AUTOINFECTION WITH CYSTS OF *ENTAMOEBA HISTOLITICA* IN EXPERIMENTAL ANIMALS AND ITS BEARING ON RELAPSES OF TREATED CASES OF CHRONIC HUMAN AMOEBIASIS *

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ABSTRACT

The belief, without any adequate data, that cysts of *Entamoeba histolytica* formed in human bowel do not undergo any further development unless injected by a new host has led to the development of efficient amoebicides that are not absorbed and those that are tissue-acting in order to eradicate the trophozoites of *E. histolytica* that may be present in the lumen and in the tissues. Combinations of these amoebicides have not led to the satisfactory cure of chronic amoebiasis and relapses are encountered frequently after therapy. The drugs discovered so far have little or no effect of the cystic stage of *E. histolytica*. Critical review of literature on autoinfection in laboratory animals with cysts of *E. histolytica* and on factors causing excystation and encystation in protozoa suggests that the caecum and the large intestine are suited for both excystation and encystation to take place. For a rational approach to the problem of chemotherapy of amoebiasis, it is important that basic research should be carried out on such problems as excystation, encystation, chemical composition and permeability of the cyst wall. These studies will lead to the design and synthesis of chemical agents to deal with cysts.

IT is generally believed that mere eradication of trophozoites of *Entamoeba histolytica* from human cases of chronic amoebiasis will lead to the cure of the disease. Manson Bahr[1] in 1951 (p. 933) has stated—"Mature cysts do not undergo any further development in the intestine and, under normal conditions, do not hatch there, but acute infection of kittens can be readily produced by intra-rectal injection of material containing them. The process of conversion of the precystic form to the fully mature cyst takes place in the lumen of the bowel and occupies a few hours. The quadrinucleate cysts can survive in the bowel for two days, but do not hatch until injested by a new host. It has been suggested that some substance is present preventing further development. Fluid is apparently necessary for excystation; but from the quadrinucleate cyst a quadrinucleate amoeba emerges, subsequently dividing by nuclear mitosis into eight uninucleate individuals. This normally takes place when cysts are swallowed by a new host. The composition of the cyst wall renders it impervious to the action of the gastric juice, and excystment normally takes place in the alkaline contents of the small intestine". Faust and Russell[2] say (p. 188)—"Once a viable cyst is taken into the mouth and swallowed, it passes through the stomach into the small intestine. No apparent change occurs at levels where there is acid reaction but as soon as the medium becomes neutral or slightly alkaline the imprisoned amoeba becomes active. This, possibly combined with the effect of the digestive juices, weakens the cyst wall and allows the multinucleate amoeba (metacyst) to squeeze out through a small rent. Under conditions unfavourable for excystment in the small intestine the cysts may pass in the faecal stream into the large intestine and then be evacuated in the stool without excystation (Faust[3], 1941). There is no evidence that excystation occurs in the large intestine, either in the case of cysts in transit from the ileum or those newly formed in the colon (Swartwelder[4], 1937)." It may be pointed out that Swartwelder[4] failed to make the cysts of *E. histolytica* to excyst during a period of 2 to 8 hr when they were introduced into the caecum and colon of dogs *per anum*. By the use of the technique employed by Sellards and Theiler[5] for infecting kittens with cysts of *E. histolytica*, Swartwelder[4] found that one dog out of three got infected. In this experiment rectum was ligated following laparotomy and cysts were administered by a needle into the lumen above the point of ligation. Swartwelder[4] says that infection in one dog occurred under extremely abnormal conditions. Based on these studies, Swartwelder[4] has suggested that autoinfection by cysts does not occur. The findings of Swartwelder[4] are not reliable because the period during which he took the observations was 2 to 8 hr. Longer observations may have revealed excystment of cysts. In one dog where excystment took place it was due to the presence of sufficient quantity of excystment factor or factors to cause excystment during 8 hr and not due to the abnormal conditions. The factors causing excystation and encystation in

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protozoa and their bearing on chemotherapy of amoebiasis will be dealt later on.

According to Faust and Russell[2], under natural conditions encystation does not occur in the tissues in spite of the fact that Faust and Kagy[6] found cysts in the depth of the mucosa of dogs experimentally infected with *E. histolytica* and fed with raw liver. Faust and Russell[2] believe that as the faecal material containing trophozoites becomes dehydrated in the lumen of the colon, the amoebae get converted into mature cysts. Belding[7] believes that immature cysts of *E. histolytica* get mature outside the host.

Wilmot[8] says (p. 50)—"One often sees the statement that a particular drug is ineffective against the cystic form of *E. histolytica.* Cysts seen in stool preparations originate from trophozoites in the gut. Drugs do not destroy cysts; it is the trophozoites that give rise to the cysts that are killed. Hence a statement that a certain preparation is or is not effective against cysts should probably be interpreted as a statement that it does not eradicate trophozoites living in the lumen of the bowel."

The belief, without any adequate data, that cysts of *E. histolytica* formed in human bowel do not undergo any further development unless injested by a new host has led to the development of efficient amoebocides that are not absorbed and those that are tissue acting in order to kill the trophozoites of *E. histolytica* that may be present in the lumen and in the tissues. Combinations of non-absorbable with tissue-acting amoebicides have not led to the satisfactory cure of chronic amoebiasis (*see* Wilmot[8] for references). After the temporary disappearance of trophozoites from the stool, they reappear shortly after the cessation of therapy. According to Wilmot[8], relapses tend to occur, most frequently, during the first six weeks after treatment. Rees[9] has stated that most cures that have been followed for considerable periods post-treatment have relapsed.

Because of the relapses in treated cases of amoebiasis, Anderson, Bostick and Johnstone[10] have suggested that an ideal amoebicide should be absorbed and distributed in the active form so that it is effective systematically against trophozoites of *E. histolytica* in tissue as well as against cysts and motile forms in the bowel.

E. histolytica trophozoites have not been found to become resistant to amoebicidal agents. It is, therefore, difficult to explain relapses after combined therapy with amoebicides acting in tissue and in the lumen of the bowel. The drugs discovered so far have little or no effect against the cystic stage of *E. histolytica.* Thus it seems that when the effect of drugs disappears, the cysts excyst in the large intestine, the trophozoites feed on suitable bacteria, multiply and cause relapse. This is supported by animal experiments and laboratory data on excystment. Sellards and Theiler[5] successfully infected six kittens out of eight by injecting cysts of *E. histolytica* into the colon following laparotomy. Hoare[11] has exhibited sections of the intestine of a kitten infected with *E. histolytica* by rectal inoculation of cysts. According to Wenyon[12], Drbohlav has confirmed this finding. WHO[13] report in 1969 says that one of the most successful methods for experimental infection is the direct injection of faeces containing cysts of *E. histolytica* into lumen of the large intestine.

Factors causing excystation and encystation in protozoa.—We anticipate basic mechanisms in the morphogenetic events leading to excystation and encystation. These processes at the molecular level are little understood. It has been stated that many factors induce excystation and encystation. It is becoming increasingly evident that excystation and encystation are caused due to the presence of chemical agents in the environment which trigger these processes and not due to favourable and unfavourable conditions as has been assumed by certain workers.

The work on the factors causing excystation in protozoa has suffered a great deal because of the use of cysts that were not free from bacteria. Unless rigid aseptic conditions are maintained during excystment experiments and viable sterile cysts, free from micro-organisms, are used the results can be very misleading and of no value. Carefully planned experiments have, however, shown that bacterial environment is necessary for excystment in protozoa. Singh[14] in *Colpoda steinii* found that excystment took place in the presence of bacteria. There was no excystment when the cysts were moistened with soil extract without bacteria. He suggested that bacteria produce some substance that induces excystment. In *Didinium nasutum,* Beers[15] reported that hay infusion, lettuce infusion, peptone, tryptone and yeast extract were ineffective for excystment till there was profuse growth of bacteria in them. Heat destroyed the effectiveness of bacterized peptone. Distilled water, neutralized plant acid sugar mixtures, metabolites of *Paramecium* and change in pH value were ineffective. Peptone and amino acids showed no accelerative effect, though the hydrolysate and amino acids, which supported bacterial growth, induced excystment. Butzel and Horwitz[16] have also found that some metabolic

product of bacteria can cause excystment of cysts of *D. nasutum*. Crump[17] studied the excystment of two species of soil amoebae which were not free from living bacteria. She observed that *Amoeba*[4] (*Schizopyrenus russelli* Singh, 1952) could not excyst without the presence of bacteria. Crump[17] suggested that bacteria produce some material inducing excystment of cysts of *S. russelli*. According to Crump[17], *Amoeba Z* (*Didascalus thorntoni* Singh, 1952) readily excysted in distilled water.

Dudziak[18] has studied the influence of 15 different strains of soil bacteria on the excystment of an unidentified small soil amoeba. Excystment depended on the presence and type of bacteria. In the presence of certain strains there was practically no excystment. Similar findings have been obtained by Singh, Das and Saxena[19] in the case of *E. histolytica* by the use of viable sterile cysts.

In order to prove conclusively that actively proliferating cells of *Aerobacter* sp. produced in the medium an amoeba excystment factor, cysts of *S. russelli* were used by Singh, Mathew and Sreenivasaya[20]. A thin layer of non-nutrient agar (2·5% agar, 0·5% NaCl, pH 6·8–7·0) was poured into a sterile collodion cup, which was placed on the surface of a papain-digest meat agar plate heavily seeded with *Aerobacter* sp. Cysts were introduced on the 'non-nutrient agar surface in the cup. Great majority of the cysts excysted within 24 hr. In the control experiment only a few active amoebae could be seen. This experiment clearly demonstrated that *Aerobacter* sp. produced some substance that passed through collodion membrane and into non-nutrient agar and caused excystment of cysts. This factor is thermolabile and its chemical nature is not known. Kunicki-Goldfinger, Drozanski, Blaszczak, Mazur and Skibinska[21] have also observed that metabolites of bacteria cause excystment of soil amoebae.

Singh, Mathew and Anand[22] have reported that aqueous extract of *Aerobacter* sp. and *Escherichia coli* caused nearly cent per cent excystment of cysts of *S. russelli* at a suitable pH. Autoclaving of the extract at 15 lb/sq in pressure for 20 min had no adverse effect on excystment. A part of the excystment inducing property of the extract was due to the presence of amino acids. Some of these amino acids have been identified by paper chromatography. They also found that some of the chemically pure amino acids caused good excystment at a suitable pH. A definite concentration of an amino acid was necessary to give maximum excystment. These findings have been confirmed by Drozanski[23], using cysts of soil amoebae belonging to the family Hartmannellidae. Singh, Saxena and Iyer[24] and Singh, Datta and Dutta[25], using cysts of several species of small free-living amoebae, free from living and dead bacteria, have shown that bacterial extract gave nearly cent per cent excystment upto a dilution of 1/400. Contrary to the finding of Crump[17], the cysts of *D. thorntoni* gave hardly any excystment in distilled water. This shows that it is absolutely necessary to use cysts free from micro-organisms if reliable and consistent results are to be obtained on the factors causing excystment. Both D- and L-forms of amino acids at 2·0% concentration, pH 6·5, caused excystment of cysts of amoebae to varying degrees (Singh, Datta and Dutta[25]). The percentage excystment of cysts of *S. russelli* with several amino acids was much higher at 2·0% concentration than at 0·25% or 0·125%. When mixtures of these amino acids at lower concentrations were used, the percentage excystment was much higher than that obtained by individual amino acids at optimal concentrations. These findings suggest that nearly cent per cent excystment of cysts of different species of amoebae obtained by aqueous extract of *Aerobacter* sp. and *E. coli* may have been due to the presence of a mixture of amino acids and other factors in the extract. Rastogi, Sagar and Agarwala (unpublished data) have shown that a mixture of amino acids *plus* riboflavine caused more rapid excystment of cysts of *S. russelli* than that obtained by the mixture of amino acids.

It may be of interest to point out that Imam, Dutta and Agarwala[26] have shown that excystment agents, mentioned above, failed to cause excystment of cysts of *S. russelli* in the presence of emetine at pH 6·5. A very high percentage of the cysts excysted in the presence of the excystment agents after the removal of emetine, showing that the treated cysts were viable. This suggests the possible binding of excystment factor to emetine, thus preventing excystment. When cysts were treated with sodium lauryl sulphate, at concentrations not lethal to them, and then with emetine or with sodium lauryl sulphate and emetine together (pH 7·0), there was hardly any excystment. Sodium lauryl sulphate rendered the cyst wall permeable to emetine and the latter killed the cysts. Imam and Dutta[27] found that 2·0% trypsin at pH 6·5 or 8·5 caused good excystment of cysts of four species of small free-living amoebae, but it, in the presence of emetine, failed to cause excystment of cysts of these species of amoebae. When emetine was removed, the cysts excysted normally. Imam[28], from UV spectroscopic data,

has concluded that some interaction takes place between excystment agents and emetine, thus preventing excystment. Singh, Datta and Dutta[25] have reported that emetine in buffers at pH 8·2 or 9·2 killed cent per cent of the cysts of four species of free-living amoebae. At pH 5·5, emetine in phosphate buffers had hardly any cysticidal property. Emetine similarly in the presence of L-glutamic acid or in *E. coli* extract at pH 8–10 killed cent per cent of the cysts of *S. russelli*. These findings show that pH also plays an important role in rendering the cyst wall permeable to emetine.

Dobell[29] was the first to produce cysts of *E. histolytica* maintained with mixed bacterial flora. Amoebae growing with bacteria were transferred to the same medium supplemented with rice starch. The rapid growth of amoebae as a result of the addition of starch was thought to be a factor inducing encystation. Balamuth[30] showed that mass encystment occurred in *E. invadens* growing with *Clostridium perfringens* in the presence of persisting reducing conditions. Abrupt shift to oxidizing levels halted both growth of amoebae and encystation. Crowding and depletion of starch have been found to be correlated with encystment. Thepsuparungsikul, Seng and Bailey[31] have reported that sterile culture filtrates of bacteria caused 50 to 80% encystment of *E. invadens* trophozoites grown axenically.

Singh[32] has investigated in detail the role of very varied bacteria on the formation of cyst in a multinucleate amoeboid organism, *Leptomyxa reticulata* Goodey. Among 40 strains of bacteria readily and completely eaten by *L. reticulata* on non-nutrient agar, 15 strains led to the production of large number of multinucleate cysts. In the presence of remaining strains few or no cysts were produced. Out of 25 strains of partly edible bacteria, 5 led to the production of numerous cysts. Few or no cysts were produced in the presence of 27 strains of slightly edible or non-edible bacteria. *L. reticulata*, when maintained with some of the strains which readily produced cysts, continued to produce cysts. *Aerobacter* sp. was readily and completely eaten by *L. reticulata* but no cysts were produced after a few sub-cultures. When the organisms were grown with *Aerobacter* sp. over a year, they were supplied with 12 strains of bacteria that readily led to the production of cysts. No cysts were produced on any one of these strains. Thus it seems that *L. reticulata* loses the property of forming cysts after being maintained with *Aerobacter* sp. for a long time. In the first few months of its isolation and culture from soil with *Aerobacter* sp., it could readily produce cysts when grown with bacteria that led to the formation of cysts.

Neff and Neff[33] have discussed the biochemistry of encystment in *Hartmannella* sp. (*Acanthamoeba* sp.) grown axenically. They have come to the conclusion that multiplication stops during encystation and the amoebae go through a period of induction followed by morphological changes leading to the formation of mature cysts. During the process of encystment new kinds of macromolecules are produced. These can be blocked by inhibitors of the synthesis of RNA and protein.

Raizada and Krishna Murti[34], using axenically grown *H. culbertsoni* Singh and Das, 1970, have shown that 80 to 90% of the trophozoites encysted in 72 hr when they were placed on non-nutrient agar containing 0·015 M magnesium chloride and 0·02 M taurine, pH 6·8–7·0. Encystment was inhibited by 1×10^{-6} M mitomycin C, or 1×10^{-4} M cycloheximide or 1×10^{-7} M actinomycin D. Raizada and Krishna Murti[35] found that cyclic AMP, incorporated into non-nutrient agar, mimicked the combined action of magnesium and taurine in bringing about encystment. The encystation induced by cyclic AMP was inhibited by actinomycin D. Amoebae exposed to magnesium ions and taurine synthesized three to four times more cyclic AMP than the control amoebae. These findings lead to the conclusion that cyclic AMP is the mediator by which the metabolic machinery of *H. culberstoni* is geared for differentiation. Theophylline, a known inhibitor of phosphodiesterase, also caused induction of cysts. Thus the biochemical events that are triggered during differentiation are presumably regulated by the relative concentrations of cyclic AMP and the phosphodiesterase acting on it. Since activation of a membrane bound adenylcyclase is implied in the increased synthesis of cyclic AMP, binding of taurine on membrane of *H. culbertsoni*, using taurine-^{35}S, has been studied by Raizada and Krishna Murti[36]. The results show that in the presence of Mg^{2+}, significantly more of taurine-^{35}S was bound to the membrane. This binding was inhibited by cycloheximide. From the specific activity of taurine bound to the membrane it was calculated that a single cell of *H. culbertsoni* in the presence of Mg^{2+} is able to bind $3{\cdot}4 \times 10^6$ molecules of taurine. Raizada and Krishna Murti[36] (*see* also Krishna Murti[37]) have shown that during encystation of *H. culbertsoni* in the presence of magnesium ions and taurine, the macromolecular synthesis is stimulated. Significantly greater amounts of uracil-2-^{14}C into RNA and leucine-^{14}C or valine-1-^{14}C into protein are incorporated into

amoebae undergoing encystation than in the control amoebae. This is also true of the ability to synthesize cellulose as judged by the extent of incorporation of glucose-*u*-^{14}C into a polymer which was degraded into labelled glucose by a fungal cellulose. During encystation there is also massive breakdown of reserve proteins, polysaccharides and lipids. As a net result of metabolic stress imposed by the encystment agent or the mediator, cyclic AMP, there ensues extensive degradation of reserve polymer constituents and the resynthesis of new polymers needed for the architecture of the cyst wall. One of the primary molecular events of differentiation is the induction of enzymes mediating the biosynthesis of cellulose and mucopolysaccharide. Degradation of reserve proteins and RNA also occurs but new species of RNA appear by transcription of specific part of the amoebic genome that determines encystation and code for synthesis of new enzyme proteins. This transcription is obviously regulated by the factors that trigger encystation (Raizada and Krishna Murti[38]). The aerobic *H. culbertsoni* switches over to anaerobiasis and glucose carbon, instead of being channelled towards electron transport and energy fixation, is diverted for synthesis of cellulose and mucopolysaccharide. Raizada, Verma and Krishna Murti[39] have reported that biological amines, such as taurine, epinephrine, dopamine, 5-hydroxy tryptamine and tyramine, cause encystation of *H. culbertsoni* through the agency of cyclic AMP.

Since cyclic AMP is involved in the regulation of the biosynthetic process of encystation in *H. culbertsoni*, it would be of interest to find out whether in anaerobic amoebae belonging to the genus *Entamoeba* cyclic AMP is elaborated by trophozoites grown axenically. It would also be interesting to find out whether *E. histolytica* grown axenically excretes high levels of phosphodiesterase into the medium which prevent encystment or the trophozoites have lost the ability to synthesize the levels of cyclic AMP needed for triggering the mechanism of encystation.

The brief review of literature on factors causing excystation in protozoa, presented above, does not support the belief of Manson Bahr[1], Faust and Russell[2] and others that cysts of *E. histolytica* formed in human bowel do not undergo any further development unless injested by a new host. Due to the presence of bacteria in large numbers in the caecum and the large intestine, large concentrations of excystment factors would presumably be produced in these localities.

Large concentrations of biological amines could be present in the caecum and the large intestine due to the presence of bacteria. It is likely that these amines could act on the trophic form of *E. histolytica* and bring about encystment. This would support the views held by workers that encystation occurs in the large intestine. The environmental factors, which bring about encystation, may not be always present in optimal concentrations. The presence of these is regulated by the dietary habit of the host.

Autoinfection in laboratory animals by cysts of *E. histolytica* and laboratory data on excystation and encystation suggest that caecum and large intestine are suited for excystation and encystation to take place. Reinfection of *E. histolytica* is due to the ingestion of cysts. If amoebae are prevented from forming cysts in human bowel, it may lead to the eradication of amoebic dysentery. WHO[13] report has suggested that biophysical, biochemical and physiological studies should be made in order to elucidate the basis of encystment under both *in vitro* and *in vivo* conditions. For a rational approach to the problem of amoebiasis, it is important that basic research should be carried out on such problems as excystation, encystation, chemical composition and permeability of the cyst wall. These studies will lead to the design and synthesis of chemical agents to deal with cysts.

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DISCOVERY OF A KOMATIITE IN THE PRECAMBRIAN OF INDIA AND ITS SIGNIFICANCE IN THE NATURE OF ARCHAEAN VOLCANISM AND OF THE EARLY CRUST IN THE INDIAN SHIELD

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ABSTRACT

The paper reports the discovery of a basaltic komatiite in the Archaean of Singhbhum District, Bihar. Peridotitic and basaltic komatiites — first described from the 3·4 b.y. old Barberton greenstone belt of South Africa — are of unusual chemistry, characterised by high Ca/Al ratios, low Na and K, and high Mg and represent a group more primitive than oceanic tholeiites. Reflecting a low degree of contamination of mantle-derived magma, these rocks are now regarded as the first eruptible magma-types in Archaean greenstone belts.

The discovery suggests that (*a*) the nature of Archaean volcanism in India was similar to that of South Africa, Western Australia, and Canada: an early, primitive, komatiitic ultramafic-to-mafic phase, followed by a mafic-to-felsic cyclic phase of calcic to calo-alkaline character, and (*b*) the earliest crustal material that developed over the Indian region was possibly composed of ultramafic-to-mafic komatiite-type rocks.

INTRODUCTION

AN important petrological discovery of the late 1960's was the recognition by Viljoen and Viljoen[1] of the occurrence of an extrusive peridotitic rock in the 3·4 b.y. old Barbeton greenstone belt of South Africa[2]. As this ultramafic lava was first found in the Komati Formation of the Onverwacht Group, Barberton Mountain Land, it was named "peridotitic komatiite" and its associated mafic lavas were designated as "basaltic komatiites". Following the Barberton discovery, komatiitic volcanic rocks have been recognized in Canada, and Western Australia and it is now realised that there is a more widespread development of these rock-types than was previously known[3].

In this paper, we report the discovery of an occurrence of basaltic komatiite in the Precambrian of India and discuss its significance in deciphering the nature of Archaean volcanism and of the earliest crustal material that developed in the Indian shield.

CHARACTERISTICS OF KOMATIITES

The distinctive feature of komatiitic rocks is best brought out by their chemical composition, the most definitive characteristics being: (*a*) high Ca/Al ratios; (*b*) low Na_2O and K_2O contents, and (*c*) high MgO[2,3]. Komatiitic volcanic rocks, therefore, represent a group more primitive than the oceanic tholeiites, and their unusual chemistry reflects a relatively low degree of contamination of mantle-derived magma during its ascent to the surface,