https://doi.org/10.70882/n764ex89

SCIENCE FORUM (JOURNAL OF PURE AND APPLIED SCIENCES) 26, (5 Special) (2026), 890 - 901

Science Forum (Journal of Pure and Applied Sciences)

Journal Homepage: https://atbuscienceforum.com.ng/index.php/jpas

Intestinal Parasitic Infections and Gut Microbiome Dysbiosis: Implications for Epithelial Integrity, Mucosal Immunity, and Host Metabolic Regulation – A Comprehensive Review

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Abstract

Intestinal parasitic infections are part of a complex ecosystem of interactions between intestinal helminths and protozoa with intestinal epithelium, mucosal immune system, and gut microbiota. Both interactions are two ways, in that parasites can alter the microbial community and epithelial physiology, and commensal microorganisms and metabolites can affect the establishment, persistence and clearance of parasites. This review discusses the host-parasite-microbiota axis focusing on three interrelated physiological pathways: intestinal barrier function, immune regulation, and metabolic homeostasis. The evidence is dealt with in relation to general principles of microbial ecology, and is presented for Giardia duodenalis, Entamoeba histolytica, Cryptosporidium spp. and intestinal helminths. Mechanisms include, but are not limited to, remodeling of the mucus, remodeling of the glycocalyx, changes in epithelial attachment/invasion, disruption of tight junctions, changes in epithelial turnover, innate immune sensing, type 2 and regulatory immune responses, and microbial metabolite signaling. Microbial ecology and host physiology are thought to be connected by short-chain fatty acids, metabolites of the amino acid tryptophan, and transformation of bile acids. Human studies show that parasite-associated microbiome patterns are heterogenous, depending on the species, geographic location, diet, age, may be impacted by co-infections and may be influenced by treatment. Experimental models give us mechanism but that may not necessarily be applicable to disease in humans. There is current evidence of a biologically significant tripartite relationship; however, there are gaps in the understanding of the causal pathways between specific changes in microbes and clinical metabolic outcomes.

Keywords

Human Intestinal Parasites;
Gut Microbiota;
Host-Parasite-Microbiota Axis;
Intestinal Barrier;
Mucosal Immunity;
Short-Chain Fatty Acids;
Metabolic Homeostasis.

EISSN: 3156-5190 (Online)
ISSN: 1119-4618 (Print)
JPAS
Faculty of Science, ATBU Bauchi

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1. Introduction

Gastrointestinal tract is one of the major sites of parasitic infections and a rich microbial community. Its physiological function requires a coordinated activity among the cells, mucus, antimicrobial molecules, immune cells, enteric nerves, dietary substrates, and the indigenous microorganisms. The microbiota has a role in nutrient processing, in providing colonization resistance, in maintaining the integrity of the epithelial surfaces and in immune education. Intestinal parasites are not just pathogens but also organisms that interact with the existing gut microbes, compete for nutrients and adapt the environment of the host (Grondin et al., 2024; Al-Rashidi & El-Wakil, 2024).

The relationship between host and parasite is mutualistic and there is an interaction between host and their microbiota as well. The impact of the parasite on the microbial community can be achieved through tissue damage, modification of mucus composition, competition for nutrients, immune modulation and through production of parasite-derived molecules. On the other hand, bacteria and their metabolites can have an impact on the survival, attachment, colonization and resistance of parasites. This phenotype is not homogeneous: one host can be a carrier with no clinical consequences, whereas the other will develop clinical disease based on the host immune response, microbial balance, nutrition, genotype of the parasite and environmental factors (Grondin et al. 2024; Loke et al. 2022).

The interactions between parasites and microorganisms and helminth associated immune regulation have been discussed in previous reviews. This review combines these themes with the physiology of the epithelial barrier and metabolic signalling. The focus of its central perspective is the interdependence of the integrity of barriers, mucosal immunity and microbial metabolism, as one physiological axis.

2. Review scope and literature approach

This article is a comprehensive narrative review, and not a registered systematic review or a meta-analysis. Literature was searched using specific search terms in PubMed/MEDLINE, PubMed Central, publishers' platforms and multidisciplinary scholarly search engines. Intestinal parasites, protozoa, helminths, gut microbiota, microbiome, intestinal epithelial barrier, mucus, tight junctions, mucosal immunity, short-chain fatty acids, bile acids, tryptophan metabolites and metabolic homeostasis were included in the search concepts. Peer-reviewed human studies, systematic reviews, mechanistic studies, and authoritative reviews from the past several years (2017-2026) were prioritized, but some of the earlier foundational studies were also included.

The results of the studies were interpreted in the light of the study design. Human observational and intervention studies were considered as direct clinical evidence, animal experiments, organoid systems and in-vitro studies were considered as plausible mechanisms and are identified as preclinical evidence. The review does not assign a specific taxonomic signature to a universal or causal effect as microbiome studies can be heterogeneous when examining the relationship between parasites and the microbiome.

3. The intestinal ecosystem and physiological homeostasis

3.1. The gut microbiota as a functional community

The gut microbiota consists of bacteria, archaea, fungi and viruses, with the best characterized being the bacteria. It functions in a variety of ways: fermentation of indigestible carbohydrates, biosynthesis and modification of metabolites, modification of bile acids, competing with pathogens, and interactions with mucosal immune cells. Many factors such as diet, age, geography, medication, sanitation, host genetics and infection history affect microbial community structure. Thus, some of these factors need to be controlled to make comparisons between different microbiomes.

3.2. The intestinal barrier

The intestinal barrier is a multi-layered and dynamic system. The outermost layer of mucus prevents direct contact between the luminal organisms and the epithelial cells, mucins in the colon, especially MUC2, are the scaffold in this separation. The antimicrobial peptides and immunoglobulin A play a role in microbial containment. Under these layers, the epithelial cells are joined by tight, adherens and desmosomes. The tight junction proteins (occludin, claudins, zonula occludens proteins) control the paracellular permeability. There are epithelial stem cells, which renew the lining; goblet cells, Paneth cells, enteroendocrine cells and absorptive enterocytes that have specialized functions. The maintenance of barrier homeostasis requires communication between the epithelial cells, immune cells and microbiota (Yao et al., 2024).

3.3. Microbial metabolites and host physiology

Microbial fermentation of food substances, especially acetate, propionate and butyrate, are known as short-chain fatty acids or SCFAs. Butyrate is a key source of energy for colonocytes, and may modulate epithelial junctions, mucus production, inflammatory signaling, and regulation T cell function. SCFAs communicate via G-protein coupled receptors and these molecules have the ability to alter gene expression via histone deacetylase inhibition. The biological consequences of their exposure are concentration, site of exposure, nutrient intake and the microbiota associated with their production (Mann et al., 2024; Liu & Lu, 2024).

Other metabolites that are relevant are indoles derived from tryptophan that can activate the aryl hydrocarbon receptor (AhR) and the microbial transformations of bile acids that can activate host receptors like FXR, TGR5. These routes link together the interaction of microbial ecology with the regulation of the immune system in addition to repair of epithelial surfaces and energy metabolism. The specific contribution in parasitic diseases of humans is an area of ongoing research and not a "formulated clinical mechanism".

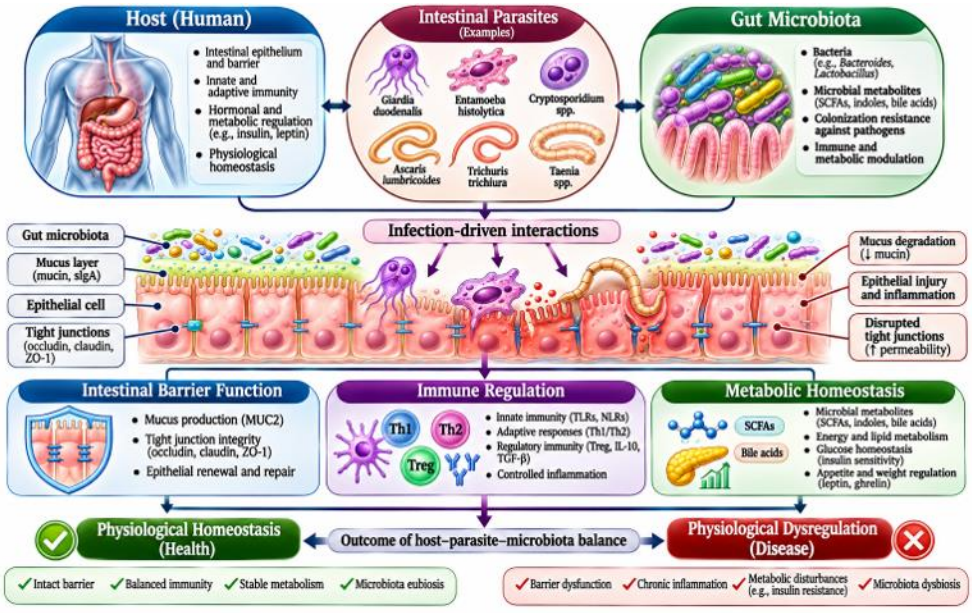

Figure 1. Conceptual framework of the host–parasite–microbiota interaction network.

4. Major intestinal parasites and their interactions with the microbiota

4.1. Giardia duodenalis

Giardia duodenalis is found in the upper small intestine and attaches to the epithelial surface. The clinical spectrum includes asymptomatic carriage, acute or chronic diarrhea, abdominal discomfort and malabsorption. Giardia may cause damage to the brush borders, microvilli, epithelial junctional complexes, mucus and local immune signaling. Experimental and translational studies suggest that secreted factors by parasites such as proteases can alter the integrity of epithelia and the structure of commensal biofilms (Allain et al., 2017; Buret, 2020).

The results obtained from the studies of human microbiome in giardiasis are not consistent. Comparisons are made difficult by differences in cohort characteristics, dietary habits, geographic and environmental factors, co-infections, diagnostic techniques, and by studying either symptomatic or asymptomatic carriers. A 2024 study found that Giardia-associated microbial dysbiosis correlated with changes in intestinal mucin glycosylation in experimental models, involving goblet cell and mucin changes and SCFA levels. These results provide a plausible microbiota–mucus mechanism, but animal model results cannot be translated to humans (Fekete et al., 2024).

4.2. Entamoeba histolytica

Entamoeba histolytica forms a relationship with the colonic mucus and epithelial cells and can lead to an invasive amoebic colitis. Pathogenesis includes penetration of the mucus barrier, binding to the host cell, lytic activity, proteinase activity and inflammatory response. The resident bacteria can affect the virulence of the parasite or the mucosal environment, and the parasite can affect the interactions between the epithelium and the resident bacteria. Not all infections lead to clinical disease; there is variability in clinical expression due to host and microbial context (Chadee et al., 2017).

4.3. Cryptosporidium species

Cryptosporidium species are found intracellular but extracytoplasmic on the apical surface of intestinal epithelial cells. Infection may disrupt the brush border, cause changes in the rate of epithelial turnover and cause villus blunting and diarrhea. Epithelial innate responses and T-cell mediated immunity are necessary for effective control, and there is a higher risk of severe disease in young children and those with abnormal cellular immunity. Recent reviews address the interactions between mucus and epithelial integrity, mucosal immunity and microbial community, though some mechanistic detail comes from animals and cell model (Pardy et al., 2024).

4.4. Intestinal helminths

Intestinal helminths comprise nematodes (Ascaris lumbricoides, Trichuris trichiura, hookworms), cestodes and selected trematodes. They are dependent on anatomical site, feeding habits, tissue migration, worm burden and duration of infection. Helminths can induce mechanical damage, inflammation of the mucosa, blood loss, competing for nutrients and stimulation of type 2 immune responses. Studies of human microbiome have reported on the variations in microbial diversity and composition. A systematic review and meta-analysis of human studies revealed a general trend towards higher bacterial diversity and richness in helminth-positive samples, but the variation in these studies did not allow for the identification of a microbial signature (Walusimbi et al., 2023).

The epithelial alarmins, innate lymphoid cells, Th2 cells, eosinophils, mast cells, goblet-cell responses and regulatory pathways are all components of helminth-associated type 2 responses. IL-4, IL-5, and IL-13 play roles in anti-helminth defense and tissue responses, whereas IL-10 and regulatory T cells could be involved in preventing hyperinflammation. The microbiota can be involved via microbial metabolites, but experimental models provide more solid evidence than humans (Loke et al., 2022).

Table 1. Representative intestinal parasites and principal host–microbiota interfaces

Parasite	Main niche	Potential host interface	Evidence
Giardia duodenalis	Upper small intestine; epithelial surface	Brush-border injury, mucus and junctional changes, biofilm interactions	Clinical and mechanistic evidence
Entamoeba histolytica	Colon; mucus and epithelium	Mucus penetration, cytotoxicity, bacterial competition, inflammation	Experimental and clinical evidence
Cryptosporidium spp.	Apical intestinal epithelial niche	Epithelial turnover, villus injury, innate and T-cell responses	Mechanistic evidence; many models
Intestinal helminths	Small intestine or colon	Type 2 immunity, mucus responses, blood loss or nutrient effects	Human microbiome evidence heterogeneous
Taenia spp.	Small-intestinal lumen	Ecological competition and microbial shifts	Human cohort evidence emerging

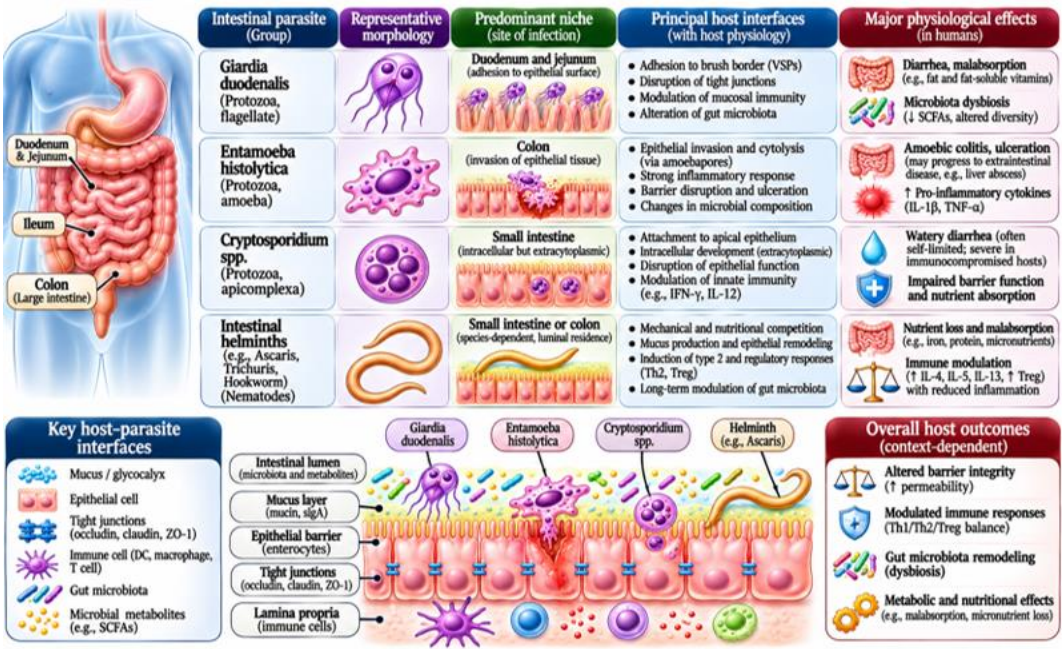

Figure 2. Major intestinal parasite niches and principal host interfaces.

5. Parasite-associated changes in microbial ecology

Parasite-associated dysbiosis shouldn't be equated with just a loss of diversity or whole population increase of pathogenic bacteria. The various parasites have different niches in the intestine and their ecological pressures vary. In some helminth infections, richness was found to be increased while in protozoan infection, there were various patterns depending on various organisms and clinical conditions. In the context of human taeniasis, the research has shown that there are significant changes in the composition of microbiome before and after treatment, and that the change might be different among different individuals (Taenia asiatica cohort, 2024).

Possible factors include an actual competition for nutrients, changes in oxygen tension, changes in luminal pH, altered mucus glycosylation, damage to the epithelium, inflammatory mediators, altered transit time, diarrhoea and effects related to therapy. Molecules derived from parasites can also have an effect on bacterial adhesion, structure of biofilm and virulence. Microbiological profiles can be modified independently by the antibiotics, antiparasitic drugs, diet, and other enteric pathogens found in co-infections and these must be taken into account. Any cross-sectional associations between the two should not be considered as directly involving the parasite unless significant confounders are considered.

6. Intestinal barrier dysfunction in parasitic infection

6.1. Mucus and glycocalyx alterations

Mucus creates a spatial barrier between microorganisms and epithelial cells, and can facilitate the colonization of microorganisms. Parasites can cause changes in the thickness of mucus, mucin expression, glycosylation and/or degradation. In experimental systems, parasite-induced changes in mucin glycosylation have been associated with changes in microbiota, indicating a potential for microbiota changes to be more widespread than to just the site of parasite colonization (Fekete et al., 2024).

6.2. Tight junctions and epithelial permeability

Tight junctions control the passage of water, ions and macromolecules between the cells of an epithelium. Attachment and secretion of parasites, activation of proteases and inflammatory cytokines and oxidative stress can lead to modification of proteins in the junctions or to the modulation of their interaction with the cytoskeleton. Experimental studies have reported that the organization of ZO proteins, occludin, claudins and actin changes in giardiasis. Disruptions of junctional organization and increased permeability have also been observed in experimental models and in epithelial models in the context of Cryptosporidium infection (Allain et al., 2017; Pardy et al., 2024).

Local immune activation may be amplified by increased permeability allowing more microbial products and/or antigens to pass through into the lamina propria. Care needs to be taken with the term ‘leaky gut’ as intestinal permeability can be scientifically measured using validated techniques and that only general symptoms are not enough to confirm barrier dysfunction.

Table 2. Key physiological components of the intestinal barrier

Component	Principal function	Potential parasite-related alteration
Mucus and mucins	Spatial separation; microbial habitat	Altered thickness, glycosylation, degradation, secretion
Epithelial cells	Selective absorption and physical barrier	Cell stress, apoptosis, altered turnover, microvillus injury
Tight junctions	Regulation of paracellular permeability	Altered occludin, claudins, ZO proteins or distribution
Antimicrobial peptides	Control of microbial proximity and growth	Altered epithelial production and inflammatory regulation
Mucosal immune cells	Pathogen control, tolerance, repair	Changes in innate sensing, Th1/Th2 balance, Treg activity

6.3. Epithelial turnover, apoptosis, and repair

Crypt stem cells constantly replace the intestinal epithelium. Infections can exacerbate the loss of epithelial cells and compensatory proliferation, but chronic inflammation or direct injury from parasites can disrupt the injury/repair equilibrium. Cryptosporidium models show crypt remodeling, blunting of villi and changes in epithelial turnover. These may result in structural changes in the intestines that reduce the surface area available for absorption and cause diarrhea and loss of nutrients. The observations are informative and are of mechanistic significance, but are not direct histological observations in the human setting.

6.4. Functional consequences

Barrier injury may play a role in diarrhoea, electrolyte transport disruption, malabsorption, gastro-intestinal symptoms and post-infectious gastro-intestinal symptoms. The magnitude will depend on the species of parasite, intensity of infection, nutrition, age, immune status and duration of infection. Enduring symptoms following elimination of parasites could be a result of residual epithelial changes, changes in the microbial ecology, an immunological response, or other post-infectious mechanisms, and a causal link must be determined by clinical evaluation.

7. Immune regulation at the host–parasite–microbiota interface

7.1. Innate immune recognition

Intestinal epithelial cells and resident immune cells detect molecules of microbes and parasites with pattern recognition receptors. Early defense cells include epithelial cells, macrophages, dendritic cells, neutrophils and innate lymphoid cells. Recruitment and activation of immune cells are mediated by cytokines and chemokines, and antimicrobial peptides and secretory IgA help to limit organisms to the lumen. These responses are modulated in terms of magnitude and nature by the microbiota, which can promote immune maturation and ensure tolerance to commensals.

7.2. Adaptive Immunity & Type 2 Immunity

The adaptive responses to protozoan and helminth infections are different but similar. In intracellular protozoa, effective cellular immunity, such as interferon-γ pathways and T-cell responses are

usually necessary. Type 2 responses with IL-4, IL-5, IL-13, eosinophils, mast cells and activity of goblet cells are common responses elicited by helminths. These are simplified models; real-life interactions between parasites and host tissues are subject to variation depending on the infection stage, parasite and host factors.

7.3 . Regulatory immunity and tolerance

Tissue-damaging inflammation can be inhibited by regulatory T cells, IL-10, TGF-β and regulatory macrophage programs. Regulation can help keep the integrity of the epithelium intact, and aid repair of tissue, but may also help the persistence of parasites if it is excessive or maintained over time. Biological outcome is the result of a balance between elimination of parasites, tissue protection and immunopathology. In humans, helminth studies have suggested a connection between the composition of the microbiota and immune regulation, but cause and effect remain poorly understood, and the role of immune modulating metabolites is still not completely clear (Loke et al., 2022).

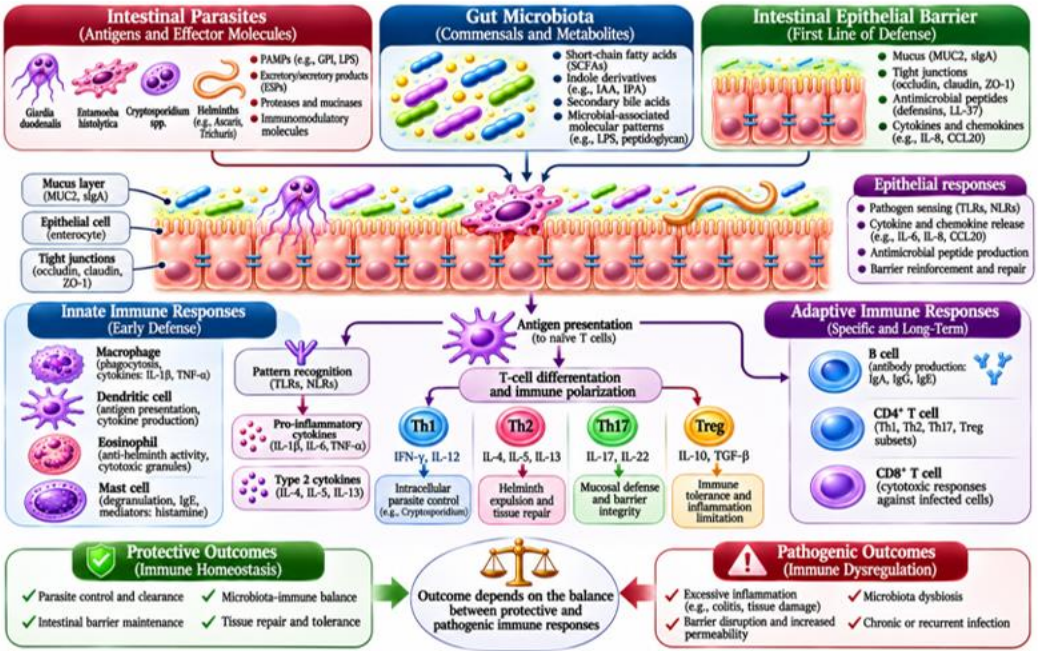

Figure 3. Immune pathways at the host–parasite–microbiota interface.

7.4. Microbial metabolites as immune mediators

SCFA act on the epithelial cells, macrophages, dendritic cells and T-cell differentiation via receptor-dependent and epigenetic pathways. Butyrate seems to be linked to the energy supply of the epithelium and to regulatory immune activity, propionate and acetate have a more generalized signaling function. Microbial tryptophan metabolites could activate AhR pathways, as could bile-acid derivatives, which could modulate immune and epithelial signaling. They are known to play important roles in gut physiology but their specific functions in people with parasites is a knowledge gap (Mann et al., 2024; Liu & Lu, 2024).

8. Metabolic homeostasis and nutritional physiology

8.1. Nutrient absorption and energy balance

Intestinal parasitic disease can impact the nutritional physiology by reducing intake, causing diarrhea, injuring the gut's mucosa, changing the activity of

the digestive enzymes, competing with other nutrients and causing blood loss. Damage to the brush-border by Giardia may affect the absorptive capacity and hookworm infection may add to chronic intestinal bleeding and iron deficiency. Significant infection by the Trichuris can result in colitis and malnutrition. The severity of the infection is related to the amount of infection present and nutritional health of the host.

8.2. The effect of the SCFAs on energy metabolism

The microbial fermentation and host energy metabolism are linked by the SCFAs. Butyrate is used for metabolism by the colonocytes, whereas acetate and propionate are metabolized in the rest of the body and are involved in signaling of appetite, glucose handling and lipid metabolism. Changes to the microbial community in the presence of the parasite may impact SCFA production, but this is not necessarily due to the presence of the parasite. These are all important factors, such as diet, fibre intake, medication and transit time.

8.3. Bile-acid and tryptophan pathways

The microbial bile acid transformations produce secondary bile acids which have the potential to signal via FXR and TGR5 and affect lipid, glucose and inflammatory physiology. Microbial tryptophan metabolites have the ability to modulate epithelial barrier and immune signaling pathways via AhR. These are potentially good candidates for systemically mediated effects of intestinal dysbiosis; however, there is limited metabolomics evidence from humans for the effects of each specific parasite.

Table 3. Microbial metabolites and potential host physiological functions

Metabolite	Origin / pathway	Potential host function	Parasite-specific evidence
SCFAs	Fermentation of dietary carbohydrates	Colonocyte energy, barrier support, immune modulation	Emerging; causal human evidence limited
Indole derivatives	Microbial tryptophan metabolism	AhR signaling, epithelial and immune regulation	Needs parasite-focused human metabolomics
Secondary bile acids	Microbial modification of primary bile acids	FXR/TGR5 signaling; metabolic and immune effects	Insufficiently characterized
Other microbial products	Amino-acid and carbohydrate metabolism	Host signaling and ecological competition	Context-dependent

8.4. Micronutrients and anemia

Blood loss due to parasitic infection, iron restriction due to inflammation, decreased iron absorption and malabsorption can all be causes of iron deficiency. Other nutritional implications can be a lack of folate, vitamin B12 and fat-soluble vitamins in specific clinical situations. Not all intestinal parasites cause abnormalities. Laboratory assessment should follow a

clinical approach and entail a proper hematological and nutritional evaluation.

9. Human evidence and translational interpretation

There is growing but inconsistent human proof with respect to different species of parasites. From a much wider literature search, 19 studies were chosen for inclusion in a systematic review and meta-analysis, which revealed an overall rise in gut bacterial diversity and richness, with a strong highlight of the need to take into account the heterogeneity of the data. This is an example of the value and current limitations of the evidence; microbial changes are detectable but the value of these changes and their association with clinical outcomes needs to be further explored (Walusimbi et al., 2023).

A recent 2024 review of the intestinal parasite community and the gut microbiota highlighted evidence of the relationship between parasites and microbes in the gut, and how the microbes affect the hosts' ability to defend against parasites. In another review from 2024, these interactions were reviewed with a focus on their therapeutic implications and dual nature. Evidence of human work showed the presence of taeniasis was associated with changes in the microbial composition and a cohort of protozoan-positives in 2024 revealed different microbial profiles when looking at intestinal protozoa. The results of these studies further underscore the importance of not assuming a "one size fits all" microbiome signature for all parasites (Grondin et al., 2024; Al-Rashidi & El-Wakil, 2024; Marangi et al., 2024).

Currently, microbiomes cannot be used as a stand-alone diagnostic tool to diagnose intestinal parasite infection and should not be used to replace stool microscopy, antigen assays, molecular analysis of parasites, or clinical evaluation. Microbiota directed interventions should not be a substitute for effective antiparasitic intervention.

10. Diagnostic and research biomarkers

A comprehensive human study should include detection of parasite, microbiome, barrier testing, immune markers and metabolic testing. Stool parasite testing can be done using the methods of microscopy, antigen detection or nucleic-acid amplification, depending on the organism. The approach to microbiome analysis can be either bacterial community profiling by 16S rRNA gene sequencing or species-level and/or functional characterization by shotgun metagenomics. SCFAs, bile acids and metabolites of tryptophan can be quantified by metabolomics.

A range of potential barrier markers (intestinal fatty acid-binding protein (I-FABP), functional permeability tests, and selected junctional or epithelial markers) are available, but none of these markers is specific enough to be used singly. Assessment of the immune system can consist of measurement of faecal calprotectin, cytokine panels, secretory IgA and other selected cellular phenotypes. Complete blood count, ferritin, transferrin saturation, albumin, glucose, lipid profile and anthropometric indices may be included in a metabolic and nutritional evaluation. Selection of biomarkers should be guided by a hypothesis, and validated laboratory procedures.

11. Therapeutic and preventive implications

The most important intervention to treat confirmed clinically important parasitic infection is antiparasitic treatment. The choice of treatment will depend on the species of parasite, severity of the disease, host factors and guidelines in the area. Treatment is supportive management which may involve rehydration, correction of electrolyte imbalances, nutritional rehabilitation and treatment for documented deficiencies. Safe water, sanitation, food hygiene, hand hygiene, health education and context appropriate public-health control measures are required for prevention.

Probiotics, prebiotics, dietary fibre and microbiota restoration are research areas that are ongoing. Barrier function, production of SCFA and immune

regulation are all possible benefits. Clinical evidence is dependent on the species and type of the parasite and products and interventions that might be beneficial in animal models cannot be assumed to be effective in human subjects. FMT should not be used for the treatment of parasitic infections as it is not a routine procedure and should not be used unless in research or accepted indications.

12. Knowledge gaps and future directions

- Longitudinal human cohorts of people are required to determine if there are changes in the microbiome before, during, and after infection, or if changes are present only after the parasite has been cleared.
- Studies should differentiate between symptomatic, asymptomatic, reinfection, mixed (dual) infection and post-treatment states.
- Integrated metagenomic and metabolomic analyses are required to establish the relationship between microbial taxa and functional outputs, such as metabolites related to SCFAs, bile-acid derivatives and tryptophan metabolites.
- The use of human intestinal organids, epithelial–immune co-cultures, and more sophisticated ex-vivo models can help elucidate mechanisms and mitigate the reliance on animal models.
- The following factors should be taken into account for the research: diet, age, geographical location, sanitary conditions, antibiotic use, antiparasitic treatment, nutritional status, and co-infections.
- The clinical studies are needed to identify microbiome or metabolite markers to define disease severity, treatment response, recurrence and/or post-infection symptoms.
- Before using microbiota-directed therapies as a standard method of parasite control, interventional trials are needed.

13. Conclusion

Parasites, their associated microbiota, the epithelial barriers and mucosal immunity play complex roles in the context of human intestinal parasitic infections. Altered mucus structure, epithelial adhesion, absorption of nutrients, immune response and production of microbial metabolites are possible changes associated with parasites. Bioactive substances produced by the microbiota in turn can affect the establishment of parasites and host defense. Evidence for the biological plausibility of this tripartite interaction and some specific mechanisms has been compelling, whereas causal relationships between specific changes in microbial communities and systemic metabolic outcomes in humans are not yet sufficiently clear. Longitudinal studies of humans on a species-by-species basis involving a combination of parasitological, microbiomical sequencing, metabolomics, barrier biomarkers and clinical physiological parameters are needed to make further progress.

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