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THE WAR AGAINST PARASITES: WHO IS WINNING?

BY

JOSHUA POPOOLA OYEWOLE OYERINDE

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THE WAR AGAINST PARASITES: WHO IS WINNING?

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BY

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Dedication

Ernest Rutherford, founder of nuclear science, once wrote: "It is not the nature of things for any one man to make a sudden violent discovery; science goes step by step and everyone depends on the work of his predecessors. Scientists are not dependent on the idea of a single man but on the combined wisdom of thousands of men."

This lecture is therefore specially dedicated to those who continue to fight in the battle against parasites to bring succour to millions, who suffer needlessly from parasitic diseases, in the developing countries.

INTRODUCTION

Preamble

It is during the preparation of this lecture, the 116th Inaugural Lecture of the University, the 32nd during the current administration and the 3rd from the Department of Medical Microbiology and Parasitology, entitled "THE WAR AGAINST PARASITES: WHO IS WINNING?", that the joy of sharing aspects of the world of parasites with the entire citizenry, both in town and gown, dawns on me. For this unique opportunity, I give the glory, honour and adoration to God the Almighty who has seen me this far.

What is a Parasite?

Any living organism, be it an animal, a bacterium, fungus or virus, that maintains an intimate association with a different but bigger and stronger species of animal or plant and depends on it, at least for its nutritional requirements and which is potentially injurious to, or which actually injures that bigger and stronger species is a parasite. The other species in the association is called the host. Conventionally, the term "Parasite", is restricted to animals that are parasitic.

It is interesting to know that every major group (phylum) of animals has its parasitic representative. The phylum, to which we humans belong, has its parasitic representative in the lamprey, which is attached to, and sucks the blood of other fishes.

Historical Aspects of Parasites

A search into the annals of medical history reveals that parasites infecting humans have existed since the early days, in fact, as far back as 1250 B.C.

For example,

1. *Schistosoma haematobium* eggs were recovered from the kidneys of the remains of XXth Dynasty Egyptian mummies (Ruffer 1910, 1921).

2. Haematuria, a symptom of *Schistosoma haematobium* infection, was written in the records of patients of the XXth Dynasty Egyptians (Sigerist 1951).
3. Other parasites, notably *Taeniarhynchus saginatus* (a tapeworm), *Ascaris lumbricoides* (the big intestinal round worm), *Dracunculus medinensis* (the guinea worm) as well as fleas, lice and mosquitoes were known during the same era.
4. The laws of Moses, Leviticus 11: 4 – 7, Deuteronomy 14: 7 & 8, forbid the eating of certain animals, such as camel, hare, pig (swine) and rabbit (shaphan). These biblical laws of public health significance have variously been cited as indicating awareness of the existence of tapeworm and *Trichinella* larvae. In the Book of Numbers 21: 4, reference was made to fiery serpents which are thought to be guinea worm, while 1 Samuel 24: 14 alludes to the flea, an ecto-parasite. The list is inexhaustible.

Malaria most certainly afflicted the ancient Egyptians. Many scientific works of the period recorded that the devastating effect of malaria may be traced back to Greek mythology (Deaderick 1909). Malaria was mentioned in the Orphic poems, that is, before 1000 B.C. Hippocrates recognised the intermittent nature of malaria episodes and characterised them as occurring daily, every 48 hours or every 72 hours.

Diarrhoea and dysentery were described in Chinese and Hebrew writings, that is, in the earliest times. It is on record that epidemics of diarrhoea ravaged the army of Napoleon during his invasion of Russia and also of Darius' army during his campaign against the Greeks. However, there was no definite link of diarrhoea with any parasite.

Awareness of the existence of parasites has been attributed to ancient Greeks and Romans. The Greek physician, Hippocrates (460–350 B.C.), the acclaimed father of medicine was credited with the knowledge of pinworm of horses (Pagenstecher 1879-

1893). The ancient Chinese physicians (300–200 B.C.) had record of parasites and in the Americas, the existence of parasites dates back to the pre-Columbian times (Ocaranza, 1934; Lastres, 1951).

However, it was in 1684 A.D. that Francesco Redi searched for and actually found parasites in the intestine and other organs of humans, other mammals, birds and fishes (Redi, 1684). For this feat, he was acclaimed the founder of Parasitology.

PARASITES AND THEIR EFFECTS ON HUMAN HEALTH Virtually every part of the human body has been successfully colonised by parasites

Parasites Inhabiting the Body Surface

The body surface is inhabited by fleas, lice, ticks and mites. Important species of these parasites include:

(a) The human flea (*Pulex irritans*)
(b) The tropical rat flea (*Xenopsylla cheopis*)
(c) The dog flea (*Ctenocephalides canis*)
(d) The body louse (*Pediculus humanus var coporis*)
(e) The head louse (*Pediculus humanus var capitis*)
(f) The pubic or crab louse (*Phithirus pubis*)
(g) The soft or Agasid tick (*Ornithodoros moubata*)
(h) The hard tick (*Dermacentor andersoni*)
(i) The itch mite

Effects on Health:

(a) The bites of these ecto-parasites induce intense pruritus and itching, thus causing a lot of irritation and discomfort to man and provide easy access for bacterial infection.
(b) Lice infestation lowers the social status of man. A louse-infested person is looked down upon by his associates.
(c) Ecto-parasites transmit a number of infections e.g. various forms of typhus, (flea borne, louse borne, tick borne and scrub typhus), various forms of fever (trench fever, relapsing fever, Q-fever), bubonic plague as well

as tapeworm infections (*Hymenolepis nana*, *H. diminuta*, *Dipylidium caninum*)

Parasites Inhabiting the Alimentary Tract

Perhaps the part of the body mostly colonised by parasites is the alimentary tract.

(a) *Entamoeba histolytica:*
A protozoan parasite, only visible with the aid of the microscope, invades and feeds on the intestinal wall and body organs, producing ulcers in the large intestine and abscesses in the liver, lungs, brain and other organs. It is a very common infection in all classes of people in both rural and urban communities. In Metropolitan Lagos, we recorded a prevalent rate of 11.2%, with adults found to be more frequently infected than children (Oyerinde *et al.* 1977, 1979).

(b) The tapeworms (*Cestodes*):
(i) Beef tapeworm (*Taenia saginata*)
(ii) Pork tapeworms (*Taenia solium)*
Man is infected, when he eats inadequately cooked infected beef or pork. Both tapeworms, mentioned above, are associated with intestinal discomfort, hunger pains, indigestion, diarrhoea, vomiting and loss of appetite. They have been associated with appendicitis. In addition, the pork tapeworm larvae (*Cysticercus cellulosae*) could develop in any part of human body particularly the brain, eyes and other vital organs with resulting consequences including epilepsy and mental disorders. Infection rate is very variable in Nigeria ranging between 0.01% and 33.3% (Oyerinde, 1982a).

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(c) The big intestinal round worm, (*Ascaris lumbricoides):*
Infection with this parasite results when infective eggs in human faeces contaminate our food or drink and such food or drink is ingested. The infection is associated with

pneumonitis, cough and fever. Heavy infection may cause broncho-pneumonia, abdominal pain, and intestinal obstruction. Recently Agbakwuru *et al.* (1998) reported a case of intestinal obstruction in a 12 year-old girl due to a load of 2050 *Ascaris* worms. Individual worms may enter the appendix, causing appendicitis, bile duct and pancreatic duct openings, leading to degeneration of liver tissue and sometimes death. About 1 billion people were estimated to be infected world wide (Walsh and Warren 1976) while about 21% of the population in sub-Sahara Africa were reported to be infected (Andrew and Man-Suen 1974). Figures ranging between 2.5% and 90% have been recorded in Nigeria (Oyerinde, 1982a)

(d) The whipworm, (*Trichuris trichiura):*
So called because of its whip-like appearance. Infection, again is by ingestion of infective eggs in human faeces with food or drink. Light infection may result in nervousness, diarrhoea, weight loss and anaemia in malnourished children. Heavy infection may induce inflammatory changes and rectal prolapse. In Nigeria, infection rate ranged between 2.8% and 70.4% (Oyerinde, 1982a).

(e) The Hookworms:
(i) Old world hookworm (*Ancylostoma duodenale*)
(ii) New world hookworm (*Necator americanus*)
Infection with these parasites results from penetration of the infective larvae into the skin when people walk bare-foot on soil contaminated with human faeces. The penetrating and migrating larvae in the body induce dermatitis (ground itch), lung symptoms (bronchitis, pneumonitis), gastro-intestinal symptoms (nausea and vomiting). Adult parasites bite into the lining of the small intestine and feed on blood and tissue fluid causing iron deficiency anaemia, diarrhoea, mental and

physical retardation of the child. An infection rate of 26.6% was recorded for metropolitan Lagos. (Oyerinde 1982b).

Parasites Inhabiting the Blood and Other Body Fluids

(a) Malaria Parasite (*Plasmodium falciparum*):
Man is infected with this parasite when an infective female *Anopheles* mosquito bites him. The parasites live and multiply in the red blood cells which ultimately rupture, releasing the parasites and the metabolic waste products. These circulate in the blood, induce fever often with complications such as severe anaemia, diarrhoea, cerebral malaria, renal and hepatic failure. Infection may terminate fatally unless prompt treatment is instituted.

Current estimates show that between 300 and 500 million clinical cases of malaria are recorded annually world-wide (WHO, 1996). Out of this figure, Africa records 90% with one million paediatric deaths. In Nigeria, mortality rate was estimated at 100,000 annually while 10% of children below 14 years of age die of the disease(Osisanya, 1985).

(b) *Trypanosoma brucei gambiense:*
The cause of sleeping sickness in tropical Africa. It is transmitted by the tsetse fly (*Glossina* spp.). The parasites pass into and multiply in the lymph, the blood and later in the cerebro-spinal – fluid, inducing generalised swelling of the lymph nodes when patient develops irregular fever, high pulse rate and head-ache. The liver and spleen become enlarged and later on, nervous symptoms, including headache, dizziness, visual disorders, muscle spasm, non co-ordination of movement, exhaustion, somnolence (sleepiness) develop. This is the sleeping sickness stage when patient falls asleep unceremoniously even at a meal or when standing. If not treated, the infection terminates fatally.

(c) The blood flukes or the schistosomes (*Schistosoma* species):
Schistosomes live in the blood. This can be either in blood vessels around the pelvis and urinary bladder (urinary schistosome) or in

blood vessels around the colon and rectum (intestinal schistosome). The blood flukes are fresh water associated parasites. Infection is by direct penetration of the infective stage (cercaria). While penetrating into the skin they induce dermatitis. Their migration in the body is associated with fever, pain all over the body, rashes, enlarged liver and spleen, pneumonia. Cough and bleeding may also occur in the lungs. In *S. haematobium* infection, there are also urinary tract symptoms with serious complications including bladder neck obstruction, urethral strictures and carcinoma of the bladder. In Nigeria, the parasite occurs in all the States of the Federation. Infection rate of up to 100% has been recorded among school children in most water and agricultural development project areas (Mafe and Olawuyi, 1997)

(d) *Wuchereria bancrofti:*

This is the cause of elephantiasis (gross enlargement of the extremities, legs, hands, scrotum, breasts). The parasite is transmitted by mosquitoes. Infection is associated with fever, mental depression, frontal headache and physical exertion (weakness in arms and legs).

Parasites Inhabiting the Body Organs and the Cutaneous Tissues

(a) The lung fluke (*Paragonimus uterobilateralis*):

This is the lung fluke of man, occurring in West African countries. Infection results from ingestion of raw or inadequately cooked crab or crayfish. The effect of the parasite varies depending on the organ in which the parasite is located. In the lungs it is associated with inflammatory changes. This results in fever, cough with pains in the chest, and blood stained productive sputum is produced. Those in the gut wall result in abdominal pain, diarrhoea and passage of blood and mucus in faeces. Those in the brain induce nervous symptoms, and paralysis of any part of the body may occur.

(b) *Onchocerca volvulus:*

This is the cause of river blindness. The parasite is transmitted by the blackfly (*Simulium* sp.). Adult parasites inhabit the sub-cutaneous tissue of man in most parts of Africa including Nigeria.

They are usually enclosed in host's fibrous tissue, forming nodules, the onchocercal nodules, under the skin over bony regions of, for example, pelvis and forehead etc. The embryos (microfilariae) migrate in the surface layers of the skin, giving rise to intense itching and rashes, which provoke the infected person to scratch. The skin becomes wrinkled, thickened or even depigmented. Often in some cases hanging groin, elephantiasis of the genitalia and general debilitation, including weight loss may develop. Heavy microfilarial load may produce extensive inflammatory reaction involving the optic nerve. In such cases, serious visual impairment including blindness may result. Mortality among blind persons may be four times as high as among non-blind persons of the same age in the same community (Samba, 1994). Onchocercal situation is shown in Table 1.

Table 1 Onchocerciasis situation

	World-wide	Nigeria
Number exposed	40 million	18 million
Number infected	20 million	1 million
Cases of total blindness	Not available	120,000
Other complications	Not available	1,000

Edungbola (1991), WHO (1995)

(c) The Guinea worm *(Dracunculus medinensis)*:

This parasite lives in the deep cutaneous tissue for between 9 and 12 months to mature, after which it migrates to the sub-cutaneous tissue and later burrows its way out through a blister. Guinea worm seriously debilitates human health. The toxic by-products of the worm induce allergic reactions. There is fever, nausea, vomitting, anorexia, pruritus and urticaria rashes. The cutaneous lesion is associated with intense painful itching. Infection may be complicated by secondary bacterial infection, particularly tetanus. There may be abscess formation, joint fixation and deformity. A few cases terminate fatally. Guinea worm disease also indirectly adversely affects agricultural production as well as school attendance (Table 2)

Table 2 Guineaworm situation in Nigeria

Number infected	2,5 million
Number temporarily incapacitated	1 million
Number irreversibly disabled	12,000 per annum
Total farm-day loss	50 million per annum
Fall in school attendance	40 million pupils day per annum

Edungbola (1985)

CONTROL EFFORTS

Mr. Vice-Chancellor Sir, distinguished ladies and gentlemen, the unprovoked activities of these parasites can only be interpreted as a declaration of war on human existence, a war we have been fighting with all the weapons at our disposal but who is winning?

Soil Transmitted Parasites

Most soil-transmitted parasites of human are gut inhabiting. Their eggs, cysts or larvae are deposited with faeces into the human environment, around human habitation, where they develop to the stage infective to man or to the intermediate host, from where they are transmitted to humans. To date, as far as I know, there is no organised control programme, nationally or locally in the country, for these gut-inhabiting parasites except treatment of individual cases. However, an international workshop held at the Conference Centre, Obafemi Awolowo University, Ile-Ife from 7th to 9th May, 1990 at which I was privileged to be a participant, considered and debated exhaustively, actions for the control of soil transmitted helminthic parasites in Nigeria. The workshop concluded its deliberations by putting forward 12 recommendations for action to control soil transmitted helminths in Nigeria. As far as I know, the document is still to receive positive action from the appropriate authorities.

Insect Transmitted (Borne) Parasites

(a) Malaria Parasites: Control of malaria parasites prior to 1900 centred around treatment of cases and prevention of infection by the use of drugs, mainly quinine. The aim was to reduce mortality and morbidity. In the forties, other drugs, paludrine (proguanil) daraprim (pyrimethamine) and chloroquine were introduced. In the fifties, efforts were directed to vector control using insecticides and other anti-larval measures to supplement available drugs (chemotherapeutic and chemoprophylactic preparations). These control strategies were thwarted by the emergence of drug resistant *Plasmodium falciparum*, the most virulent and the main aetiology of malaria in Nigeria, as well as emergence of insecticide resistant *Anopheles* mosquito that transmits the malaria parasite. Presently, research efforts are being directed towards development of malaria vaccine, which hopefully, will not only control but also eradicate malaria parasites, world-wide, if our experience with some bacterial and viral diseases, such as smallpox is to go by. It is heartening to report that potent sporozoite and erythrocytic stage vaccines for *Plasmodium falciparum* have been developed and are presently undergoing tests in human volunteers (Stoute, 1997). However, malaria parasites are host-specific (Greenwood, 1997) and, as far as I know, not much work has been done on the antigenic specificity of the different species and the different geographical strains of the same species. Therefore, before these vaccines could be put into general use, a lot of questions must be answered. These include the following:

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* Is the malaria vaccine effective against all strains of *P. falciparum* or against other species of malaria parasites?
* What is the duration of the immune response?

* Does it protect semi-immunes and non-immunes?
* Is there any side effect in people of all ages and both sexes?
* Is it easily administered and affordable to the generality of the population?

Answers to these and similar questions may take time to find.

Malaria situation: In spite of our persistent efforts over the years to control malaria parasites, the prevalence has consistently been on the increase. Data obtained from the Federal Ministry of Health, Medical Statistics Division (1983/84), recognised malaria as one of the major causes of death from notifiable diseases in Nigeria, ranking 5th highest in 1978, 4th in 1979, 3rd in 1980, 2nd in 1981 and again in 1982, and 1st in 1983. During the same period (1978 to 1983) it was consistently the highest cause of morbidity of the 10 reported major notifiable diseases in the country. In 1985, mortality rate among Nigerians as mentioned earlier, was estimated at 100,000 annually while 10% of children below 14 years of age die of the disease (Osisanya, 1985). Malaria situation world-wide is shown in Table 3.

Table 3 Malaria situation world-wide

	1979	1980	1981	1982	1993	1996
No. exposed in million	N/A	N/A	N/A	N/A	N/A	700
No of cases in million	7.0[2]	8.0[2]	7.8[2]	6.5[1]	100[3]	300-500[2] (90% in Africa)
Mortality rate in million	N/A	N/A	N/A	N/A	N/A	2.5[2] (50% in Africans below 5 years)

N/A – Not available
[1] – WHO (1985)
[2] – WHO (1985)
[3] – Najer *et al.* (1993)

(b) ***Onchocerca volvulus:*** Up to the late 1940s, two anti-onchocerciasis drugs were in use for the treatment of the disease,

namely diethylcarbamazin, a very effective drug against the larvae (microfilariae) and suramin which kills adult onchocercal worm. Unfortunately, both drugs are associated with serious side effects, which preclude their administration on a large scale. Their use is therefore restricted to treatment of cases under strict medical supervision (Samba 1994). In 1968, the Onchocerciasis Control Programme (OCP) in West Africa was launched and activities in eleven countries began in 1974. The objective of the programme was to eliminate onchocerciasis as a disease of public health importance. The strategy adopted was to break the life cycle by destroying the larval stages of the vector, the blackfly (*Simulium*). This was later supplemented with mass treatment of endemic communities with ivermectin, a non-toxic microfilaricide which the manufacturers, Merck Sharp and Dohme (MSD) promised to supply to all endemic communities free of charge for as long as required, about 20 years, to bring the incidence of the disease to an insignificant level. The success of the strategy has been very encouraging and it is hoped all the eleven countries in the disease control programme should be free from onchocerciasis by the year 2000 (Hougard *et, al.* 1997) when the OCP activities will be wound up (Molyneux 1995) and its activities transferred to the respective governments of the participating countries. Unfortunately, Nigeria was not included in the countries participating in the onchocerciasis control programme. However, the Nigeria Onchocerciasis Control Programme (NOCP) was launched in 1985 and commenced activities in the following year. The main control strategy adopted was large-scale treatment with ivermectin to be supplied free of charge by the manufacturers (MSD). Although Nigeria has been guaranteed free supply of the drug, much is still expected from the various levels of governments (Local Government Authorities, State and Federal Governments) in terms of absolute commitment with respect to logistic support, political will and adequate funding among many others.

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Water Associated Parasites

(a) Guinea worm (*Dracunculus medinensis*): The first national conference on guinea worm disease in Nigeria was convened in Ilorin, Kwara State in 1985. The main objectives of the conference were (1) to assess the status of guinea worm disease in Nigeria and (2) to recommend appropriate steps to control or eliminate the disease. Following the conference, the Nigeria Guinea-worm Eradication Programme (NIGEP) was inaugurated, with the target date for complete eradication of guinea worm, set at December 1995. The main interventions used were health education including non-pollution of water supplies, boiling and provision of safe drinking water, use of filters, and chemical (temephos) vector control, where feasible. Periodic active case searches of the disease were conducted to assess the impact of control efforts, the result of which is shown in Table 4. The table shows that guinea worm cases have drastically reduced in number to 2% (about 12,000) of the pre-control intervention figure.

Table 4 Reported cases of Guineaworm disease in Nigeria

Year	No. of cases	Remark	Source
1984	8,777	Prior to control intervention	WHO (1989)
1985	5,234		
1986	2,821		
1987	216,484		
1988	653,497	First active case search (Prior to control intervention).	Guineaworm Report (1990)
1989	604,008		
1997	12,000	Approximately 2% of 1st active case search.	

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(b) The Schistosomes (Blood Flukes): In 1984, the then Federal Ministry of Agriculture and Water Resources (FMAWR) constituted a committee on the health component of irrigation projects. The committee was charged with the duty of looking into the health implications of irrigation projects with a view to evolving a pragmatic policy and programme of action for Government to embark upon. The committee met and reviewed the inherent hazards of irrigated agriculture and came to the conclusion that a programme of action must be evolved, which will lead to the formulation of a central approach for combating the health problems, topmost of which is schistosomiasis. A technical committee was set up to work out the scope of the baseline data collection vis-a-vis its cost element. The committee agreed that a sampled survey of all the project areas of each of the River Basin Development Authorities should be carried out and that the programme should be nationally co-ordinated. I was privileged to be nominated the National Co-ordinator. However the project, laudable as it was, had to be abandoned mid-stream for lack of logistic support from most of the River Basin Authorities as directed by the then Department of Water Resources.

Another effort at initiating National Control Programme was made by the Federal Ministry of Science and Technology in 1987. A technical committee was constituted. The committee, of which I was privileged to be a member, had hardly settled down to work when it was disbanded as a result of re-organisation of Federal Ministries, which transferred all health related programmes to the Federal Ministry of Health.

In August 1988, the Federal Ministry of Health inaugurated a National Expert Committee on Control of Schistosomiasis (NECCS) with a mandate to prepare a

blue print for the control of schistosomiasis. The committee:

1. identified 5 pilot project areas;
2. initiated health education campaign to promote awareness of schistosomiasis among the populace;
3. conducted a National schistosomiasis survey, and
4. embarked upon control measures using chemotherapy.

The control effort suffered a fate similar to those efforts preceding it, that is, lack of funds.

In December 1994, the Federal Ministry of Health and Social Services again inaugurated another National Expert Committee on the Control of Schistosomiasis with the following terms of reference:

1. To review the blueprint for the control and prevention of schistosomiasis in Nigeria.
2. To articulate and formulate a 5 year-control programme of activities for the country and
3. To technically advise the Honourable Minister of Health and Social Services on the control and prevention of the disease in Nigeria.

A Sub-Committee of the National Expert Committee, of which I was privileged to be Chairman, prepared a draft blueprint, which the National Expert Committee scrutinised and submitted to the Ministry of Health and Social Services. In it, a 5-year National Plan of Action was itemised. Although a limited degree of success was achieved, the programme had to be suspended due to lack of funds.

MY RESEARCH EFFORTS IN THE SPECIALISED FIELD OF PARASITOLOGY

Mr. Vice-Chancellor Sir, permit me to highlight very briefly some of my modest contributions to knowledge during my over 3 decades of interaction with parasites. In doing this, and because of time constraints, I shall restrict myself to 2 parasites – the

schistosome, a blood parasite and the hookworm, an intestinal parasite.

Background Training

I graduated B.Sc. Special Honours in Zoology from the University of London, with specialisation in Parasitology. I then proceeded to the Liverpool School of Tropical Medicine for a Master of Science Degree in Applied Biology. The courses included Medical Parasitology, Veterinary Parasitology, Medical Entomology, Parasites of fresh water Animals and Marine Biology, with specialisation in Medical Parasitology. Following this, I then chose to conduct research into the schistosomes for my Ph. D. degree.

The Schistosome:

At the time I was to register for a PhD Degree, the transmission and the scanning electron microscopes had just come into prominence. I therefore decided to study "the fine structure of *Schistosoma haematobium* and *Schistosoma mansoni*".

The Fine Structure of *Schistosoma haematobium*

My observation with the electron microscope revealed that the body surface of the parasite is not cuticular as revealed under the light microscope but that it is cellular, with the secretory (tegumentary) cells located in the parenchymma. The tegumentary inclusions (mitochondria, endoplasmic reticulum etc) are transported via cytoplasmic tubules to the body surface (Figs. I and) (Oyerinde 1970).

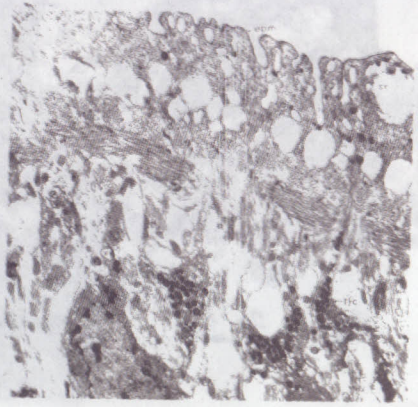

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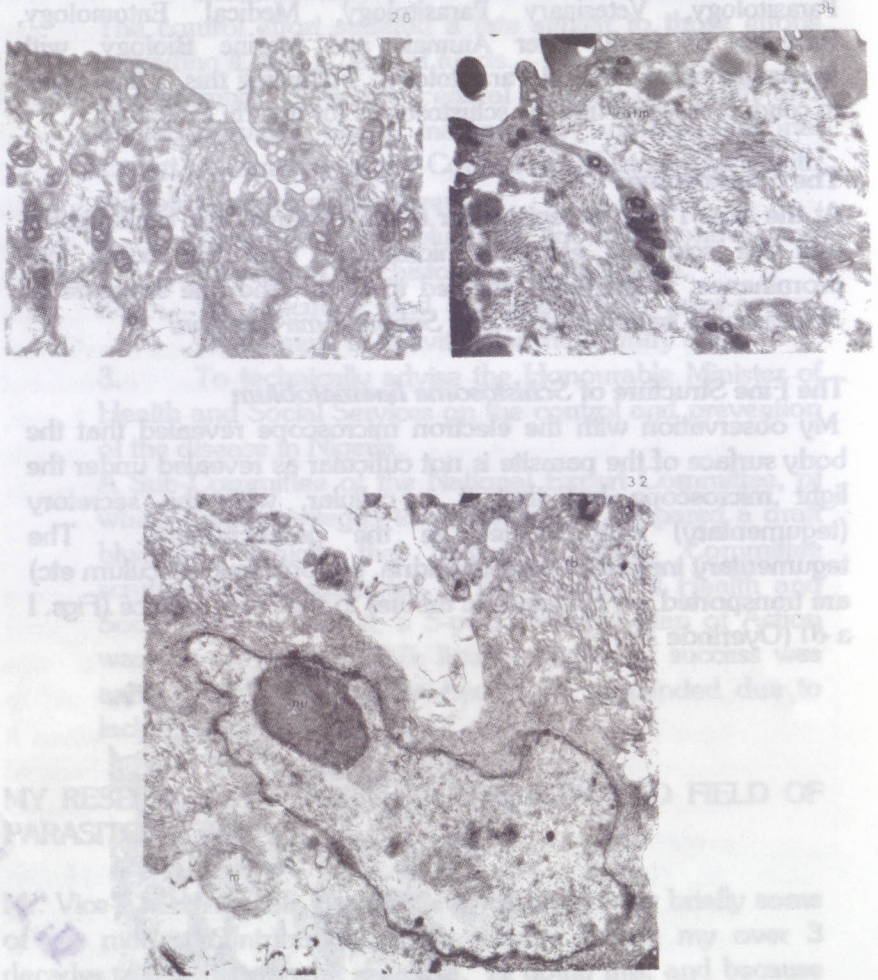
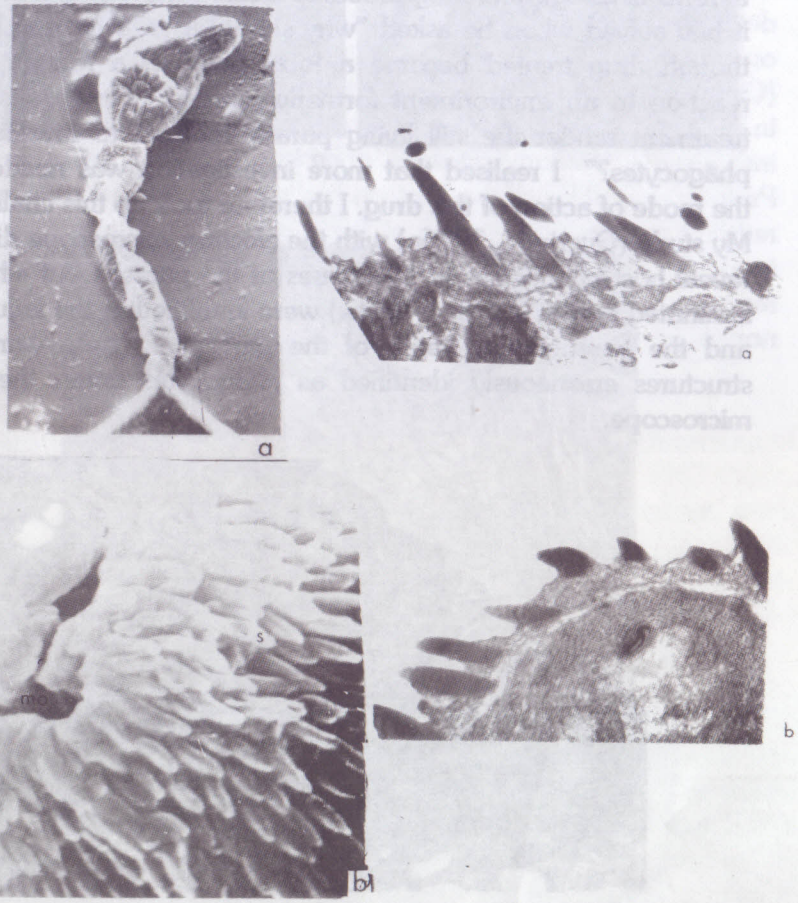
Fig1b-d



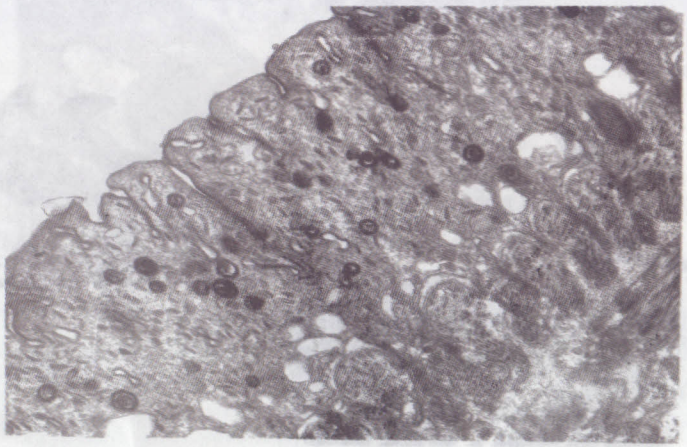
Cercarial Penetration Mechanism

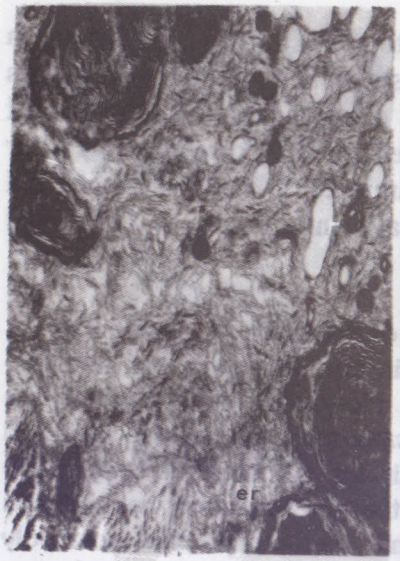
The mode of infection with schistosome is by penetration of the cercariae (larvae) through the skin. We know that cercariae have oral and ventral suckers, penetration glands and stylet which play different roles in the penetration process. My observation with the scanning and transmission electron microscopes (Oyerinde, 1970) revealed that the spines covering the body of cercariae are backwardly recurved (Fig. 2a-d) These spines provide efficient anchorage for the penetrating cercariae. This observation thus provides additional morphological adaptation mechanism for cercarial penetration into the host.



Drug Action on *Schistosoma mansoni*

Perhaps the most effective schistosomicidal drug that has ever been synthesized is the diphenoxyalkanes, the heptane, octane and nonane salts which showed marked activity only in schistosomal infection of mice. Standan (1962) investigated the mode of action of 1.7 bis (p-aminophenoxy) heptane, a diphenoxyalkane, and found that within 6 hours of administering the drug, the worms were swept into the liver, stimulated a foreign body reaction and were ultimately destroyed by host phagocytes which entered their bodies. Standen himself realised that his histological finding probably created more problems than it had solved when he asked "why should a parasite, still living, though drug treated become a focal point of a foreign body reaction in an environment formerly benign? How does drug treatment render the still living parasite subject to invasion by phagocytes?" I realised that more investigation was needed on the mode of action of this drug. I therefore took up this challenge. My study (Oyerinde, 1976a) with the electron microscope did not reveal host phagocytes in the tissues of the parasite but whorled laminated bodies (Fig 3a-c below) were identified in the tegument and the parenchymal tissue of the parasite. These were the structures erroneously identified as phagocytes under the light microscope.





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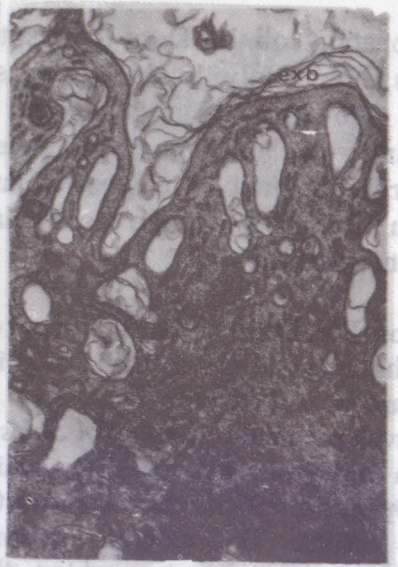

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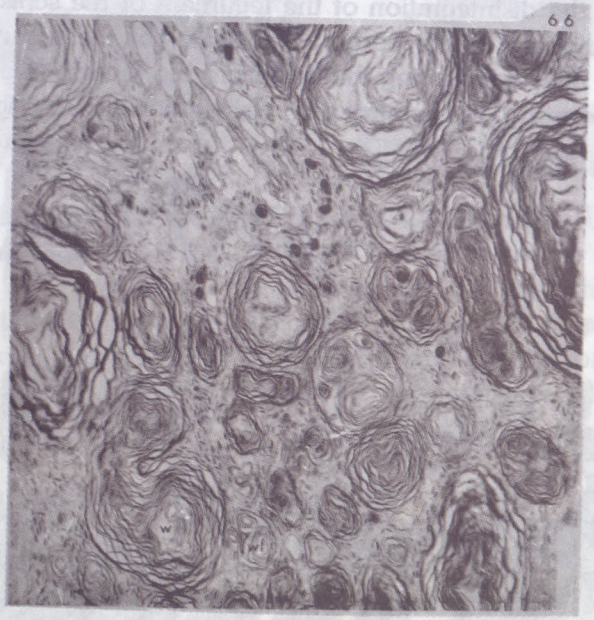
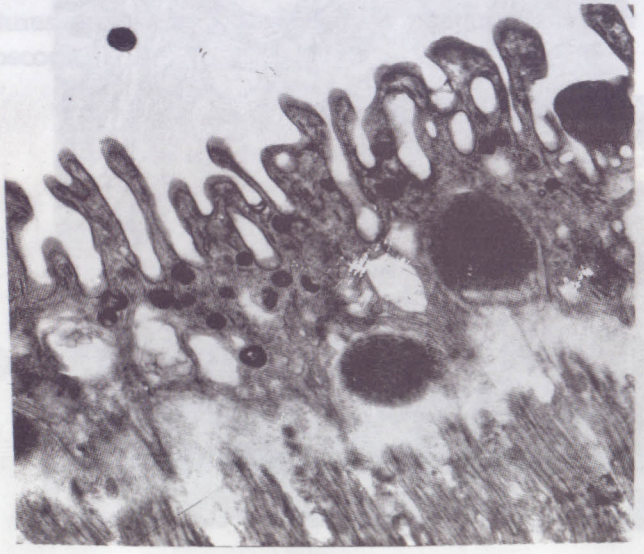
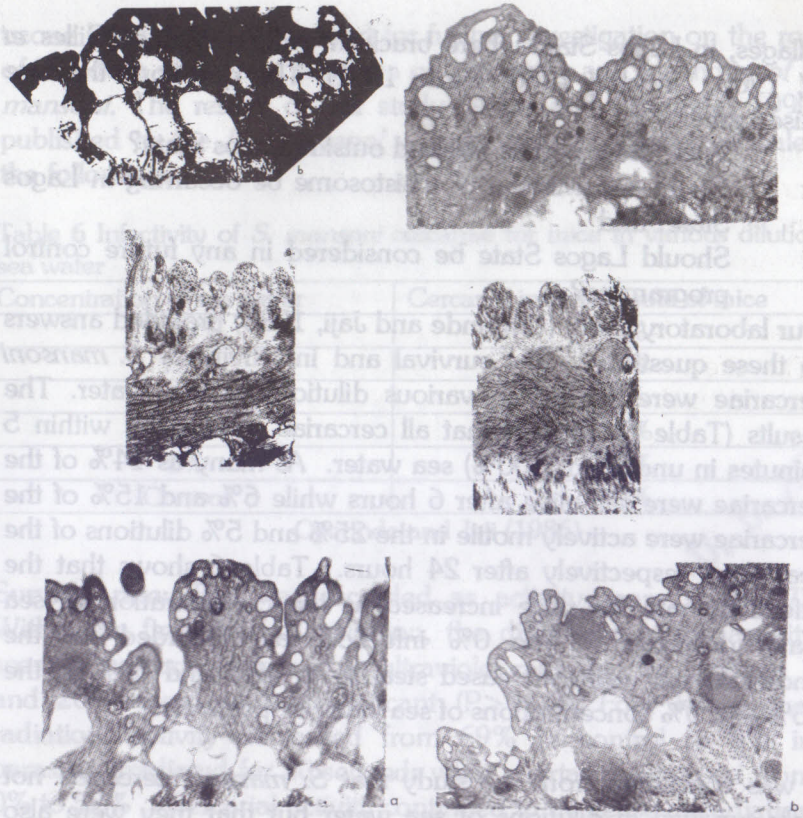


Donatio Cioli, M. D. of the Institute of Cell Biology, Rome, in his letter of July, 13, 1984 to me stated among others as follows "I enjoyed reading your paper on diphenoxyheptane, and I think you should be congratulated for the correctness of your early observation on "whorled laminated structures" which if I am not mistaken, are now-a-days interpreted as lysosomal abnormalities."

Host-Diet Effect on the Tegument of *Schistosoma Mansoni*

The effect of protein-deficiency in human diet is well known - stunted and retarded growth. However, knowledge of the developmental processes of *Schistosoma mansoni* in a host, fed on protein-free diet had to wait for the result of my study (Oyerinde, 1975) which showed that the tegument was reduced in height. The external plasma membrane was markedly invaginated, almost reaching the basal plasma membrane. Prolonged feeding of the host on the protein-free diet ultimately resulted in the disintegration of the tegument of the schistosome in localised areas of the body. The ability of the tegument to regenerate after transferring the host from the protein-free diet to normal laboratory diet (control diet) was demonstrated (Fig. 4a-f).





I vividly recall that the late Professor. D. Lee of the University of Cambridge who was the external examiner for my PhD thesis, asked me at the end of the examination if I knew that my work was the first to demonstrate regeneration of parasitic platyhelminths (flat worms), a common feature among the free-living platyhelminths; I could not show my happiness but I did not fail to thank him for the very useful information.

Transmission of *Schistosoma Mansoni*

Experimental Transmission in Brackish Water: *Schistosoma* species are associated with fresh water in which transmission takes place. However, there are reports in the literature of *S. mansoni* occurring in the inhabitants of Epe, Ajara and adjoining

314445

villages, in Lagos State where brackish waters abound (Gilles *et al,* 1965, Nnochiri 1966, Ejezie 1981). The questions that are raised are:

1. Were these people infected outside Lagos State?
2. Could transmission of schistosome be occurring in Lagos State? and
3. Should Lagos State be considered in any future control programme?

Our laboratory study (Oyerinde and Jaji, 1986) provided answers to these questions. The survival and infectivity of *S. mansoni* cercariae were studied in various dilutions of sea water. The results (Table 5) showed that all cercariae were dead within 5 minutes in undiluted (100%) sea water. As many as 14% of the cercariae were still alive after 6 hours while 6% and 15% of the cercariae were actively motile in the 25% and 5% dilutions of the sea water respectively after 24 hours. Table 6 shows that the infectivity rate for mice increased as the concentration of sea water decreased. The 0% infection rate recorded for the undiluted sea water increased steadily to 18% and 52% in the 25% and 5% concentrations of sea water respectively.

It was concluded from the study that *S. mansoni* cercariae not only survived in dilutions of sea water but that they were also infective for mice. The greater the dilution of the sea water the higher the infection rate. Rivers flowing from the hinterland discharge their water into the lagoons. The salt content of the estuaries are therefore subjected to variations from one site to the other and from one season to the other, thus implying that foeus of infection can be set up in brackish waters.

Effect of Ultraviolet Radiation on Transmission: Most studies on the effect of ultraviolet (UV) radiation on schistosomes focused on the development of immunity (Cox 1978, Dean *et al.* 1983, Moloney *et al.* 1985, 1987). Only few reports are available on the influence of ultraviolet radiation on parasite transmission and morbidity (Coggle, 1971). Reports (Imos, 1975) on the increase of ultraviolet radiation due to decrease in the earth's protective

ozone layer dictated the need for further investigation on the role of low dose ultraviolet radiation on infectivity and fecundity *of S. mansoni.* The results of our study (Ariyo and Oyerinde, 1990) published in the *International Journal for Parasitology* revealed the following:

Table 6 Infectivity of *S. mansoni* cercariae for mice in various dilutions of sea water

Concentration of sea water	Cercarial infectivity rate of mice
100%	0%
75%	0%
50%	2%
25%	18%
5%	52%
Control	49%

Oyerinde and Jaji (1986)

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Survival rate: This was recorded as activity score (Table 7). Within the first hour of radiation, the difference in the activity score of cercariae exposed to ultraviolet radiation for between 1 and 20 seconds was insignificant ($P>0.05$). Four hours post radiation, activity decreased from 69% in control to 9% in parasites irradiated for 20 seconds while mortality increased from 0% to 52% . Cercarial activity continued to decrease as radiation levels as well as age of cercariae post irradiation increased. The cercariae eventually became immobile before they died.

Worm Recovery (Fig. 5a): Maximum worm recovery of adult worms was recorded at 6 weeks post infection regardless of radiation dose. The percentage worm recovery of adult worms decreased significantly from 49% in the control to 37.5, 38.5, and 21% as the radiation exposure dose increased from 1 sec. to 3, 5 and 10 sec. respectively. (1 S.D =D = 4.28 for $P = 0.05$ and 15 degree of freedom).

Worm Fecundity: Table 8 shows the inhibitory effect of UV radiation on the fecundity of *S. mansoni.* There was a significant ($P< 0.001$) decrease in the egg load as well as in the viability of eggs (Fig. 5b) produced by irradiated worms as compared with

TABLE 7—ACTIVITY SCORE (%) OF IRRADIATED *Schistosoma mansoni* CERCARIAE

Time post-irradiation (h)	No radiation (control)				Duration of exposure to radiation (s) 1				3				5				10				20			
	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§
0.00	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0
0.25	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0
0.50	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0
1.00	100	0	0	0	96	4	0	0	100	0	0	0	97	0	0	3	100	0	0	0	93	0	5	2
4.00	69	0	31	0	7	59	7	27	17	28	22	33	14	33	20	33	3	79	3	15	9	5	34	52
8.00	14	33	25	27	0	12	15	73	0	20	11	69	7	3	0	90	0	0	0	100	0	2	5	93
11.00	0	11	11	78	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	4	96
20.00	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100	0	0	0	100

Key: * Actively motile; † sluggishly motile; ‡ immobile but alive; § dead.

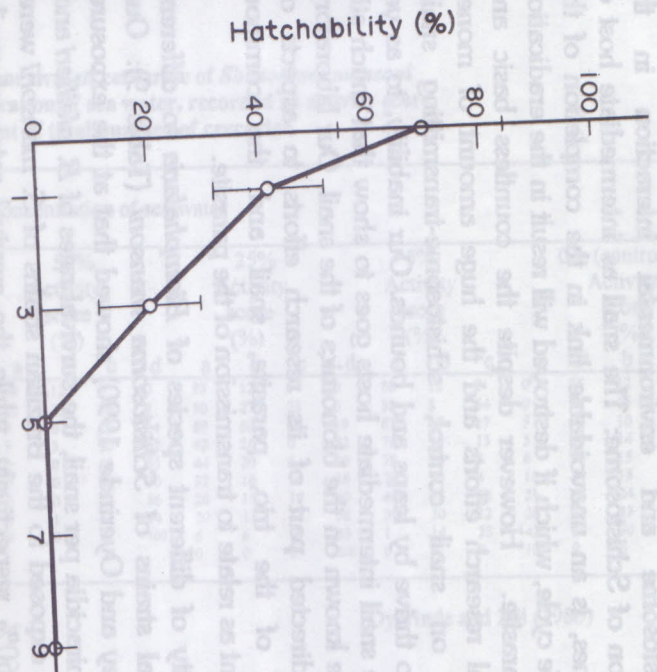

FIG. 12. Viability of eggs in the liver of mice 10 weeks post-infection with irradiated cercariae in relation to radiation exposure.

control worms. It was concluded that UV radiation has an inhibitory effect on the activity and fecundity of the parasite and thus reflect the possibility of a reduction in the number of parasites that will reach maturity and is hence a limiting factor in the epidemiology or transmission of the parasite.

Snail-Schistosome and environmental interaction in the transmission of Schistosome: The snail as intermediate host of schistosomes, is an unavoidable link in the completion of the parasite life cycle, which if destroyed will result in the eradication of the parasite. However despite the countless basic and operational research efforts and the huge amount of money expended on snail control, schistosome-transmitting snails continue to thrive by leaps and bounds. Our inability, to as yet control the snail intermediate hosts goes to show that much still needs to be known on the bionomics of the snail. Our laboratory therefore directed part of its research efforts to aspects of interaction of the trio, parasite, snail and the common environment as relate to transmission of the parasite.

Susceptibility of different species of *Biomphalaria* to different geographical strains of *Schistosoma mansoni* (Table 9): Our studies (Wey and Oyerinde 1990) showed that at the exposure level of 5 miracidia per snail, the survival rates of *B. pfeifferi* and *B. glabrata* exposed to the Brazilian strains of *S. mansoni* were 40% and 60% respectively, while the survival rates of snails exposed to the Kenyan strains were 50% and 80% respectively. The infection rates and mean cercarial production per snail per day for *B. pfeifferi* exposed to the Kenyan strain of *S. mansoni* were 20% and 60 cercariae per snail per day while *B. glabrata* had an infection rate of 65% when exposed to the Brazilian strain of *S. mansoni* and 60% when exposed to the Kenyan strain of *S. mansoni*. Its mean cercarial output per snail per day were 1178 and 1160 respectively. It was concluded that *Biomphalaria* species from a particular geographical area exposed to *Schistosoma mansoni* miracidia from different geographical regions not only survived the infection but also supported the

growth of the parasite to the stage infective to man. The implication of these findings is that importation of schistosome

Table 5 Showing survival of cercariae of *Shistosoma mansoni* in different concentration of sea water, recorded as activity score percent of total number of cercariae.

Time after Exposure	Concentration of sea water																							
	100% Activity score (%)				75% Activity score (%)				50% Activity score (%)				25% Activity score (%)				5% Activity score (%)				0& (control) Activity score (%)			
	a	b	c	d	a	b	c	d	a	b	c	d	a	b	c	d	a	b	c	d	a	b	c	d
0 minute	88	12	0	0	88	12	0	0	88	12	0	0	88	12	0	0	88	12	0	0	88	12	0	0
5 minutes	0	0	0	100	0	0	35	65	22	15	55	3	50	27	23	0	58	8	34	0	85	15	0	0
15 minutes					0	0	0	100	5	6	25	64	69	60	60	19	61	17	17	5	90	10	0	0
30 minutes									5	3	30	62	42	26	0	32	70	13	13	5	86	14	0	0
60 minutes									0	0	45	55	44	20	6	30	78	12	4	6	85	15	0	0
90 minutes									0	0	30	70	32	16	36	16	70	10	4	16	80	13	0	0
6 hours									0	0	14	86	26	15	19	40	49	16	14	21	68	24	2	6
21 hours									0	1	5	94	20	11	11	58	30	30	12	28	40	33	8	20
24 hours									0	0	0	100	6	6	2	85	1	14	33	42	30	30	12	28
30 hours													0	0	0	100	0	0	0	100	9	6	9	75

* Activity Score
A actively motile cercariae
B sluggishly motile cercariae
C immobile but alive
D dead cercariae

Oyerinde and Jaji (1986)

Tab 9

Table 9 Survival rate, infection rate and mean cercarial production of *Biomphalaria pfeifferi*, *B. glabrata* separately exposed to the Brazilian and Kenyan strains of *S. mansoni*

S. mansoni strain	*B. pfeifferi*			*B. glabrata*		
	Survival rate	Infection rate	Mean cercarial production per snail per day	Survival rate	Infection rate	Mean cercarial production per snail per day
Brazilian	40%	0%	0	60%	65%	1178
Kenyan	50%	20%	60	80%	60%	1160

Wev and Oyerinde (1990)

transmitting snails from one geographical zone to any other schistosome endemic zone may worsen an already bad situation and should be banned.

Effect of aestivation of *Biomphalaria pfeifferi* on the snail and its schistosome parasite: The aquatic habitat of the snail intermediate hosts periodically dries up during which period snails of all ages whether infected or uninfected aestivate. We (Badger and Oyerinde 1998) studied the effect of drought both on aestivating snails and on their intra-molluscan parasites and found that;
infected snails are poor aestivators as compared with uninfected snails, and the older the snails, the longer the period they survived desiccation (Badger and Oyerinde In Press). We (Badger and Oyerinde 1996) also found that:

(c) when infected snails were aestivated for various periods of time and were re-activated, the proportion of snails that were alive at weekly intervals was related to the length of time the snails had spent between infection and aestivation, varying from 100% survival at 0 hour to 5% survival at 21 days.

(d) the mean number of cercariae produced by the snails after re-activation also varied, with period between infection and aestivation, being highest in those snails that aestivated immediately post-infection.

(e) Cercarial development was delayed by prolonged desiccation of infected snails.

The implication of the results of this study is that the application or intensification of anti-snail measures in the control of

schistosomiasis will be more cost effective when applied as soon as the rains arrive and I SO RECOMMEND.

***Schistosoma mansoni* – *Salmonella typhi* Interaction**

In most tropical countries like Nigeria polyparasitism (infection with many parasites) is the rule rather than the exception. Oyerinde *et al* (1981) recorded 9 different species of intestinal parasites in a 16 year old girl. Where more than one parasite species inhabit the same microhabitat, there will be interaction among the parasites, which may modify the behaviour of the parasites as well as that of the host. Njunda and Oyerinde (1996) investigated the consequences of such interactions between *Schistosoma mansoni* and *Salmonella typhi* in experimental infection of mice and found that survival rate of infected mice was related to age of schistosomal infection prior to *Salmonella typhi* infection. Further studies (Njunda and Oyerinde 1997) showed that *Salmonella typhi* was ingested by the parasite (*Schistosoma mansoni*) in which, the *Salmonella* (bacteria) continued to proliferate even after successful treatment of the mice with chloramphenicol. The proliferating bacteria within the parasite were regurgitated periodically into the mice, resulting in relapse of the *Salmonella* infection. The implication of this study is that in schistosome endemic areas like Nigeria, confirmed typhoid patients should be screened for schistosome and if positive should be treated for both infections simultaneously.

The Hookworms

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Hookworm situation in Lagos

In the sixties and early seventies, the favoured drug for the treatment of hookworm was alcopar (bephenium hydroxy naphthoate). However, at the Lagos University Teaching Hospital, we were recovering hookworm eggs in faeces of patients after treatment with the drug. I therefore carried out laboratory studies to elucidate the ineffectiveness of alcopar in the treatment of hookworm in the Lagos Metropolis. My studies (Oyerinde 1978) confirmed that two species of hookworm, *Necator*

americanus and *Ancylostoma duodenale*, occur in the Lagos Metropolis and that both species often occur in the same individual. The study (Table 10) further showed for the first time that

Table 10 Estimated ratio of adult female *A. duodenale* to adult *A. americanus* in metropolitan Lagos

% of total positive specimen	No. of female *A. duodenale*	No. of female *N. americanus*
59	0	100
12	3	95
11	8	85
6	13	75
8	18	65
3	23	55
1	28	45

Oyerinde (1978)

(a) all eggs recovered after treatment with the drug (alcopar) were those of *N. americanus*.

(b) Fifty-nine percent of hookworm infection were due to *N. americanus* only.

(c) In 12% of hookworm infections, there were 3 female *A. deodenale* to 95 female *N. americanus*.

(e) The highest number of *A. duodenale* in mixed infections was 28 female *A. duodenale* with 45 female *N. americanus* and occurred in only 1% of the infections.

The study thus showed that *N. americanus*, the predominant hookworm, occurring in metropolitan Lagos is not susceptible to alcopar.

Transmission of Hookworm

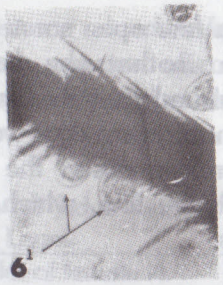
In a survey of human intestinal parasites in Lagos Metropolis, Oyerinde *et al.* (1981) recorded a case of hookworm infection in a 10-month old baby girl who had not started walking. Her mother claimed that she had never been left to play outside the

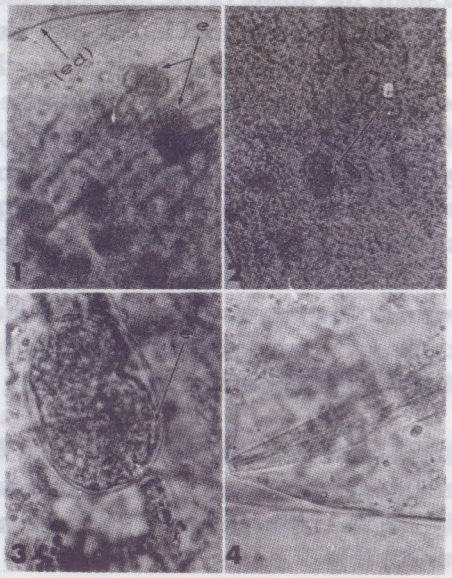
house. The baby had a few open sores and boils on her arms, and according to the mother she had had frequent boils previously. It was presumed that this was not a case of intra-uterine infection as no hookworm eggs were found in the stool of the mother. This therefore raised the possibility of infection transmitted mechanically by flies, which had been attracted to feed on the baby's sores.

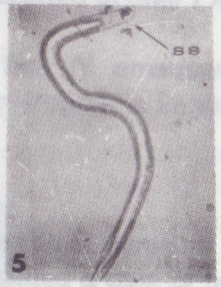
Mechanical Transmission by Flies: Oyerinde (1976b) carried out experiments to study the ability of the housefly (*Musca domestica*) to disseminate hookworm eggs and larvae and also to determine the viability of the eggs and the survival of the larvae after different periods of exposure on the body surface and in the gut lumen of the fly. The results (Oyerinde 1976b) published in the *Annals of Tropical Medicine and Parasitology* confirmed earlier observation that the house-fly can disseminate eggs (Fig. 6) and larvae of parasites and went further to show for the first time that,

(1) hookworm eggs that pass through the alimentary tract of *M. domestica* remained viable and hatched after incubation for 48 hours (Fig. 7.1 - 7.3);

(2) infective larval stage of hookworm (Fig. 7.4) pass through the alimentary tract of *M. domestica* and is recovered from its faeces without losing its morphology (normal appearance) (Fig. 7.5.)







The Role of Humans in the Transmission of Hookworm: The determining factor for the transmission of hookworm is the degree of contamination with eggs to which the environment is subjected.

However the degree of contamination of the environment with parasite egg does not always correlate with the infection rate. Nevertheless my study (Oyerinde 1982b) showed that in the Lagos Metropolis:

(1) The prevalence of hookworm infection was 26.6% of either the male or the female population.

(2) The 16-25 year age-group had

(a) the highest infection rate of 48.7%;

(b) the highest intensity of infection (44 adult female worms per infected individual) and

(c) the greatest transmission index of 3.0365 or 26.90% of the total; although the mean daily egg output did not always correlate with the transmission index.

It was concluded that the 16-25 year-age group

(a) should be given special consideration in hookworm control programmes;

(b) that hookworm should first be suspected as the possible aetiology of any anaemia disease in the age-group;

(c) that any person within the age-group who is likely to lose blood other than to hookworm e.g. through operation and child birth should be treated for hookworm at an appropriate time.

CONCLUSIONS

Transmission of parasites from an infected individual (host) to another susceptible individual can be illustrated thus (Fig. 8a-c).

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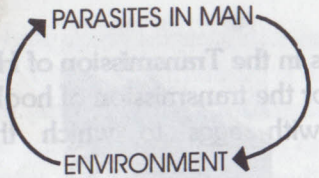

FIG. 8a: DIRECT TRANSMIISSION OF PARASITES

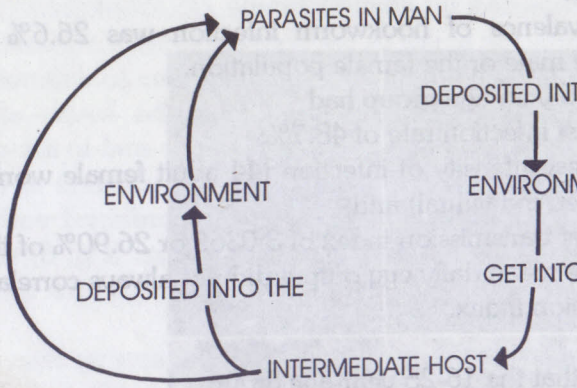

FIG. 8b: PARASITE TRANSMISSION INVOLVING INTERMEDIATE HOST

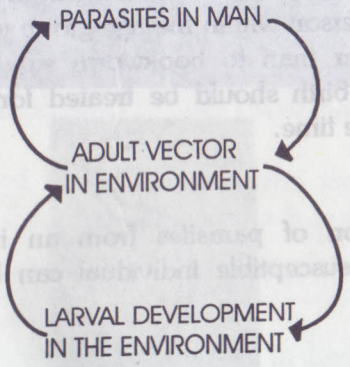

FIG. 8c: TRANSMISSION INVOLVING VECTORS

The emphasis in this figure, is to highlight the prominent role our environment plays in the transmission and hence the perpetuation of parasites. The environment therefore, if appropriately taken care of can adequately reduce the menace of parasites. Unfortunately, human efforts at controlling or eradicating these parasites have only yielded insignificant results Instead, new and more deadly parasites are evolving. The emergence of the AIDS virus is a case in point. This is probably what Professor S. A. Fashuyi of the Federal University of Technology, Akure, had in mind when he titled his inaugural lecture "Parasites for ever" (Fashuyi 1994). None-the-less, I am confident that if the under-listed recommendations could be implemented, "The War Against Parasites" would be won.

RECOMMENDATIONS

The conclusions drawn above implicate the environment as the single most important factor in the perpetuation of parasites. The environment is contaminated with cysts, eggs and larvae of parasites where development to the stage infective to man or the intermediate host takes place, but only under conducive environmental conditions. In like manner, favourable environmental conditions are a *sine qua non* for insect vectors of parasites not only to thrive but also to be capable of transmitting parasites

The basic approach to interrupt transmission include:

(a) Hygienic disposal of human wastes – faeces, urine, etc.
(b) Provision of potable drinking water.
(c) Maintenance of good personal and food hygiene; and
(d) Improved environmental hygiene.

The rationale for this strategy has been the subject of extensive discussion at many local and international scientific meetings; from which, recommendations had also been made and from which I have taken a cue in making the underlisted recommendations.

Health education

Health education aims at increasing the awareness of the relationship between infection and personal hygiene including basic information on mode of human infection and preventive measures.

It is recommended, as a first step, that massive health education be mounted at both primary and secondary school levels as well as community level on the advantages of cultivating habits such as hand washing before and after every meal and wearing of shoes all the times etc.

Environmental sanitation

Local Government Authorities (LGAs) should provide

(a) adequate and efficient means of faeces disposal (water closet or pit laterines) in all public places. The use of bucket system encourages dissemination of parasites by flies, cockroaches and other insects and hence must be discouraged.

(b) LGAs should provide clean and potable drinking water (stand pipes where ever pipe borne water is available, bore holes or dug up wells) in all public places like markets, motor parks as well as strategic places within the township.

(c) LGAs should ensure provision of these facilities in building plans before approval is given for construction of new buildings.

(d) LGAs should ensure prompt and hygienic removal of waste generated in the communities within their authority as well as waste generated during the environmental sanitation exercises. Unattended generated waste is a sure source of epidemic outbreak of parasitic, bacterial and viral diseases.

(e) It must be emphasised that LGAs should, through persuasion band motivation, encourage maintenance of environmental cleanliness. The gutters and drains must be cleaned up regularly to avoid stagnation of water. All

potholes and ditches must be filled, broken bottles, empty cisterns and other containers that can retain water around the homes must be discarded or buried. Overgrown weeds must be cut and discarded or burnt. All domestic water receptacles must be tightly covered to prevent access to mosquitoes, other insects, lizards, rats etc.

The community must be involved in this exercise to instil in them a sense of belonging. In my opinion the current monthly environmental sanitation exercise, bi-monthly in some states, is a right step in the right direction. Nevertheless, its scope should be expanded to include all the above. The aquatic larval development of mosquitoes takes 4 to 5 weeks to complete, depending on the environmental weather conditions, I recommend a bi-monthly environmental sanitation exercise all over the country. This done, larval mosquitoes that escape the onslaught of one environmental sanitation exercise may not be lucky at the following clean-up exercise.

The populace should be motivated, through prize awards for environmental cleanliness at community, Local Government, State and Federal Government levels.

In order to achieve the desired result promptly and dramatically, the above measures must be supplemented with chemotherapeutic treatment of clinically infected (symptomatic) persons.

Advantages of the Recommendation

The benefits that are accruable from the manipulation of the environment in the control of parasitic infections cannot be quantified. The approach;

(a) eliminates the fear of drug or insecticide resistance in the parasite or in the insect vector and also eliminates drug toxicity in humans;

(b) preserves our scarce foreign exchange earnings;

(c) is cheap;

(d) does not involve sophisticated equipment that need special training to operate and above all;

(e) gives members of the participating communities a sense of belonging.

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First and foremost, I thank my God and Creator who has guided me from cradle through a tortuous life's journey to witness this day. O, wonderful Lord, I glorify thy name.

I remember today as I do everyday my late parents and I thank God for the lives they lived, particularly for instilling in them the value of education. May their great souls rest in perfect peace. They unhesitatingly provided for my education within the limits of their resources. My mother died while I just finished my primary education, and left behind 2 boys and 2 girls. The care of my 2 sisters and brother, the youngest of us and now of blessed memory, May his soul rest in perfect peace, then rested squarely on my father, my 3 step-mothers, who are still alive today and on so many auntie's. I am eternally grateful and indebted to them for the motherly care and love they all extended to us.

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the few people who were around me could not be convinced how serious the illness was. It was through her efforts and care that I survived. For this singular act I thank her most sincerely. May the good Lord repay her abundantly.

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Mr. Vice-Chancellor Sir, Deputy Vice-Chancellors, Very Distinguished Ladies and Gentlemen, I thank you all most sincerely for your attention.

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Fig. 1a. Longitudianal section through posterior dorsal surface of a male *Schistosoma haematobium.* A cytoplasmic connection could be traced from tegument to the tegument-forming-cells.

Fig. 1b. Section through dorsal tegument containing mitochondria, endoplasmic reticulum, round and rod bodies.

Fig 1c. An oblique section through a male tegument showing cytoplasmic tubules between the tegument and the tegument-forming-cells. A few tegumental structures can be seen in the cytoplasmic tubules.

Fig 1d. A tegument-forming-cell with prominent nucleus, nucleolus, mitochondvia, round and rod bodies.

Fig 2a. Stereoscan photomicrograph of the ventral surface of *S. mansoni* cercaria.

Fig. 2b. Higher magnification of the head to show the back-wardly directed spines(s) which surround the mouth.

Fig. 2c. Section through the oral cavity of *S. mansoni* cercaria showing details of the arrangement of the spines.

Fig. 2d. Section through the head of *S. mansoni* cercaria showing arrangement of the spines.

Fig. 3a. Section through untreated 42 day-old *S. mansoni* i.e. control experiment.

Fig. 3b. Section through mid-dorsal region of a male *S. mansoni* 6 hours after administration of the drug *Note.* Appearance of laminated whorls and extensive formation of endoplasmic reticulum(er).

Fig 3c. Section of a male *S. mansoni,* 16 hours after drugging showing different sizes of the laminated whorls indicating continuous action of the drug.

Fig. 4a. Section through ventral tegument of 42 day-old *S mansoni* recovered from mice fed on protein-free diet. Note the greatly reduced size of the tegument and the extensive invagination of the external plasma membrame as compared with the control (Fig. 3a)

Fig. 4b. Section of a 59 day-old worm from mice fed on protein-free diet. The evaginations of the basal plasma membrane has greatly increased in size.

Fig. 4c. Section of a 64 day-old worm. By this time the membranes between adjacent evaginations start breaking down. (arrows)

Fig. 4d. Section of a 64 day-old worm. In this region, the tegument is completely discarded. (shed)

Fig. 4e. Section through the dorsal tegument of *S. mansoni* recovered from mice fed on protein-free diet for 50 days and then transferred to normal laboratory diet for 10 days, Note: The structures of the tegument tend towards normal with the basal evaginations restricted basally

Fig. 4f. Section through the dorsal tegument, 27 days after transfer to normal laboratory diet. By this time the tegument of the worm has compeletely changed to normal structure i.e. regenerated

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