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Review article

The persistence of amoebosis caused by *Entamoeba histolytica* in Nigeria and the role of malnutrition

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ABSTRACT. Amoebosis, caused by the protozoan parasite *Entamoeba histolytica* is a gastrointestinal infection and the second leading cause of death from parasitic disease worldwide. The disease is endemic in many developing countries and kills over one hundred thousand persons annually. Adequate nutrition composed of macro- and micronutrients in their balanced proportions is central to effective gut immune response and the homeostasis of commensal organisms in the gastrointestinal tract. *Entamoeba histolytica* is a gut pathobiont that can exploit a shift in nutritional status to cause amoebosis, with extra-intestinal complications. Although undernutrition is rarely a public health concern in high income settings, bioavailability of functional nutrients remains suboptimal. On the other hand, nutrient deficiencies constitute a chronic challenge in very low-income regions. This study sought to review the pivotal influence of malnutrition on intact microbiota and functional immunity, as determinants of susceptibility to amoebosis in the Nigerian example of tropical regions. The dynamics of the infection such as possible coinfection with opportunistic pathogens were also, evaluated. Based on the available reports, we posit that amoebosis is a common tropical infection perpetuated by malnutrition following poor living standard including unhygienic environmental exposure.

Keywords: amoebosis, malnutrition, tropical, *Entamoeba histolytica*, gut, pathobiont

Introduction

Amoebosis, a gastrointestinal infection is the second leading cause of death from parasitic disease worldwide. The disease is endemic in the developing world and is caused by the protozoan parasite, *Entamoeba histolytica*. Susceptibility to infections is closely linked with the nutritional status of the host wherein malnutrition, including undernutrition and specific nutrient(s) deficiency could adversely affect immune response of the host to certain enteric infections [1,2]. A similar common relationship exists between food quality and the microbial composition of the gastrointestinal tract (GIT) superimposed on the immunity of the host. Infections of gastrointestinal tract with protozoan parasites such as *E. histolytica* which outcome is a function of the pathogen's interaction with components of resident gut microbiota and immunity, have contributed significantly to the global burden of diarrhoea [3]. Malnutrition and young age are two prominent predisposing factors for amoebosis. Other identified risk factors include malignancy, pregnancy, alcoholism, and cortico-steroid medication [4]. Hence, acute diarrhoea is commonly associated with high infant mortality [5], with *E. histolytica* evolving as one of the major etiologic agents of paediatric diarrhoea in the tropical regions worldwide [6,7].

Poor prognosis and diagnosis tend to limit reliable update of informative epidemiological and clinical data on the infection occurrence. This follows the observation that more than 90.0% of amoebosis are asymptomatic, especially among individuals with chronic non-dysenteric colitis [8]. Clinical manifestation of amoebosis ranges from ulcerative colitis, Crohn's disease, microscopic colitis, and other forms of inflammatory bowel diseases (IBD), that may eventually result in chronic diarrhoea [9]. These gastrointestinal diseases are often associated with impaired absorption of micronutrients [2,10]. However, amoebic liver

abscesses during amoebosis is the most frequent complication with higher male occurrence [11–13]. This is related to the earlier observed gender variation in complication of the infection, where the lower prevalence of hepatic amoebosis in females was hypothetically attributed to the stimulation effect of oestrogen on the phagocytic system, enhancing resistance to *E. histolytica* [14].

Google Scholar and Pubmed databases were used to access relevant literatures. Search items used to generate study materials in this paper included amoebosis, *E. histolytica*, malnutrition, micronutrients, macronutrients, undernutrition, tropical diseases, gastrointestinal infections, protozoans, faeco-oral infections, opportunistic infections, impaired immunity, amoebic diarrhoea, pathobionts, flooding in Nigeria, functional nutrients, dysbiosis, enteric parasitic infections. More than 90% of the selected articles were published between 2010 and 2019. Prevalence of *E. histolytica* reported in the past decade and five years in Nigeria were compared using the Fisher exact test. Statistical result was evaluated at $p < 5\%$.

Amoebosis: a persistent tropical infection

The dynamics of *E. histolytica* infection depend on the characteristics of the population. Unlike in urban population, a seroprevalence study reported negative association of *E. histolytica* infection with consumption of unpasteurized cow milk and overcrowding, which are common risk factors in urban settings; but rather identified lack of access to potable water as well as illiteracy as major predisposing factors for amoebosis in the rural population [15]. Water is an identified epidemiological factor in the transmission of amoebosis. Thus, as an environmental parasite, *E. histolytica* has been detected in sewage and sea water, activated sludge, soil, surface and drinking water, some of which are used for irrigation of food crops, or taken as untreated drinking water, and can cause post-harvest contamination of edible fresh crops [16]. These have characterized some flood-prone tropical countries such as Nigeria, where recent reports (Table 1) suggested that amoebosis is a persistent tropical disease with similar predisposing factors [17–26], and despite various interventions, no difference in prevalence between last 5 years and last decade has been recorded (Fisher exact test, $p = 1.0000$).

Malnutrition and impaired immunity complicate amoebic diarrhoea

Diarrhoea as a symptom of infection has dual deleterious effect on the gut immune cells, whereby the cells already starved following loss and poor absorption of nutrients are simultaneously impaired to resist the infectious agent, thereby aggravating the clinical course of the infection [27]. In other words, amoebic diarrhoea initiates a cascade of poor utilization of nutrients, as the onset of diarrhoea sequentially leads to reduction in food intake following loss of appetite culminating in malnourished state, which further impairs the immune function to worsen the health condition (Fig. 1). One of the presumptive effects of malnutrition on immune cells may include suppressed interferon production (IFN-γ) and loss of

Table 1. Recent reports on the prevalence of amoebosis in Nigeria

Investigative studies	Prevalence [%]	Age range	Sources of infection
Orji and Okpala [17]	60.0	4 -15	drinking water, poor toilet facilities
Adedoja et al. [18]	75.1	1-15	malnutrition, improper personal hygiene
Reuben et al. [19]	26.7	3-20	open defaecation, poor sanitation, unsafe water
Obadiah et al. [20]	42.6	0-5	caregivers, poor sanitation
Simon-Oke and Ogunleye [21]	67.6	2-12	water and food, poor toiletry
Mbagwu et al. [22]	21.43	1-15	poor personal hygiene, food and water
Amaechi et al. [23]	16.0	0-14	poor sanitation, lack of healthcare
Iboyi et al. [24]	8.31	0-5	child caregivers, drinking water source
Mohammed et al. [25]	56.9	6-15	drinking water, personal hygiene
Mohammed et al. [26]	40.0	1-70	open defaecation, sewage disposal

Prevalence between last decade and past five years was not significant ($p = 1.0000$ Fisher exact test)

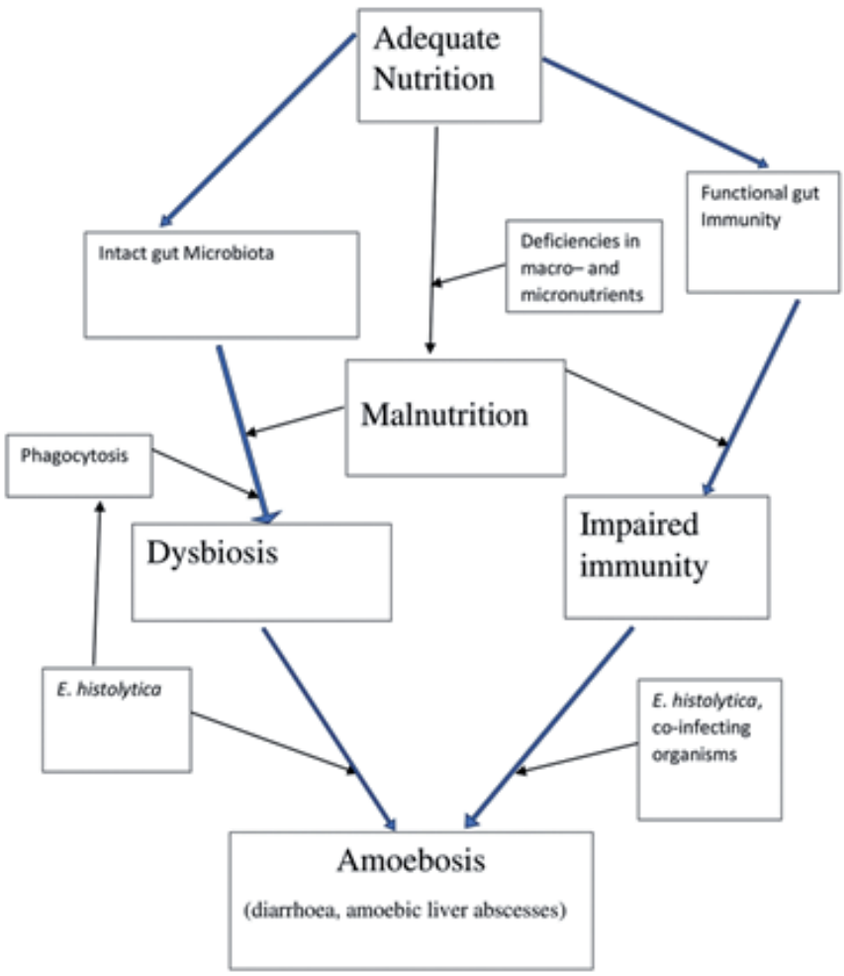

Figure 1. Schematic representation of the influence of nutrition on the gut microbiota-parasite interactions

phagocytosis by immune cells [5]. Immunosuppression can even cause retrogression of the infection to an advanced clinical stage including fulminant colitis. This was corroborated by the identification of corticosteroid medication as one of the key factors in degeneration of infections due to *E. histolytica* [28]. Similarly, chronic diarrhoea due to amoebosis commonly arise as a complication from immunocompromised state of a host, which makes the population more vulnerable [9]. Thus, chronic diarrhoea can be an indicator of amoebic colitis, and this has made it necessary to recommend screening for *E. histolytica* during diagnosis of individuals suspected to have HIV/AIDS [29].

Attenuation or progression of amoebosis is influenced by the parasite–gut microbiota interaction

The intestinal normal microbiome interacts with gut immune system in a manner that keeps invading pathogens in check, where in most cases they serve as adhesion barrier in resistance to infections [30]. Evidently, the role of host resident microbiome is indispensable in the pathogenesis and outcome of gastrointestinal infections [7]. *E. histolytica* is a pathobiont moderately present in the gut, and normally in commensal relationship with the host. Among the roles of resident pathobionts is alteration of the host immune response which can be beneficial in helping to clear the infection, or harmful worsening the clinical course with possible extraintestinal involvements. Thus, the outcome of gut microbiota–parasite interaction hangs in the balance because of a potential attenuation or promotion of virulence. Berrilli et al. [31] observed that while the host is protected from *E. histolytica* infection by the intestinal microbiome, however, under certain host nutritional status, such interactions tend to favour the parasite upregulating expression of its virulence. Emerging evidence has revealed association between composition of host

nutrient, including its absorbability or bio-availability, and susceptibility to amoebosis. This is based on the speculation that in some instances, the parasite feeds directly on the microbiota (Fig. 1) which in turn is sustained by the host nutrient, resulting in either loss or increase in virulence of the parasite through eubiotic or dysbiotic induction, respectively [11,32]. The virulence attenuation may follow a short course or become prolonged depending on the level of activation of certain components of the adaptive immunity [33,34]. Dysbiosis due to gut microbiota alteration often modulated by bioavailability of micro- and macronutrients [35], can trigger infection by *E. histolytica* [7]. This is consistent with the observation that colonic dysbiosis following malnutrition, and immunosuppressed conditions can induce a switch to virulence in *E. histolytica* commensal trophozoite [36]. A study implied close association between decreased population of *Lactobacillus* and amoebosis where *Lactobacillus ruminus*, represents the prime target of *E. histolytica* [37]. Investigating other risks of dysbiosis, Gaulke et al. [38] indicated that host's micronutrient status partially determines the extent of response during exposure to toxicants such as arsenic, which also can alter the gut microbiome.

The regulatory functions of nutrient on the colonization and progression of *E. histolytica* infection, and maintenance of homeostasis of both the gut immune system and resident microbiota [39], suggest that the parasite and gut microbiota interact directly or indirectly to determine the outcome of the infection [40]. Alternatively, metabolic products released via the activity of bacterial component of the gut microbiome on certain long chain polymeric substrates are utilisable carbon sources for the amoeba parasite. In addition to providing nutrition for the parasite, the microbiota also protects *E. histolytica* from oxidative stress (OS), thereby enhancing its successful infection [41]. The parasite resistance to oxidative stress in the gut is found to be *Escherichia coli*-mediated whereby *E. coli* malate dehydrogenase and the oxaloacetate product were strongly associated with this physiological role during the microbiota–parasite interaction [42]. Although *E. histolytica* requires interaction with intestinal microbiota to drive its pathogenesis, the parasite conversely needs the disruption of the microbiota homeostasis during infection. Invasion by amoebic trophozoites, activates the host immune response as a result of its interaction with mucosal immune cells [40]. Similarly, the extent of immune response to the parasitic infection depends on prior exposure to normal microbiome, since a separate finding showed a sharp contrast in amoebosis between animal tissues colonized by resident microbiota and the unexposed animal group [43]. Also, the secretory immunoglobulin A (sIgA) appears to be the major component of intestinal mucosa immunity produced in the intestinal lamina propria in response to amoeba invasion [44]. A decreased level of sIgA in the lamina propria of malnourished children has been correlated with amoebosis in the malnourished children [5].

Despite the immunological resistance to mucosal invasion by secretory IgA, *E. histolytica* can bypass the adaptive immune system through mechanism likely to involve enzymatic breakdown of intestinal lumen sIgA by its virulent cysteine proteases [45]. As the infection progresses and assumes chronic stage, the level of the host immune response to the infection diminishes such that anergic state sets in, giving way for the parasite to overwhelm the entire host immunity [44]. *Entamoeba histolytica* accomplishes immune cells anergy by inducing apoptosis of neutrophils and multiple cytotoxic effects which facilitate host immune evasion [45]. Moreover, as the infectious parasite transverses through the host cell, it adapts to counter the host defence. The secreted and membrane–bound cysteine proteases of *E. histolytica* is quiescent virulence factor which helps to maintain transmission and circulation of the parasite mediating encystation following insults from the environment [46]. Similarly, *in vivo* encystment of *E. histolytica* may constitute an important adaptive feature protecting the parasite from the prevalently low pH of the upper gut enroute to the small intestine [47].The parasite–microbial interactions can trigger an unusual mechanism of gut infection resistance in a susceptible host. One of the possible consequences of altered microbiota composition following a shift in the host nutritional status could be the introduction of a new component of the gut microbiota which may influence a change in innate immune response, conferring protection against unrelated pathogens, like *E. histolytica* in a trained immunity fashion [7]. This may contribute to the basis of therapeutic use of probiotics against certain infections. Findings by Moayyedi et al. [48] showed that a donor enteric pathogen like *E. coli* may provide an alternative approach for the treatment of

ulcerative colitis where the infection severity is reduced following administration of the therapy in some recipient individuals. A related *in vivo* study using a murine model, found that segmented filamentous bacteria (SFB) in the gastrointestinal tract is protective during amoebosis due to *E. histolytica* [49]. Their bloom in the gut may be beneficial as they were suggested to have immunomodulatory effect in enhancing postnatal gut immune system [50]. In contrast to the positive outcome enhancing infection resolution, the trained immunity which alters specific host immune function can initiate a cytotoxic pathway resulting in fulminant colitis and tissue damage due to exaggerated immune response [7,33]. There are other evidence indicating how host and coinfecting pathogens can modulate the pathogenicity pattern of enteric parasites. In one of the studies on the contribution of microbiota – *E. histolytica* interactions to host cell damage in the gut, Galvan-Moroyoqui et al. [51], observed that enteropathogenic infection of epithelial cell monolayer increases its susceptibility to amoebic cytolysis, while further cell damage is enhanced with the phagocytosis of the bacterial pathogen by *E. histolytica*. Also, during phagocytosis by the parasite, the host cell can be ingested along with microbial cells suggesting a possible link between efficient phagocytosis and virulence (Fig. 1). Similar to *Entamoeba* species representing unicellular, anaerobic, parasitic organisms; some enteric pathobionts are anaerobic with diarrhoeagenic potential. Thus, their coinfection of the colon could be detrimental as already demonstrated in a prevalence study, that disruption of the host mucosa during amoeba infection can result in an increase in host inflammatory response due to bloom of the *Prevotella copri* with consequent episodes of diarrhoea [52].

Bioavailability of nutrients regulate amoebosis

Macronutrients

Proteins function to generate new cells and regenerate or repair damaged ones during infections and or necrosis. The physiological role of protein as a macronutrient culminates in cell growth and consequently, it is a very essential nutritional requirement particularly in children. Unfortunately, malnutrition attributed to protein deficiency is consistently on the rise [53]. Moreover, the quality and nature of a protein can influence the progression or resolution of an infection. It was indicated for instance, that diets rich in collagen promotes virulence of amoeba, with a possible involvement of the secreted collagenase [54]. Although the therapeutic effect of protein diet in the treatment of gastrointestinal diseases such as diarrhoea is non-evident, glutamine amino acid however, was shown to improve the gut immune system where it also, functions as a vital component of the energy metabolism of the cells of GIT [55,56]. Ornithine, cysteine and other forms of dietary proteins show immunomodulatory and anti-inflammatory functions at low doses thereby improving the gut homeostasis [57]. This is often accomplished through their effects on apoptosis and regeneration of epithelial cells [58], while their opposite effects at high doses by excess production of nitrogen oxide (NO) and osmotic pressure, can be deleterious resulting in diarrhoea.

Glucose starvation enhances *E. histolytica* virulence, motility, and pectin expression [59]. There are instances when sugars are poorly absorbed such that colonic secretion is at an overwhelming level to cause osmotic diarrhoea [58]. Both the *E. histolytica* and members of the intestinal microbiome have glucose requirements for cellular functions which they must acquire from the host. However, scarcity of this basic macronutrient during undernutrition may induce a switch to the parasite's alternative pathway of obtaining the nutrient from other carbohydrate and non-carbohydrate precursors. The entire process tends to impose extra burden on the normal host cell homeostasis which may ultimately impact negatively to damage the cells thereby increasing the parasite's virulence. Enteropathogens' penetration of the mucosal epithelial cell surface is implicated in intestinal dysbiosis, exacerbating inflammatory reactions of the underlying immune system leading to the inflammatory bowel disease-associated diarrhoea [60]. Mucin as an essential biopolymer in the gut is often the target of *E. histolytica* when free glucose is limiting. *Entamoeba histolytica* among other species produce mucolytic enzymes enabling penetration of the mucus barrier while simultaneously inhibiting access and attachment of resident beneficial organisms to the epithelial surface [34]. Since breakdown of intestinal barrier often results in unregulated contact between the submucosal immune cells and immunogens including the microorganisms in the lumen and antigens in circulation, it is possible that this may represent the first step in the initiation mechanism of inflammatory diarrhoea [61]. This aligns with the

observation that the ensuing inflammation is exploited by the protozoan parasite for the progression and perpetuation of its virulence [62]. Although it was already indicated that cells of the gastrointestinal tract are constantly exposed to lumen bacteria [63,64], similar findings are necessary to unravel their interactions with protozoan parasites. The continuous degradation and impairment of the anatomical barrier provided by the mucus secretion, and subsequent penetration of the epithelial cells by trigocytotic process of the parasite may give rise to necrosis associated with amoebic colitis and dysentery [54,65,66]. Therefore, histolysis is an important pathogenic mechanism of *E. histolytica* that results from the combined effects of the parasite's secreted cytolytic enzymes and the complementary inflammatory response of the gut innate immunity. This observation probably reaffirms the evidence of leptin-mediated resistance to amoebosis found to be domiciled in the intestinal epithelial cells [67].

Composition of the gut membrane phospholipid is crucial to the virulence of *E. histolytica*. Unlike the stronger electrostatic interaction between bacterial cell surface and antimicrobial peptides (AMPs), there is seemingly inefficient interaction involving the mammalian cell membrane and these peptides. This observation was associated with the high cholesterol content of the mammalian cell membrane, a property implicated in the maintenance of the membrane phospholipid structure that interferes with the activity of the peptides [68]. The parasite can evade the innate immune system through the cell surface glycosylphosphatidylinositol (GPI)–anchored proteins. This lipopeptidoglycan (LPPG) forms protective layers indicated to shield the infectious form of the parasite from the immune complement [40]. It was shown that even though the parasite lacks cellular machinery for *de novo* synthesis of lipids and cholesterol, it can acquire these nutrients exogenously, mediated by lipoprotein or independent of any transporter [69]. Evidence of a link between dietary lipid level and enteric disorders is accumulating. Thus, Levine et al. [60] opined that the source of dietary fat may play an important role in promoting colitogenic bacteria. This understanding may imply similar ascending effect on coinfecting parasitic *E. histolytica* which readily explores a tip in gut microbiota balance. Earlier results of a mice *in vivo* study concluded that certain metabolic disorders following excess dietary lipid and carbohydrate intake common in obesity, may be a direct consequence of alteration in microbiota composition of the gut [50,70].

Amoebosis and micronutrients

Gut cells in a malnourished state are often deprived of the essential micronutrients, and this can worsen morbidity from diarrhoea [71]. Deficiency in micronutrients contributing to certain forms of electrolyte imbalance (hyponatremia, hypokalaemia, and hypocalcaemia) is a potent predisposing factor for fulminant necrotizing sequelae of intestinal amoebosis [72]. Hypokalaemia is multifactorial namely, decreased potassium intake, trans-cellular shifts, or increased potassium loss, with gastrointestinal losses indicated as the most common cause of hypokalaemia during severe or chronic episodes of diarrhoea. It was previously shown that zinc and selenium are important micronutrients stimulating immune resistance to parasitic infections [2]. Related findings concluded that dietary zinc is very beneficial to the host gastrointestinal tract through modulation of mucosal permeability, reduction of enterocytes apoptosis, and immunity to diarrhoeagenic pathogens [73]. Earlier studies on prevalence of intestinal disorders and low serum zinc concentration in children with diarrhoea established a strong association between high morbidity from diarrhoea and zinc depletion [71,74]. The metabolic importance of zinc and its involvement in maintenance of epithelial cell surface integrity may provide the basis for the claims of anti-diarrhoeal effect of Zn^{2+} [75].

Optimal calcium bioavailability improves gut health, modulating mucosal integrity against amoebic colitis [76]. Calcium homeostasis including its mobilisation and absorption is regulated by vitamin D levels in the gut, which also plays a veritable role in electrolyte uptake, and prevention of invasion by enteric pathogens [63,77]. These divalent ions such as Ca^{2+} and Zn^{2+} are therefore, physiologically important to the gut cells where they function to interfere with certain pathophysiological triggers of diarrhoea [78]. Anti-diarrhoeal effect of dietary calcium in both the immunocompromised and immunocompetent individuals has been described [79]. It was also, found that nutrient-based calcium can exert this effect by direct mechanism through specific interaction with extracellular receptor, CaSR in the gut, or indirect mechanism involving nonspecific binding to biomolecules [80]. While extracellular calcium sensing receptor has been

associated with stimulation of absorption and secretion inhibition, it was further shown to control the permeability and immunity of the intestine [81]. Although hypocalcaemia precipitating diarrhoea in an inflammatory gut have not been demonstrated in humans, an animal study had associated susceptibility to enterocolitis due to loss of mucosal integrity or breakdown by pathogens, with low Ca^{2+} micronutrient [78]. In their investigation, Li et al. [82] observed that one of the mechanisms by which vitamin D controls inflammatory bowel diseases (IBD) is by driving a balance shift in the intestinal mucosal microenvironment in favour of inhibition and blockade of inflammation, and intestinal epithelial cells apoptosis.

Micronutrient supplementation to an exceedingly high bioavailability level can alter the intestinal microbiota in favour of enteropathogens. It was reported that dietary iron may enhance the virulence of the parasite, whereby a correlation between infection mechanisms such as adherence, chromatic activity; and increasing iron concentration was reported [36]. Based on a previous observation whereby iron-supplemented diet resulted in the proliferation of gram negative enteropathogenic bacteria [83], it is arguable that gut pathobionts like *E. histolytica* could exploit such an iron-rich microenvironment to express their virulence. Moreover, since phagocytosis is essential for multiplication and virulence of *E. histolytica*, it is evident that the presence of these enteric bacteria and their subsequent phagocytosis by the parasite's trophozoite may positively impact its virulence [84]. Mg^{2+} is an important biomolecule which bio-availability and function are influenced by the extent of gastrointestinal absorption and rate of renal excretion, and consequently, hypomagnesemia is often associated with chronic diarrhoea among other factors [85]. Many enteral foods classified as functional nutrients play significant roles in regeneration and maintenance of the gut mucous membrane [86]. As a functional nutrient, dietary zinc in form of supplement has been efficiently used as a nutraceutical for diarrhoea and inflammation of the bowel, with the beneficial outcome in reduction of infant morbidity and mortality [74]. Interestingly, the physiological function of some micronutrients is coupled to the bioavailability of the other (i.e. mutually dependent). Results of an *in vitro* investigation revealed that the uptake of Zn^{2+} in the gastrointestinal tract where it is involved in the regulation of mucosal gland acidity and integrity, is facilitated by intracellular Ca^{2+} store [87].

Amoebosis, as with other enteric infections is associated with increased intestinal oxidative stress. The parasite survives in the resulting oxidative microenvironment by the secretion of an antioxidant enzyme trypanothione reductase, which neutralizes the harmful reactive oxygen species and free radicals [88]. Also, given that almost all cells possess the sequential immune response, SR1, which confers them with the ability to respond to stress, amoeba cells are able to protect themselves against microbicidal products such as hydrogen peroxide and singlet oxygen [89]. Similarly, exogenous supplementation of antioxidants such as vitamins can enhance host protection against OS damage in the gut [90]. Vitamins C and E particularly, serve as antioxidants and free radical scavengers, while protecting mucosal epithelium during enterocolitis, and often participate in damaged cell repair [35,91]. Flavonoids are natural pigments present in vegetables and they act as exogenous antioxidants as a result of their high iron-chelating ability [88]. Also, because of their antioxidant and anti-inflammatory properties, dietary polyphenols have been shown to interact with enteric microbiota selecting the proliferation of probiotic bacteria members [39].

Deficiency in vitamins impacts significantly on microbiota structure and the meta-transcriptome, wherein suboptimal bioavailability of vitamin A for instance, was correlated with increased population of growth-influencing members of microbiota such as *Bacteroides vulgatus* in young children [92]. Among the nutritional importance of vegetables is their antidiarrheal properties which are often comparable to loperamide conventional anti-diarrheal drug. This was corroborated by *in vivo* observation that *Piper nigrum* of the Piperaceae family was able to confer protection from diarrhoea, which may be associated with its calcium channel blocking effect [93]. Vegetables rich in flavonoids, alkaloids, tannins, steroids, and saponins pharma-cologically active components have been found to possess antidiarrhoeal properties [94]. In addition to their antidiarrhoeal properties, vegetables and other plant-derived nutrients containing sufficient quantity of tannins bioactive constituents are also, suggested to exhibit antimicrobial activity in the intestinal mucosal surface [95].

In conclusion, malnutrition plays a central and multifaceted role on the persistence of enteric parasitic infections. Consequently, the public health

burden of malnutrition-associated disorders and diseases have remained on the increase. Dysbiosis and impaired immunity due to malnutrition favours the virulence switch of *E. histolytica* to cause amoebosis (Fig. 1). Poor living standards such as inadequate nutrition and poor sanitation, resulting in contamination of drinking water during flooding and improper sewage disposal, have maintained the infection in the tropics. Thus, a renewed attention should be directed towards eradication of this neglected tropical disease through improved living conditions, health education and other public health services.

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