Air & Water Borne Diseases
ISSN: 2167-7719

Review Article Open Access

Keywords: Protist parasite; Pathogenesis; Motility; Adhesion; Phagocytosis; Calmodulin

Introduction

Human amebiasis is caused by the protist parasite *Entamoeba histolytica*, which is a major public health issue in developing nations. Even though research into the disease's pathobiological mechanisms has progressed signi cantly over the past few decades, little is known about the molecular pathways that lead to tissue damage and invasion in both intestinal and extraintestinal diseases. Clear linkage between the genotype of the parasite with intrusive infection or with extraintestinal attack has not been seen, however various destructiveness factors have been recognized lately. In amebiasis, the relationship between the host and the parasite is also in uenced by host factors, which include the micro ora of the gut and host genes like leptin. In addition to providing food, gut bacteria also provide an anaerobic environment and pH that encourage trophozoites to multiply and transform into cysts. It is progressively trusted that the stomach climate and parasite genotype, along with the host genotype, all connect to establish the right climate for *E. histolytica* to attack. We do not, however, have a clear understanding of the nature of these interactions or how they ultimately a ect the parasite's capacity to invade tissues [1].

during modi ed cell passing of trophozoites. Some of the nucleotidase enzymes are present in the inner membrane of cytoplasmic vacuoles that may or may not be phagolysosomes, whereas it was also found in the cytoplasm and near the nucleus. Additionally, it is unclear whether these enzymes contribute to this organism's calcium homeostasis. In *E. histolytica*, a repertoire of 27 CaBPs with multiEF-hands was discovered through genomic analysis [4, 5].

Cells for interest

E. histolytica adhesion to target cells is necessary for subsequent cell lysis and tissue invasion, as clearly demonstrated. *E. histolytica*'s hydrolytic and cytotoxic molecules or the stimulation of the apoptotic pathway that begins a er contact with the parasitic cells can directly cause the death of the target cells. Numerous proteolytically active genes are encoded and expressed by amebic cells. Among these, cysteine favorable to teinase 5 (Ehcp5) has acquired consideration since it is situated on the cell surface and due to the shortfall of a practically dynamic homolog in the nonpathogenic species *E. dispar*. Porin like proteins of *E. histolytica*, amebapores, were likewise ensnared in cytolysis done by amebic cells. A er *E. histolytica* interacts with target cells, one of their outcomes is a signi cant increase in Ca2+ levels. e death of the target cell was slowed down by blocking Ca2+ channels. Because the puri ed protein itself raises Ca2+ levels in target cells, it is hypothesized that Gal/GalNAc lectin initiated this. However, upon contact with *E. histolytica*, the mechanism by which target cells release Ca2+ is unclear [6, 7].

Phagocytosis process

Phagocytosis is closely related to *E. histolytica*'s biology. It has a high rate of pinocytosis and phagocytosis, resulting in a renewal of the plasma membrane every 30 minutes. It phagocytoses a variety of cells, including RBCs, bacterial cells, live and apoptotic cells from mammals, and so on. According to a number of reports, phagocytosis is essential to the virulence of amebae. e observed direct positive relationship between an isolate's phagocytic ability and virulence potential constitutes the majority of the evidence. In general, less virulence is correlated with a

5. Mann BJ (2006) Structure and function of the Entamoeba histolytica Gal/GalNAc lectin. Int Rev Cytol 216: 59-80.
6. Dodson JM, Lenkowski PW JR, Eubanks AC, Jackson TF, Napodano J (1999) Infection and immunity mediated by the carbohydrate recognition domain of the Entamoeba histolytica Gal/GalNAc lectin. JInfect Dis 179: 460-466.
7. Vines RR, Ramakrishnan G, Rogers JB, Lockhart LA, Mann BJ (1998) Regulation of adherence and virulence by the Entamoeba histolytica lectin cytoplasmic domain, which contains a beta2 integrin motif. Mol Biol Cell 9: 2069-2079.
8. Katz U, Ankri S, Stolarsky T, Nuchamowitz Y, Mirelman D (2002) Entamoeba histolytica expressing a dominant negative N-truncated light subunit of its gal-lectin are less virulent. Mol Biol Cell 13: 4256-4265.
9. Daniela M, Faust NG (2014) Cell-Surface Molecules as Virulence Determinants in Entamoeba histolytica. In Nozaki T, Bhattacharya A, editors. Amebiasis. Tokyo: Springer Tokyo.
10. Chadee K, Johnson ML, Orozco E, Petri WA JR, Ravdin JI (1988) Binding and internalization of rat colonic mucins by the galactose/N-acetyl-D-galactosamine adherence lectin of Entamoeba histolytica. J InfectDis 158: 398-406.
11. Yadav R, Verma K, Chandra M, Mukherjee M, Datta S (2016) Biophysical studies on calcium and carbohydrate binding to carbohydrate recognition domain of Gal/GalNAc lectin from Entamoeba histolytica:insights into host cell adhesion. J Biochem 160: 177-186.
12. Vaithilingam A, Teixeira JE, Miller PJ, Heron BT, Huston CD (2012) Entamoeba histolytica cell surface calreticulin binds human c1q and functions in amebic phagocytosis of host cells. Infect Immun 80: 2008-2018.
13. Moreau C, Cioci G, Iannello M, La f y E (2016) Chouquet A Structures of parasite calreticulins provide insights into their f exibility and dual carbohydrate/peptide-binding properties. IUCRJ 3: 408-419.
14. Ravdin JI, Sperelakis N, Guerrant RL (1982) E f ect of ion channel inhibitors on the cyto pathogenicity of Entamoeba histolytica. J Infect Dis 146: 335-340.
15. Ravdin JI, Murphy CF, Guerrant RL, Long-Krug SA (1985) E f ect of antagonists of calcium and phospholipaseA on the cytopathogenicity of Entamoeba histolytica. J Infect Dis 152: 542-549.