



Emergence and Characteristics of *Entamoeba histolytica*

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DESCRIPTION

Different kinds of amoeba are noticed in human. Some of them are non-pathogenic whereas some are pathogenic. Intestinal amoeba like *Entamoeba coli*, *Entamoeba nana*, *Lodamoeba butschlii*, *Dientamoeba fragilis* live as commensals in human large intestine. *Entamoeba gingivalis* is found in scrapings from teeth and gums of pyorrhoea cases. Dobell found *Entamoeba gingivalis* as commensal like *Entamoeba coli* which feeds on bacteria. *Dientamoeba fragilis* is generally non-pathogenic anaerobic amoeba. Yang and Scholten found few diarrhoea cases associated with *D. fragilis*. Talis found involvement of *D. fragilis* in disorders of gastrointestinal tract. *D. fragilis* to have closer affinity with flagellates (Mastigophora). Besides *E. histolytica*, free living amoeba of two genera *Naegleria* and *Acanthamoeba* are known to cause fatal primary amoebic meningoencephalitis. *Entamoeba polecki* a parasite of pigs has been found to cause human infections. Besides pathogenic *E. histolytica* which causes various types of symptoms such as amoebic dysentery, liver abscess, brain abscess in human beings, and non-pathogenic *E. histolytica* like amoeba have also been found to invade human beings e.g. Laredo, Huff 403, JA and AG strains. Neal found these strains to be less sensitive to anti-amoebic drugs and have little or no pathogenicity to human beings. These strains can grow at room temperature at 37°C and it can survive and complete its life cycle in a hypotonic medium. Lewis and Cunningham reported human intestinal amoebae from faecal samples of cholera patients. These amoebae were not considered pathogenic by them. Losch observed active amoebae in the stool samples containing blood and mucous. Autopsy of patient showed ulcers in the wall of large intestine which were full of amoebae. Losch fed and introduced these amoebae in rectum of four dogs. Amoebae could produce infection in one of the four dogs. He named these amoebae as *Amoebaecoli*. Later, it was designated as *E. histolytica* by Schaudin. Koch and Gaffky, Osier, Kartulis and Stengel reported the involvement of these amoebae in causing dysentery, liver and brain abscess. Quinck and Roos observed cysts of amoebae in stool of patients. They could produce dysentery in cat by introducing these cysts. *E. histolytica* has worldwide distribution. The distribution of laminal or asymptomatic infection measured by the presence of

cyst in stool is worldwide and may affect 5-50% of any given population. It was estimated that 450 million people were asymptomatic cyst passers. Serological surveys for antibodies which measures current and past infection indicated that approximately one tenth of the total number of infected people i.e. 48 million people have intestinal mucosal or liver invasion. Amoebic dysentery occurs 5-50 times more frequently than amoebic liver abscess. Amoebiasis causes death mainly when it manifests itself as liver ulcers or fulminating colitis. 2-10% of persons with liver abscess may die whereas mortality among fulminating colitis is 70%. Invasive amoebiasis accounts annually for 40000-110000 deaths in world. The disease is distributed throughout the world. But it is more prevalent in countries like Mexico, India, China, Eastern portion of South America, South east and West Africa, and the whole of South East Asia. In India it is estimated that the carrier rate of amoebiasis is 4-63%. The cyst passers are carriers and are the potential source of spread of the disease. Jalan studied asymptomatic cyst passers and concluded that asymptomatic cyst passer have invasive amoebiasis and need therapy.

Life cycle of *E. histolytica*

The life cycle of *E. histolytica* is simple as sexual stages and intermediate hosts have not been found. Infection with *E. histolytica* is acquired by ingestion of cysts in contaminated water or food. Excystation in small or large bowel leads to the release of quadrinucleate metacyst. This by a complex series of nuclear and cytoplasmic divisions gives rise to eight uninucleate trophozoites which on reaching the large intestine begin to feed on bacteria and food particles. In condition of non-invasive amoebiasis, the trophozoites continue to feed and multiply until encystment occurs. The immature cyst then undergoes two cycle of nuclear division to give the mature, infective quadrinucleate cyst which is excreted in faeces.

E. histolytica trophozoites range in size from 10-60 micron with an average size 25 micron. Trophozoites show presence of nucleus, peripheral chromatin and central nucleolus. The cytoplasm consists of a clear ectoplasm and granular endoplasm that

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contain numerous vacuoles. Eaton demonstrated Golgi dictyosome which was closely associated with the surface active lysosome. Proctor and Gregory found stack of smooth walled lamellae which was identified as Golgi complex. Golgi complex provides acid phosphatase and other hydrolytic enzymes to the cytoplasmic lysosomes and surface active lysosomes of *E. histolytica*. Electron microscopic studies of *E. histolytica* reveals

the presence of two types of lysosomes interior or cytoplasmic lysosomes which is required in digestive process and surface active lysosome which are located on the amoeba surface and may be used for attacking host cell. In electron microscope each surface lysosome appears as 0.1-0.2 micron wide surface depression in the amoebic plasma lemma, beneath a cub shaped membrane bound vacuole.