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Review Article

Parasites and Bacterial Interaction in the Digestive Tract

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Abstract: In our intestine, there is an interface between immunity and intestinal pathogens, which is important for maintaining homeostasis and influencing the other. Maintaining health seems to depend on the human body's gut microbiota being balanced. Protozoans and helminths are two types of intestinal parasites that interfere with the microbial atmosphere altering the host stability. However, the microbiota of the gut is a special constituent that could seriously impede the infection pathophysiology. Probiotics can be supportive in lowering the many parasite pathogenicity, in addition commensal microbiota of the gut play a significant role in helping many parasite occurance, such as the synthesis of nutritious particles. For these reasons, there is a rising interest in elucidating the logic behind potential relationships between: intestinal parasites, inflammation immune response, and microbiota.

Keywords: Parasites, bacteria, gastric infection

INTRODUCTION

Enormous microbial population have been connected to the human body making up the complex ecosystem which is gut of the human. Each person has a distinct species of bacteria that may make a variations during time, and the species composition differs widely between individuals (Bäckhed *et al.*, 2005; Eckburg *et al.*, 2005; Qin *et al.*, 2010). While the environment significantly influences the acquisition of species, genetic factors are significant in the formation of microbiota of intestine (Zoetendal *et al.*, 2001) human body and intestinal microbiota within it have recently been called as "superorganisms," where a vast coordination of metabolic processes and bodily functions take place (Nicholson *et al.*, 2004). The human returns greatly from the occurrence of the gut microbiota, which plays a key role in controlling lipid storage of the host (Bäckhed *et al.*, 2004), inducing the regeneration of epithelium layer of the intestine (Rakoff-Nahoum *et al.*, 2004), and affecting the host immune system development (Mazmanian *et al.*, 2005). According to a recent review (Sekirov *et al.*, 2010; Clemente *et al.*, 2012), disruption of the microbial arrangement has been linked to a number of illnesses (Bäckhed *et al.*, 2005; Palming *et al.*, 2006). Maintaining proper balance between the human body and the gut microbiota is essential for maintaining health. Additionally, the intestinal epithelium's "barrier effect," which serves as the host's main line of defense and a physical barrier against pathogen invasion, is facilitated by the commensal microbiota (Bancroft *et al.*, 2012). In this intricate situation, the equilibrium between the host and gut microbiota is altered by intestinal parasite interactions with the microbial population. These organisms all interact with substrates to metabolize and change them. The physiology and survival of many parasites, as well as the course of many parasitic diseases, may be significantly impacted by resident microbiota products. However, intestinal protozoans and helminths always release chemicals disturbing the atmosphere and cause changes in the composition of the gut microbiota. Additionally, some of the energy that resident bacteria derive from the metabolism of nutrients may benefit eventual parasitic species as well as the host (Sekirov *et al.*, 2010). Therefore, it is important to think of the intestinal setting as an environment where host, parasite, and microbiological populations interact biologically and chemically at different organizational levels (Nicholson *et al.*, 2004; Bancroft *et al.*, 2012).

Infection with protozoa and gut microbiota

The human gastro-intestinal tract is commonly infected with a broad variety of protozoans. Biochemistry and also their physiology are mostly adapted to the parasitic convention, are not found in a homogeneous group. A variety of host invasion strategies are demonstrated by them; some are intracellular (like Cryptosporidium spp.) and others are

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host-specific (like Entamoeba histolytica), and many are multi-host adaptable (like Giardia duodenalis). While few species actually cause harm, a few do occasionally cause symptoms, most commonly diarrhea caused by damage to the intestinal wall. One protozoan species that could serve as a useful model to illustrate certain mechanisms pertaining to the current communications with the microbiota of the gut is G. duodenalis. It is acknowledged that this flagellate is among the most prevalent harmful intestinal parasites in human also for other species (Thompson,2000). clinical symptoms includes chronic or acute diarrhea, malabsorption and losing weight and that lasts for several months, in addition to a moderate self-limiting disease (Farthing, 1996).Several investigations evaluated the idea that pathogenesis arises from the combination of parasite products, like epithelial barrier-breaking proteinases, and host immunological and inflammatory responses, could be seen in the cases of Giardia (Scott *et al.*, 2004; Ankarklev *et al.*, 2010) and Cryptosporidium (Chai *et al.*, 1999; Guk *et al.*, 2003). in vitro studies have been shown that monocytes of human during infection with E. histolytica (Maldonado *et al.*, 2000) and also during Trichomonas vaginalis infections in vivo (Zariffard *et al.*, 2004), also of protozoans parasitizing surface mucosa involving the innate immune system, such as toll-like receptors (TLRs). Furthermore, macrophages, neutrophils, T cells (especially those that involve CD8+ cells), and antibodies (IgA, IgM, and IgG) are the significant parts of the "acquired immune response "that is essential to eradicate giardiasis.

Another element that could have a significant impact on the pathophysiology of parasite infections is gut microbiota. Nonetheless, little is known about the current interactions between protozoan parasites and the intestinal flora. Normal intestinal flora has been demonstrated to reduce susceptibility to Cryptosporidium parvum infection in animal models (Harp *et al.*, 1992). On the other hand, according to some research, the expression of pathogenic and enteric protozoans, including Blastocystis hominis (Phillips and Zierdt, 1976), various Eimeria species (Visco and Burns, 1972; Owen, 1975; Gouet *et al.*, 1984) E. histolytica (Phillips *et al.*, 1955), appears to require the presence of gut microbiota. Various theories have been put up to explain the mechanisms underlying this bacterially-induced pathogenic activation. Axenization of the protozoa is responsible for some of these alterations. In this instance, the occurrence of intracellular symbionts type of bacterial modifies the surface ligands of saccharide of the plasma membrane. As a result, axenic protozoa that have been became free of their endosymbionts may exhibit a probable diminution in adhesion or inva-sive capabilities (Phillips, 1973; Dwyer and Chang, 1976). In the last ten years, endosymbiotic microbes have also been discovered in the ultrastructure of Giardia muris in a murine model. These observations, according to the authors, may be connected to differences in the metabolism, pathogenicity of the trophozoite, features of the antigen surface, infectivity range, specificity of the host (Nemanic *et al.*, 1979). El-Shewy and Eid also showed that Giardia trophozoites harbored peripheral bacterial endosymbionts (2005). The authors' findings, based on TEM analysis, confirmed the protective function of the host, bacterial endosymbionts within Giardia trophozoites and provided additional evidence for the theory that intestine microbiota may influence either directly or indirectly giardiasis pathogenesis. trophozoites with endosymbionts were lysed when in close proximity to the stimulated Paneth cells. Parasite virulence expression may be influenced by intestinal axenization of the host is equally fascinating. Research of Escherichia histolytica, Mirelman and associates (1982, 1983) demonstrated that contacts between low pathogenicity amoebae and a range of Gram-negative bacteria, primarily strains of Escherichia coli, is the cause of the rise in amoebic virulency.

e. Galván-Moroyoqui *et al.*, (2008) have shown in a more recent study that enteropathogenic bacteria strains phagocytosis, such as Shigella dysenteriae and E. coli, that have been co-cultured in vitro with Entamoeba dispar and E. histolytica, increased the expression of the amoebic surface Gal/GalNAc lectin and the activity of cysteine proteinase. All of these effects were enhanced by E. histolytica. The E. dispar infection persisted. Studies on G. duodenalis have also demonstrated that the gut microbiota can promote the pathogenicity expression but not the growth of the parasites (Torres *et al.*, 1992, 2000). Researches have been indicated that that bacteria present in the duodenal are partially responsible for stimulating the pathogenicity of G. duodenalis. In some studies, the ability of facultative and strictly anaerobic microorganisms from the duodenal microbiota to induce G. duodenalis pathogenicity in gnotoxenic mice was examined. microorganisms were taken from the biopsies of five children who had giardiasis symptoms. Protozoan multiplication in the various mouse groups was assessed by quantifying trophozoites in the small intestine and cysts in the feces. Infected mice displayed pathological modifications between control and infected mice; lastly, no pathological changes were observed in non-infected or conventional animals. As observed, controls animals did not develop intestinal pathological changes during Giardia infection. It was because fecal cyst levels did not differ among groups of mice during the experiments, these data funding suggest that other pathogenic protozoans of the intestine, intestinal microbiota structure have similar factors for Giardia infections but not for development of the protozoa.

Intestinal infection and helminth:

For a vast majority of parasitic worms, the intestine serves as the perfect home. Human gut mucosae and lumen are intimately interacting with flatworms, specifically cestodes belonging to the genera Hymenolepis, Taenia, and Diphyllobothrium as well as digeneans like Fasciolopsis, Heterophyes, and Schistosoma. In terms of nematodes, Enterobius vermicularis and geohelminths (Trichuris, Ancylostomatidae, Strongyloides, and Ascaris) are the most prevalent intestinal roundworms. While intestinal helminthiases are primarily studied in less-favored areas for the parasitic disease, recent studies in industrial countries have focused on the close associations between intestinal helminthes and microbiota and the latent "host-pathogenic" immune defense. These studies have arisen as a result of growing concerns about autoimmune disorders, asthma, Crohn's disease, childhood allergies, and ulcerative colitis (Patel *et al.,* 2008). According to Eckburg *et al.,* (2005), the human gut microbiota plays a crucial role in innate immune instruction, regulation of epithelial growth, and nutrition provision. There are many descriptions of notable variability and variations in the makeup of communities, all of which are congruent with the idea of a highly diverse ecosystem. There is a suggestion that helminth infections cause notable alterations in the amount of the microbiota in the gut. Molecules produced by intestinal nematodes have the potential to change the gut microbiota's environment. Walk *et al.,* (2010) have been shown that mice infected with the duodenal parasite "Heligmosomoides polygyrus" may results in alterations in the bacterial community composition of ileum "but not in the colon"; where the most bacteria species found were Lactobacillae spp. Remarkably, "H. polygyrus" can detect changes in the function of the colon's epithelial barrier and dramatically decrease colitis inflammation in mice (Elliott *et al.,* 2004).

Furthermore, following 21 days of infection, Li *et al.,* (2012) showed a substantial change in the microbiota of the pig colon inf3cted with Trichuris suis. According to Wu *et al.,* (2012), variations in the genera profusion of up to 13% were found, including Ruminococcus and Fibrobacter, can be attributed to the initial infection even if the parasitosis is not persistent. A significant additional factor associated with infections of helminth is the possible interplay of macrofauna, microbiota, and immunity of the host. There is evidence of a general decline in proinflammatory cytokines linked to chronic inflammation during helminth infections; additionally, autoimmune disorders are less common in geographic areas where parasitic infections are reported to be more common (Sewell *et al.,* 2002). Reports of recurrence of parasite infections exist (Sewell *et al.,* 2002). According to Reddy (2010), a lower contact to infective creatures in industrialized nations may be the cause of a mild immune system activation and a rise in the prevalence of autoimmune and allergy disorders in the general population. Evidence supporting the involvement of intestinal nematodes in preventing allergic reactions has been shown based on a number of research conducted in underdeveloped nations (van den Biggelaar *et al.,* 2004; Summers *et al.,* 2005a; Croese *et al.,* 2006; Leonardi-Bee *et al.,* 2006; Flohr *et al.,* 2009). The term "Hygiene hypothesis" refers to this phenomenon (Wills-Karp *et al.,* 2001; Weinstock and Elliott, 2009). Specifically, the way that infections of helminth can interact with the host immune system may benefit both the parasite and the host in terms of managing autoimmune illnesses (Maizels *et al.,* 2009).Understanding the relationship between gut microbiota, helminthes and "immune-mediated "intestinal inflammatory status—such as in celiac patients—is becoming more and more important as a result, according to a recent review by Bancroft *et al.,* (2012).Some studies have been focused on the infections of Trichuris sp., and took into account the immunomodulation caused by helminths as well as the integrated interface between the immune system and the microbiota, where in Th17 (T-helper) and Tregs (regulatory T cells) are impacted by the microbiota's action and can therefore influence the survival of parasites. Parasitic worms simultaneously create chemicals that might change the gut microbiota's environment. In addition to nematodes, it has also been reported that digeneans like Schistosoma mansoni can cause microbial disruption. Wang *et al.,* (2004) conducted a metabonomic investigation on mice infected with Schistosoma pneumoniae. The study revealed various intricate consequences of the disruption of metabolism caused by Schistosoma infections, such as compromised liver functions, disruption of amino acid metabolism, and disruption of acid cycle of the tricarboxylic. Furthermore, S. mansoni-infected mice have high excretions of trimethylamine, phenylacetylglycine, and p-cresol glucuronide in their urine, which suggests disruptions in the gut microbiota. This is likely because the altered microbial ecosystem brought on by the parasite increases the production of these microbial agents. Wang *et al.,* (2010) evaluated the findings of similar alterations at the Nuclear Magnetic Resonance (NMR) metabolic profiles during infections by Necator americanus, Fasciola hepatica, and other human helminth parasites. Similarly, Li *et al.,* (2011) recognized 12 urine and five fecal metabolites as biomarkers of Schistosoma infection, able to discriminate infected and not-infected mice, adding further evidence to the hypothesis that infection with S. mansoni either directly or indirectly controls microbial activity of the host intestine. Balog *et al.,* (2011) established the disruption of metabolites related to intestinal microbes and microbial co-metabolism during

a study based on urine retort of human and rodent hosts to S. mansoni infection.

NEMATOTODES AS A MEDICATION

Due to encouraging findings about the possible therapeutic benefit of worms or their compounds in animals, various human trials investigating seemingly benign helminthes, such as T. suis, a whipworm that infects pigs naturally, have been conducted. Promising outcomes were obtained when T. suis ova was used to treat colitis patients, and similar treatments are now being developed (Summers *et al.,* 2005b).

But particular consideration has to be given to any potential negative effects. The first worry is related to T. suis's potential for zoonotic disease. The group's systematics are still up for debate, and it's very difficult to define which species genuinely infect people. The second component concerns the likelihood that intestinal pig epithelial cells internalized by bacteria (such as Campylobacter jejuni) and subsequent bacterial invasion are associated to T. suis infection (Wu *et al.,* 2012). Finaly, studies have been suggested that various parasite species could operate as immunosuppressants or as amplifiers of allergic phenomena, hence the impact of helminth infections on allergic infections may change liable on the species (Pinelli, 2012).

PARASITE-REVIVING PROBIOTICS

Probiotics could also have the capability to stop the growth of some infections. Probiotics have been shown to be effective in treating, allergy symptoms, respiratory infections, and gastrointestinal disorders, as studied by Travers *et al.,* (2011). They can also obstruct pathogens through strain-specific machineries that depend on:molecule secretion ,competition, and immune initiation. Probiotics' effects on parasites, including helminths like Ascaris and Trichuris and protozoans like Cryptosporidium and Eumycerium, have been documented in a number of investigations. With respect to Giardia, a wealth of information has been gathered since Singer and Nash's initial study from 2000, which offered some indications that the extremely diverse signs of giardiasis in people and animals were probably caused by the intestinal flora's makeup. The impact of six Lactobacillus acidophilus strains and Lactobacillus johnsonii La1 probiotic bacteria on G. duodenalis strain WB trophozoites was investigated in vitro by Pérez *et al.*, (2001). The results showed that only L. johnsonii La1 significantly inhibited the proliferation of Giardia trophozoites. s. Humen *et al.,* (2005) verified the activity of L. johnsonii La1 (NCC533) in in vivo experiments wherein spleen cells from La1-treated animals demonstrated a cellular response to Giardia antigens and a protection against parasite-induced mucosal damage, ultimately leading to an infection resolution. Furthermore, by reducing or preventing Giardia trophozoites from adhering to the mucosal surface and inducing a humoral response, Lactobacillus casei MTCC 1423 strain and Enterococcus faecium SF68 were both successful in curing Giardia infection in probiotic-fed mice (Shukla *et al.,* 2008). (Benyacoub *et al.,* 2005). Several scientists have recently conducted additional assessments on the efficacy of several lactobacilli species in treating and preventing murine Giardia infection (Shukla *et al.,* 2009, 2010; Goyal *et al.,* 2011). The findings of Shukla and Sidhu (2011) and Shukla *et al.,* (2012), which demonstrated the beneficial effect of Lactobacillus casei in renourished BALB/c mice infected with Giardia intestinalis, validate the function of probiotics in mitigating the duration and cruelty of giardiasis by means of the physiological and morphological recovery of the gut. Regarding worms, Bautista-Garfias *et al.,* (2001) hypothesized that oral L. casei therapy looks to lower the load of Trichinella spiralis parasites in mice. In vivo and in vitro larvacidal effect of Enterococcus faecalis CECT7121, a probiotic with inhibitory effect of both Gram-positive and Gram-negative bacteria, has been shown to reduce Toxocara canis larvae amount in laboratory mice's lung and liver by up to 90% (Basualdo *et al.,* 2007; Chiodo *et al.,* 2010). It has been shown that the bioethanol-producing bacteria Zymomonas mobilis can shield mice against S. mansoni infections by more than 60% (Santos Jde *et al.,* 2004).

CONCLUSION

The host body, parasites and gut microbiota are intricately linked, creating a complex ecosystem in which changes to one of these elements trigger a counterreaction in the others. It is believed that an enhanced knowledge of the mechanisms that determine infections can only be attained by a -omics approach that incorporates comprehensive, interdisciplinary, and integrated activities from several perspectives. There are still many unanswered questions that need to be investigated. These include the mechanisms underlying the actions of probiotics in contrast to intestinal parasites and the potential for their healing use in the host, as well as the current interactions between microbiota, inflammatory processes, immune response and intestinal parasitic diseases. Last but not least, novel and fascinating fields like the investigation of the parasites and the metabolism of intestinal microbiota throughout chronic infection of the parasite, as well as connection to immunoregulatory systems, must be addressed within the context of an combined strategy.

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