spread in places with poor sanitation (1).

Contaminated water is a major source of infection (7). People can ingest cysts when drinking untreated water or eating food washed with it. The risk is high in rural areas where animals and humans share water sources (1,5). In some pig-farming communities, human infection rates are much higher (5).

Foodborne cases occur when hygiene is poor during food handling (16). Vegetables grown in soil fertilized with pig manure can carry viable cysts. B. coli has also been detected in camel feces, showing its presence beyond pig reservoirs (6). Outbreaks

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have been reported after floods and other disasters when water supplies are contaminated (3,7). This pattern is similar to other waterborne protozoa, such as Cyclospora and Blastocystis, which also spread through contaminated water and cause intestinal illness (17). Understanding these routes is key for prevention.

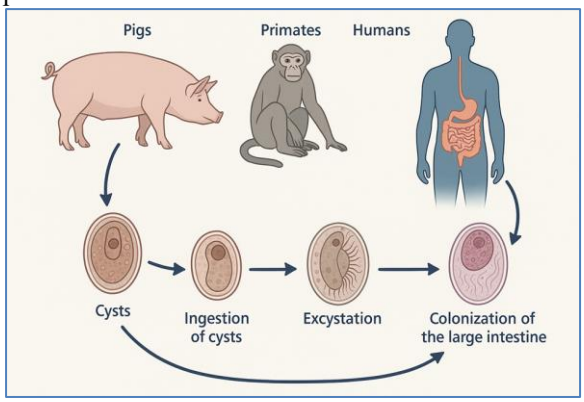

Figure 1: Life cycle of *Balantidium coli*. The diagram shows the major hosts (pigs, primates, humans) and key developmental stages of B. coli. Infective cysts are excreted in the feces of reservoir hosts and can survive in moist environments. Transmission occurs through ingestion of contaminated food or water. In the host, excystation occurs in the small intestine, releasing trophozoites that migrate to the large intestine. Trophozoites multiply by binary fission and may invade the mucosa, causing disease. Some trophozoites encyst before being shed in feces, completing the cycle.

Microbiome Alterations

Infection with *Balantidium coli* can cause marked disturbances in gut microbial communities. Similar to other intestinal protozoa and helminths, this parasite interacts with the host microbiota directly and indirectly. It can feed on bacterial populations, alter nutrient availability, and damage mucosal surfaces, leading to a shift in bacterial composition (18). Changes often include a drop in beneficial commensals and an increase in potentially pathogenic species. Evidence from protozoal infections, such as Giardia spp., shows that altered microbiota composition and function are common in enteric diseases and can influence disease severity (18).

Microbiome alterations in parasitic infections are also influenced by immune responses. The gut microbiota shapes and is shaped by the host's innate and adaptive immunity (16). During parasite colonization, inflammation, oxidative stress, and epithelial damage can create an environment favoring opportunistic bacteria (14,17). This interplay is seen in malaria, where Plasmodium modifies the microbiota of both vector and human hosts (17). For B. coli, similar processes likely contribute to dysbiosis, which can persist beyond the active infection phase and influence long-term health outcomes (15).

Loss of Beneficial Bacteroidota

A key sign of B. coli-related dysbiosis is the loss of bacteria from the Bacteroidota phylum. This group includes species that digest complex carbohydrates and produce short-chain fatty acids. These metabolites help maintain the mucosal barrier and control inflammation (13). When these bacteria are lost, gut integrity can weaken. This makes the host more open to secondary infections and chronic inflammation (15). Similar effects occur in helminth infections, where beneficial fermenters are reduced, affecting both nutrition and immunity (15,16).

The decline of Bacteroidota can also harm overall health. Their metabolites support immune balance, metabolic stability, and even brain function (15). Loss of these bacteria has been linked to metabolic disease, poor cognition, and more inflammation (13,15). In B. coli infection, this problem may be worse due to direct damage to the gut lining and changes in nutrient flow in the colon. Such imbalance can give harmful microbes, or pathobionts, a chance to grow and further disturb gut ecology (18).

Rise of Campylobacteria and *Brachyspira*

During B. coli infection, increases in *Campylobacterota* and *Brachyspira* have been reported. Both groups contain species with pathogenic potential. Campylobacter spp. can trigger inflammatory responses, diarrhea, and in some cases, long-term gut disorders (18). Brachyspira spp. are linked to colonic inflammation in animals and humans. Their overgrowth suggests that B. coli infection shifts gut conditions—such as oxygen levels, nutrient gradients, and immune signals—toward those favoring pro-inflammatory bacteria (14,18).

The expansion of these taxa can worsen disease severity. They may synergize with B. coli to intensify epithelial damage and immune activation (12,14). Research on co-infections shows that combined microbial and parasitic insults can amplify pathology (12). In this way, dysbiosis is not only a result of infection but also a contributor to more severe outcomes. This aligns with broader evidence that microbial shifts during parasitic infection influence both local gut health and systemic immune status (11,13).

Reduced SCFA-Producing Bacteria

SCFA-producing bacteria, such as certain Clostridia and Lactobacillales, play an essential role in maintaining gut barrier integrity and modulating inflammation. Their reduction during B. coli infection

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can have direct health consequences (13). SCFAs like butyrate fuel colonocytes, reinforce tight junctions, and promote anti-inflammatory signaling (13,15). Loss of these producers is documented in multiple parasitic infections, including helminths, and is associated with poorer disease outcomes (15,16).

When SCFA levels fall, the gut barrier becomes more permeable, allowing translocation of pathogens and microbial products into the bloodstream (13,15). This can trigger systemic inflammation and complicate recovery. In the context of B. coli, reduced SCFA production may be driven by direct parasite-bacteria interactions and by immune-mediated environmental changes in the gut (12,14). Long-term depletion of these beneficial taxa may also hinder post-infection microbiota recovery, prolonging dysbiosis and its associated risks (16,18,19).

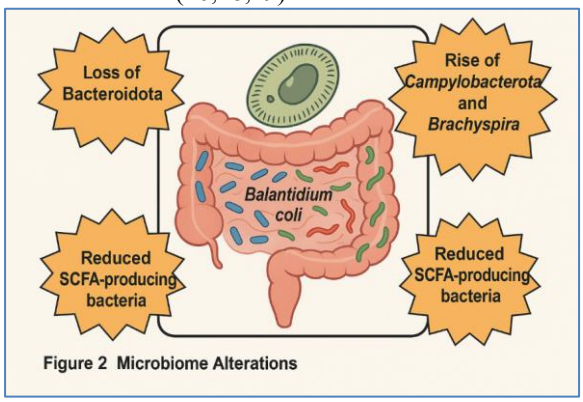

Figure 2: Microbiome alterations during *Balantidium coli* infection. Diagram showing the main microbial changes in the large intestine during B. coli infection. The parasite is present in the lumen and is associated with three key dysbiotic shifts: (1) loss of Bacteroidota phylum members, (2) rise of Campylobacterota and Brachyspira, and (3) reduction in short-chain fatty acid (SCFA)–producing bacteria. These changes can weaken the mucosal barrier, promote inflammation, and alter gut ecosystem stability.

Mechanisms of Dysbiosis

Direct parasite–microbe contact

Intestinal parasites such as Balantioides coli, Trichuris muris, and Entamoeba histolytica inhabit the gut alongside a diverse community of bacteria. In many cases, these parasites live in close proximity to the mucosal surface where commensal bacteria also thrive. Physical contact between parasites and bacteria can lead to changes in the microbial community structure, either by direct predation on bacteria, competition for nutrients, or by altering the availability of mucosal binding sites (20-24). Parasite-secreted molecules, including proteases and other effector proteins, may disrupt bacterial biofilms or change the growth conditions for certain bacterial groups, indirectly leading to the overgrowth of opportunistic or pathogenic species (25).

Experimental studies in helminth models have shown that worm colonization can selectively promote or suppress specific bacterial taxa. For example, T. muris infection in mice modifies the proportion of beneficial Lactobacillus and Bacteroides species, sometimes favoring the expansion of mucin-degrading bacteria that can undermine the mucus barrier (22,25). These shifts may weaken colonization resistance against other pathogens, providing an ecological niche for harmful bacteria. Direct interactions therefore act as a primary trigger for dysbiosis, setting the stage for subsequent immune and epithelial changes (26).

Immune response effects

Parasitic infections often trigger strong immune responses, which can influence gut microbiota composition. For example, immune cells responding to helminths or protozoa produce cytokines that can either suppress or promote the growth of certain bacterial species (21,24). Chronic infections may lead to prolonged immune activation, which is associated with the upregulation of inhibitory receptors such as P2X7 on immune cells (20). This can impair the clearance of both pathogens and dysbiotic bacteria, allowing harmful microbes to persist.

Parasites such as E. histolytica avoid immune attack by changing host cell signals, breaking down immune molecules, and controlling local inflammation (26-28). These actions can shift the immune response from protective Th1 activity to regulatory or Th2-dominant patterns (21,24). This reduces inflammation against the parasite but lets other microbes grow without control. Over time, the mix of immune suppression and selective bacterial growth leads to chronic dysbiosis. This imbalance can worsen disease and increase the risk of other infections or immune-related problems (28).

Changes in gut environment

Parasites also affect gut health by altering its physical and chemical conditions. They attach to the epithelium, invade the mucosa, and feed in ways that damage the intestinal lining (22). These actions can change mucus production and disrupt normal epithelial turnover. Infections like cryptosporidiosis break the tight junctions between epithelial cells (29). This raises gut permeability and allows bacteria to cross into the lamina propria. The leakage triggers both local and systemic inflammation, causing more harm to the mucosal barrier.

Additionally, parasitic infections can alter gut motility, pH, and oxygen gradients, creating favorable

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conditions for certain bacteria to thrive while suppressing others (25,30). Reduced SCFA production a result of diminished populations of fermentative commensals leads to less energy for colonocytes and weaker anti-inflammatory signaling (29). These environmental changes reinforce the altered microbial community, sustaining dysbiosis and making it harder for the microbiome to revert to a balanced state even after the parasite is cleared (30,31).

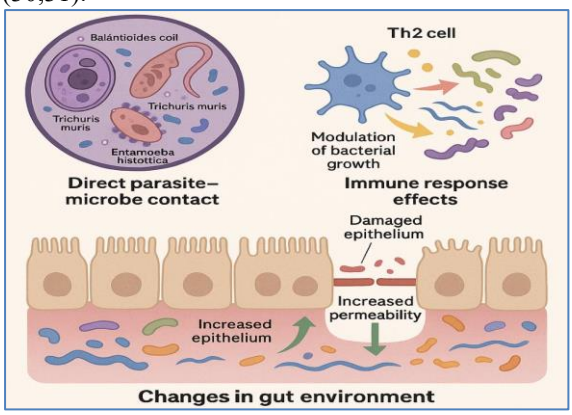

Figure 3: Mechanisms of Dysbiosis in Intestinal Parasitic Infections. This schematic shows three main ways parasites alter the gut microbiome: Direct parasite–microbe contact – Balantioides coli, Trichuris muris, and Entamoeba histolytica interact with bacteria on the mucosal surface. They compete for nutrients, disrupt biofilms, and release proteins that change bacterial growth. Immune response effects – Infection changes immune signaling. Cytokines alter bacterial composition. Parasites evade immunity and shift the balance from Th1 to Th2 responses, allowing dysbiotic bacteria to persist. Changes in gut environment – Parasites damage the epithelium and mucus barrier. They increase permeability, change pH and oxygen gradients, and reduce short-chain fatty acids. These changes favor harmful bacteria over beneficial ones.

Health Impact

Intestinal inflammation

Parasitic infections such as Balantioides coli, Entamoeba histolytica, and intestinal helminths can disrupt the normal gut microbiota, leading to immune dysregulation and inflammatory responses. Chronic colonization stimulates persistent activation of mucosal immune cells, which release pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6, promoting tissue injury and disease progression (32-35). This chronic inflammation can create a favorable environment for secondary bacterial infections and worsen pre-existing gastrointestinal disorders, including inflammatory bowel diseases (IBD).

In helminth-infected hosts, immune modulation may initially suppress excessive inflammation to benefit the parasite's survival, but the long-term presence of the parasite can trigger a low-grade inflammatory state (31,34). This ongoing immune activation not only damages the epithelial lining but also alters the gut ecosystem, reducing microbial diversity and fostering the growth of pro-inflammatory bacteria (34,35). These changes can perpetuate a cycle of inflammation and dysbiosis, increasing the risk of chronic gut pathology.

Increased permeability

Parasitic infections can impair the intestinal barrier by damaging epithelial cells, disrupting tight junction proteins, and thinning the mucus layer. These structural changes allow bacteria, bacterial products, and dietary antigens to cross the mucosal barrier into the underlying tissue, initiating immune responses and inflammation (36,37). For example, Schistosoma mansoni and other intestinal parasites can damage the gut barrier. This damage allows microbes to cross into the body and trigger systemic inflammation (36,37). The resulting "leaky gut" increases exposure to microbial antigens. This can drive stronger immune activation and more tissue injury (35). Over time, a cycle of barrier breakdown and immune response can develop. It may cause long-term diarrhea, abdominal pain, and other systemic problems. In some cases, parasite-induced immune suppression lets harmful bacteria survive in deeper tissues. This can slow recovery and raise the risk of other infections (31,38).

Nutrient malabsorption risk

Gut parasites and dysbiosis can lead to poor nutrient absorption. They damage intestinal villi, destroy epithelial cells, and change the microbiota (29,35). A loss of short-chain fatty acid–producing bacteria reduces the energy available to colonocytes. This can cause mucosal thinning and reduced absorptive function (29). As a result, the body may become deficient in vitamins, minerals, and essential amino acids. These deficiencies can lead to weight loss, stunted growth, and anemia.

In addition, parasitic infections may compete directly with the host for nutrients or alter gut transit time, reducing the efficiency of nutrient extraction from food (34,38). Chronic malabsorption has broader consequences, including weakened immune function, poor wound healing, and impaired vaccine responses, particularly in children from endemic regions (32). This nutritional deficit, combined with immune modulation by parasites, increases vulnerability to co-infections and long-term health problems,

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highlighting the importance of early diagnosis and integrated treatment approaches.

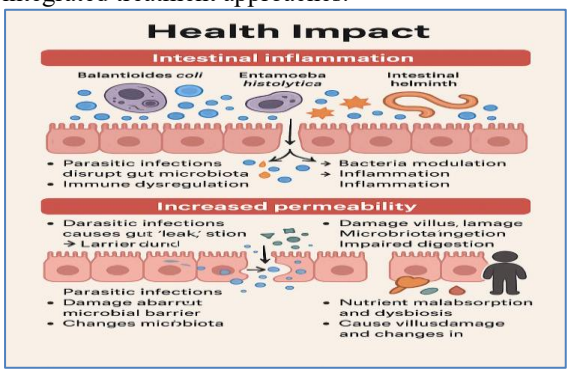

Figure 4: Health Impact of Balantioides coli and Related Parasitic Infections on the Host Gastrointestinal System. The figure illustrates three major consequences of parasitic infection–induced microbiome dysbiosis: (1) Intestinal inflammation, showing persistent activation of mucosal immune cells with the release of pro-inflammatory cytokines; (2) Increased permeability, depicting epithelial damage, disrupted tight junctions, and bacterial translocation; and (3) Nutrient malabsorption risk, highlighting villous atrophy, reduced SCFA production, and impaired nutrient uptake. Each panel integrates host–parasite–microbiota interactions and their pathological outcomes.

Effects on Distant Organs

Parasitic infections that alter the gut microbiota can also affect distant organs. Disruption of the intestinal microbial balance influences immune regulation throughout the body, which can worsen disease in other systems (39-45). For example, helminth and protozoan infections can modify cytokine profiles, leading to systemic inflammation or altered immune tolerance. This can influence the progression of conditions such as tuberculosis, where microbiota shifts may change the host's ability to control infection (40). Chronic immune activation also promotes fibrosis in organs like the liver by stimulating matrix-producing cells and inflammatory mediators (43).

Microbiota alterations caused by parasites can also affect metabolic and neurological functions. Dysbiosis can influence gut–brain signaling, contributing to disorders such as irritable bowel syndrome and possibly neuroinflammatory conditions (46-48). In experimental models, intestinal parasites reduced microbial diversity and altered metabolite production, which in turn impacted the function of distant tissues like the lungs and reproductive organs (42, 47). These findings show that parasite-induced microbiome changes are not limited to the gut but can contribute to multi-organ pathology.

Effects on Immune Regulation

The microbiome plays a central role in shaping immune responses, and parasitic infections can significantly alter this regulation. Helminths and protozoa may drive a shift from pro-inflammatory Th1 responses to regulatory or Th2-dominant patterns, which can help the parasite persist but also change the host's susceptibility to other diseases (41, 45, 49). These immune shifts can protect against certain inflammatory conditions but may impair the ability to control infections by bacteria and viruses (40). For instance, studies have shown that cohabitation of helminths with certain bacterial species can create a tolerogenic immune environment, reducing harmful inflammation but also limiting pathogen clearance (49).

Nutritional status interacts closely with immune regulation in parasitic infections. Inadequate diet can weaken the host's ability to mount effective immune responses and may worsen dysbiosis (41). This interaction is particularly important in pregnancy, where immune adaptations are necessary to support fetal development while maintaining defense against infection. Ultimately, parasite-driven microbiota changes can either amplify or dampen systemic immunity, with outcomes depending on the host's nutritional, microbial, and genetic context (50).

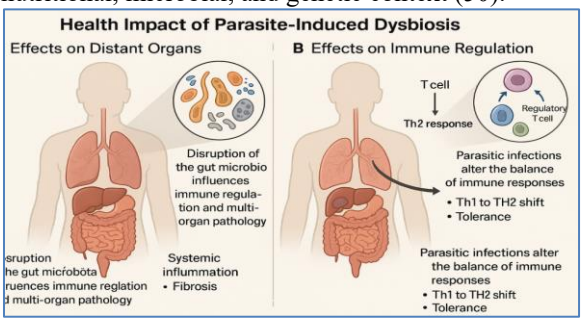

Figure 5: Health impact of parasite-induced gut microbiota alterations on distant organs and immune regulation. (A) Parasite-driven dysbiosis influences immune pathways, leading to systemic effects such as fibrosis, neuroinflammation, metabolic dysregulation, and altered cytokine profiles that can exacerbate diseases in distant organs (e.g., liver, lungs, reproductive system). (B) Parasitic infections shift immune regulation from pro-inflammatory to tolerogenic states, influenced by microbiota composition and nutritional status, with consequences for infection control, inflammatory disease risk, and maternal–fetal health.

Parasitic infections and their impact on the gut microbiome reveal a complex relationship between pathogens, host immunity, and systemic health. Evidence from recent studies shows that protozoa like

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Giardia lamblia not only disrupt intestinal function but also alter glucose and lipid metabolism through immune modulation (51). Blood microbiome shifts, observed in chronic and acute parasitic infections, highlight that microbial alterations are not confined to the gut (52). Helminths, including Schistosoma and soil-transmitted species, reshape both alpha and beta diversity of the gut microbiota, often collapsing microbial gradients essential for host health (53-55). These changes contribute to inflammation, immune suppression, and broader physiological effects that extend beyond the intestine. Dietary factors, such as amino acid availability, can further influence host immunity and microbial ecology, emphasizing nutrition as a cofactor in parasite–microbiome interactions (54).

Emerging treatments are shifting toward combined strategies. Probiotics are being studied to restore healthy bacteria and reduce helminth-related dysbiosis in low-resource areas (56). In schistosomiasis, microbiome-based biomarkers may help with diagnosis, prognosis, and treatment monitoring (57). Natural products, such as the polyherbal mix triphala, have antioxidant, immune-supporting, and antimicrobial effects. These could be useful against parasitic infections (58). Neglected ectoparasites, like Demodex mites, remind us that public health work must address all parasitic threats. This includes both intestinal protozoa and skin-dwelling arthropods (59). These findings highlight the need for joint work across disciplines to turn microbiome research into prevention and treatment tools.

Conclusion

Parasitic infections interact closely with the gut microbiome. Direct contact with gut bacteria can upset the normal microbial balance. This may weaken the gut lining and allow harmful species to grow. The result is often less microbial diversity, loss of beneficial bacteria, and more opportunistic organisms. Such changes can harm nutrient use and alter key microbial products that protect the gut and support immunity. This imbalance can worsen symptoms, promote inflammation, and raise the risk of other infections.

The effects are not limited to the gut. Parasites can change immunity and metabolism throughout the body. They may suppress or overstimulate immune activity, which can affect responses to other diseases. Changes in glucose and lipid metabolism, linked to altered microbiota, can add to metabolic problems and inflammation. Shifts in gut pH, oxygen levels, and nutrients can make dysbiosis harder to reverse. Understanding these processes is vital. Restoring microbial balance could give new ways to treat parasitic diseases and related health problems.

Conflict of interest

There is no conflict of interest in this study as stated by the authors.

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