Suid-Afrikaanse Tydskrif vir Geneeskunde
South African Medical Journal

Posbus 643, Kaapstad P.O. Box 643, Cape Town

Kaapstad, 9 Augustus 1958 Weekliks 2s. 6d. | Deel 32 No. 32 Vol. 32 | Cape Town, 9 August 1958 Weekly 2s. 6d.

THE EPIDEMIOLOGY OF AMOEBIASIS IN DURBAN

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The aetiological agent of amoebiasis, *Entamoeba histolytica*, is a cosmopolitan parasite whose distribution throughout the world is relatively uniform. Survey figures from various areas vary considerably, but there is good reason to suppose that most of this variation is due to selective sampling and to differences in laboratory technique. A fair average figure for the incidence of *E. histolytica* throughout the world would be 10%. From what evidence is available, it does not appear as if the incidence of this parasite is higher in places where the standard of hygiene is low.

A wide range of symptoms is often attributed to infections with *E. histolytica*, but it is difficult to be certain of the validity of this assumption in all cases. One can, however, be confident of the casual relationship in amoebic dysentery, its complications, and amoebic liver abscess. Symptomatic amoebiasis of this type is almost totally confined to rare, strictly localized epidemics and a few scattered endemic foci.

Blende (1955) has reviewed the sporadic epidemics. In all those which had been investigated in detail, there was, in a strictly localized group of people, the sudden appearance of large amounts of fresh sewage in the drinking water. When the link between sewage and water supply was removed, the outbreak was in each case halted, even though the parasites were still present in the population, and were presumably still being circulated by the routes available before the epidemic. A good recent example was the epidemic in a single factory in South Bend, Indiana, USA (LeMaistre *et al.*, 1956). The severely localized nature of the epidemics, and the immediate cessation of cases when the direct link between water supply and sewage was broken, makes the possibility of the coincidental introduction of a new pathogenic strain of *E. histolytica* in each of these epidemics remote. It would seem far more likely that either the number and kind of bacteria present in fresh sewage, or the number of amoebic cysts initially ingested, was the major cause of the new manifestation of the parasite.

The absolute dependence of *E. histolytica* on a living associate is well illustrated by the way in which certain antibiotics halt acute amoebic dysentery whilst not affecting amoebic liver abscess (Elsdon-Dew, Armstrong and Wilmot, 1952 and Wilmot, Armstrong and Elsdon-Dew, 1952). Further, in animal experiments, Phillips *et al.* (1955) have proved the inability of *E. histolytica* to survive and invade the bowel wall of germ-free guinea-pigs. In test-tube cultures, *E. histolytica* has not yet been grown in the absence of living cells. In our own experiments, we have shown that the mean size of a line of *E. histolytica* derived from bacteria-free human amoebic liver abscesses can vary greatly according to the bacterial associates provided (Freedman and Elsdon-Dew, 1958). In other experiments we have shown that a line of *E. histolytica* will form cysts in culture when accompanied by certain bacterial floras, but not when in the presence of others (Geekie, Freedman and Elsdon-Dew, 1958). As bacterial associates are so vital to *E. histolytica* in so many ways, it would not be surprising to find that they play a part in determining whether amoebae remain in the gut lumen as commensals or invade the tissues to become pathogens.

As to what the part played by bacteria may be, we have very little evidence, and most of that is confused. From the germ-free animal experiments for example, it is not clear whether it is for survival, rather than invasion, that the bacteria are essential. If bacteria are of importance for invasion, the epidemiological evidence suggests that the types responsible in man are likely to be those species which cannot survive long in sewage and sewage-contaminated materials.

The suggestion proposed here that the initial number of *E. histolytica* cysts ingested may play a part in deciding the course of an infection with this parasite has not been examined experimentally. It is based on the circumstantial evidence from epidemiological observations discussed in this paper, and by analogy with certain other protozoan parasites. An example of how different numbers of infecting parasites can alter the course of the disease which follows is particularly well illustrated by the work of Jankiewicz and Schofield (1934) on the coccidian, *Eimeria tenella*, which infects chickens. In their experiments they showed that doses of up to 150 sporulated oocysts produced no symptoms,

* The Amoebiasis Research Unit is sponsored by the South African Council for Scientific and Industrial Research, the Natal Provincial Administration, the University of Natal, and The United States Public Health Service (Grant E-1592).

150-500 caused slight haemorrhage but no deaths, 1,000-3,000 produced a fairly heavy degree of haemorrhage and a light mortality, 3,000-5,000 produced marked haemorrhage and a moderate mortality, while doses of more than 5,000 caused severe haemorrhage and a high mortality. The fact that clinical manifestations of *E. histolytica* are linked with bad hygiene, where large numbers of cysts would be ingested, suggests that some similar effect to the above may apply.

Durban is an endemic focus of symptomatic amoebiasis. In this city, there are 3 ethnic groups and in each the incidence of disease due to *E. histolytica* is different, although the incidence of the parasite itself appears to be much the same. In all its forms amoebiasis is common and severe in the African, but in the Indian and European, in that order, the disease is much less frequent and severe (Elsdon-Dew and Freedman, 1952). When the socio-economic backgrounds of the 3 groups are examined, it is clear that, in addition to the racial factor, hygiene and diet are also positively correlated with the frequency of the disease.

In considering the possible causes of the different behaviour of *E. histolytica* in the 3 groups in Durban, it is possible that the factors involved are different from those in the epidemics. However, it is far more likely that they are similar. Thus, the possibility of strain differences being responsible in Durban is as unlikely as in the epidemics. In Durban the Africans and Indians are the principal food handlers for the whole population, the former as cooks and the latter as market gardeners and purveyors of vegetables.

Diet as such played no part in the North American epidemics, but in Durban it may be of importance by (for example) altering the bowel flora to one favouring invasion. However, Africans with equally deficient diets in other parts of South Africa show no more symptomatic amoebiasis than their European neighbours. The possibility of climatic and topographical differences being important for the survival of bacteria and amoebic cysts must be borne in mind here as a possible complicating factor.

A racial difference *per se* seems unlikely to be involved in view of the fact that no racial discrimination was reported in the Thai Railway epidemic. Further, a people of closely related stock to the local African, the Natives of Lourenço Marques, have no more amoebiasis than their Portuguese coinhabitants (Elsdon-Dew, 1950). Lourenço Marques is only 300 miles to the north of Durban but the living conditions of the Natives are not of the slum type found in Durban.

In Durban then, as in the epidemics, we are left with the probability that either the types or number of bacteria in fresh sewage, or the initial number of *E. histolytica* cysts ingested, or both, underlie the invasion of the bowel wall by the parasite. In Durban, however, the process would be acting continuously owing to the bad hygienic conditions under which the majority of Africans in the area live.

Why the African does not have a higher over-all incidence of *E. histolytica* than the European is a paradox for their incidence of helminthic parasites is very much higher. (In Cato Manor, a bad slum area of Durban, 80% of the Africans examined had at least one species of helminth present—Elsdon-Dew and Horner, 1958.) Perhaps the body has certain natural defences against the establishment of *E. histolytica* in the bowel. Some such concept might well fit with the suggestion postulated above, that the number of cysts initially ingested, is the prime factor in deciding the future course of infection.

SUMMARY

Though on the evidence available we are unable to rule out completely the possibility that diet plays a part, it seems probable that the bad hygienic conditions under which the Africans live in Durban are the major cause of their high incidence of amoebic dysentery, its complications, and amoebic liver abscess. The ingestion of a very large number of *E. histolytica* cysts, or possibly the simultaneous ingestion of certain types of bacteria from fresh sewage, is the postulated precipitating factor which induces the amoeba to invade the bowel wall.

I should like to thank my colleagues in the Amoebiasis Research Unit, particularly Drs. R. Elsdon-Dew and A. J. Wilmot, for valuable discussions and suggestions on the subject matter of this paper.

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