PARASITIC INFECTIONS AND STORAGE IRON DEFICIENCY IN CHILDREN IN IMPOVERISHED REGIONS OF MAURITIUS

A thesis submitted by

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Using a combination of techniques including relative deprivation index developed by the Government, school performance, household survey and drawing of lots, a sample frame constituting of poverty areas of Mauritius was constructed and four test areas identified.

Relevant haematological, parasitological and biochemical parameters of all school-going children living in the four test areas were determined so as to study the possibility of correlation between parasitic infections, plasma ferritin, haemoglobin concentration, white blood cells count, packed cells volume and blood group.

It was found that there is a negative correlation between the number of parasites and haemoglobin concentration, packed cells volume of blood and degree of infestation, number of parasites and ferritin and number of parasites and age of subject.

It has also been found that, children with blood group 'A' and blood group 'O' tend to harbour the most parasites.

As regards to storage iron depletion, this is significant only with hookworm infestation. Additionally it has been noted that hookworm infestation is directly related to age contrary to other parasitic infestations.

Key words: Children, Poverty, Parasites, Iron deficiency, Blood group.

TO MY PARENTS,

WIFE TIRWANTEE, SON SANDEEP

AND DAUGHTER ROSHNI

For their support and encouragement

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ABBREVIATIONS

ASC, Asc, Ascaris	*Ascaris lumbricoides*
CSO	Central Statistical Office
E.coli	*Entamoeba coli*
E.hist	*Entamoeba histolytica*
GL, Giardia	*Giardia lamblia* (now called *Giardia duodenalis)*
Hb, HB	Haemoglobin
Hk	Hookworm
LSMS	Living Standards Measurement Survey
Ml	Millilitre
MOH	Ministry of Health
NPS	No parasite seen
P.amoeba	Precystic amoeba
PCV	Packed cells Volume of blood
Rh	Rhesus factor
rpm	Revolution per minute
Rs.	Mauritian rupees (Rs. 42 = 1 £)
tri, TRI, trichuris	*Trichiuri trichiura*
Troph	Trophozoites
µl, µL	Microlitre
WBC, Wbc	White blood cells
Wrt	With respect to

CHAPTER 1

INTRODUCTION

Children and Iron deficiency

As Carol Bellamy, the Executive Director of United Nation Children's Fund rightly puts it, the body needs iron to produce haemoglobin, the protein in red blood cells responsible for carrying oxygen. Iron is also a component of the many enzymes essential for the adequate functioning of brain, muscle, and the immune system cells (Bellamy, C., 1998).

A certain amount of iron is stored in the liver, spleen and bone marrow. Iron deficiency develops when these stores are depleted and there is insufficient iron absorption due to insufficient intake or malabsorption or both. This is a typical situation of malnutrition.

Malnutrition, as it is widely accepted nowadays, is a result of a combination of inadequate dietary intake and infection. The interplay between the two most significant immediate causes of malnutrition - inadequate dietary intake and illness- tends to create a vicious circle: A malnourished child, whose resistance to illness is compromised, falls ill thus worsening malnourishment. Children who enter this malnutrition-infection cycle can quickly fall into a potential fatal spiral as one condition feeds off the other.

Malnutrition lowers the body's ability to resist infection by undermining the functioning of the main immune-response mechanisms. Infection on the other hand, causes loss of appetite, malabsorption, and metabolic changes.

These days people are very concerned about damage of the immune systems of some 23 million individuals world wide by HIV (Piot, P., 1997). However it is also quite alarming to note that malnutrition impairs the immune systems of as many as 100 million young children in the world.

Malnourishment is responsible for shifting the balance from infection towards disease, as a malnourished person is much less able to withstand the adverse effects of disease. In the developing world, where malnutrition is common, a shifting in the balance from infection to disease is a particular problem (Muller, R. Et al, 1990). One should be more concerned about this when we know that nutrition is severely affected by parasitic infection in several ways including ingestion of blood by the parasite, which leads to loss of iron and other nutrients. Parasites also cause the lining of the intestine to change. This reduces the surface membrane available for digestion and absorption. As a result, fat, certain carbohydrates, proteins

and several vitamins are not absorbed properly, (Bellamy, C., 1998), with major reduction of growth rate, (Mahendra Raj, S., 1997). Also, as a result of parasitism, the host gives priority to the reversal of the pathophysiological consequences of parasitism over other body functions, including the expression of immunity (Coop, R. L., Kyriazakis, I., 1999).

Iron deficiency anaemia is probably the most prevalent nutritional problem in the world. It impairs immunity and reduces the physical and mental capacities of the populations. It shows problems with coordination and balance. These factors also hinder a child's ability to interact with and learn from the environment (Draper, A., 1997).

Infection with parasites has been shown to aggravate diarrhoeal disease or malnutrition, causing morbidity and mortality in children (Ighogboja, I.S.,1997). It is also suspected that parasitic infection causes anaemia and stunting in children (Stoltzfus, R.J., 1997).

Studies carried out in Tanzania demonstrated that while inadequate iron nutrition is responsible for iron deficiency anaemia in children and adults, anaemia due to parasitic infections could not be ignored (Tatala, S., 1997). In Alaska, on the other hand, it has been demonstrated that iron deficiency anaemia may be related to loss in faeces and not due to

inadequate or inappropriate iron intake (Petersen, K.M., 1996). It has also been shown that, parasitic infection (particularly hookworm) may contribute to the high prevalence of iron deficiency in the Aboriginal children population in Australia (Hopkins, R.M., 1997), in children in Zanzibar (Stoltzfus, R.J. 1997) and in children in Papua New Guinea (Pritchard, D. I., 1991).

Iron deficiency is a slowly evolving complication and it essentially passes through the following stages: progressive, pre-latent, latent, and manifest iron deficiency, (Cook, J. D., 1982). Storage iron depletion is in fact the earliest state, where loss of sequestered iron reserves occurs without a decrease in iron supply to the developing red cells. This condition can be monitored in the laboratory by the measurement of serum/plasma ferritin along with others like haemoglobin (Finch & Cook, 1984).

The prevalence of iron deficiency anaemia among infants remains the leading cause of anaemia, particularly among children in a relatively low-income population (Oski, 1993). On the other hand infectious diseases and parasitic afflictions are responsible for 71 percent of the deaths of children age one to four and 62 percent of the deaths of children ages five to fourteen (World Bank, 1994).

1. 1 Children in Developing countries.

When a child is ill, the illness interferes with the delicate balance between several physiological mechanisms which determine optimum growth. Similarly, nutrition being an important environmental influence, affecting the health and growth of children, lack of food and nutrition also interferes with optimum growth (Court, S 1971).

In developing countries, a low standard of living in a harsh physical environment is common in most rural areas. Housing is poor with inadequate ventilation. Environmental sanitation is virtually non-existent. Water is often brought from a distant source, and most of the time, is not sufficient to maintain a good standard of personal cleanliness. Drinking water is invariably unsafe. While the entire community is exposed to ill health, children succumb easily because they are more vulnerable. In this group, inadequate nutrition, physiologic demands, and lack of resistance make the effect of disease more serious as it should be understood, the entire child community is at risk normally, but only the fortunate survives (Bryant, J., 1969). Again, according to Kofi A. Annan, the Secretary – General of the United Nations, among the survivors, it is very likely that

growth potential gets hampered, and the body's physiological processes become sub-optimal, and the children grow up with lasting mental and physical disabilities (UNICEF, 1998)

Sickness and death amongst children in the developing world are usually not due to rare tropical disorders but mainly due to the 'disease of poverty' of which undernutrition and infection make a lethal combination (Bryant, J., 1969). Undernutrition causes a reduction in resistance to infection, so that the children are frequently ill. Every episode of illness causes further deterioration of the nutritional status and tends to take a severe form. The vicious cycle created culminates in serious illness (Court, S., 1971).

As stated above, anaemia is a common accompaniment of malnutrition. Generalised nutritional deprivation affects haemopoeisis. Haemopoeisis is severely affected when there is a remarkable depletion of the storage iron (iron deficiency anaemia). Iron deficiency hinders a child's ability to interact with and learn from the environment and may lead to lower intellectual abilities, yet, iron deficiency anaemia is probably the most prevalent nutritional problem in the world. (UNICEF, 1998)

Iron deficiency anaemia is believed to be brought about by two main mechanisms: dietary iron deficiency and parasitic infestation (Kemp, C.H.,

Silver, H.K., & O' Brien , D., 1970). Iron deficiency anaemia of infancy may therefore be corrected by the early introduction of the staple, vegetables and meat in the diet of infants.

Parasitic anaemia is believed to be widespread under insanitary conditions. Adult worm, in particular hookworm, lives in the small intestine attached into the mucosa. The attachment site of the worm is not fixed and changes every 4–6 hours to enable the worm to feed and mate (Kalkofen, U. P., 1974). In so doing the lining mucosa is bruised and left bleeding. Blood loss results from two mechanisms: Direct ingestion of blood by parasites (Roche & Larice 1066) and tissue trauma (Kalkofen, U. P., 1974). The amount of blood lost from the gut determines the development of anaemia. *Trichuris trichiura* on the other hand is found to be associated with undernutrition, growth stunting and possibly iron deficiency anaemia in intense infections (Cooper et al 1990).

The severity of this anaemia depends upon the type of worm, the worm load, the age of the child, the nutritional status of the child including his dietary iron intake. Also, children on adequate iron intake may keep pace with blood loss so that anaemia does not develop (Gill, Watson & Ball, 1964; Ebrahim, G. J., 1966; Pritchard, Quinell & al, 1991). In impoverished regions, adequate iron intake may not be practical, hence

the interest in investigating further, the extent of iron deficiency in relation to parasitic infection. There is also an interest in investigating the correlation, negative or positive, between parasitic infections and ABO blood group. It is believed that oligosaccharides and protein blood group antigens, may act as receptors for bacterial, viral and parasitic infectious agents (Mudad, R., & Telen, M. J., 1996). Besides, studies carried out in Germany have shown that people with blood group 'A' are predisposed to pseudomonal infection of the ear (Steuer, Hofstadter et al 1995). In the UK, it has been shown that blood group antigens can act as epithelial cell receptors for *Candida albicans* (Cameron, Douglas 1996). In France, studies of the combined effect of various histo-blood group genetic systems indicate some relation with breathing difficulties, possibly with reference to susceptibility to infectious agents (Kauffmann, Frette et al 1996). Opposed to the above, studies carried in India, have shown that people with blood group AB are less prone to malarial infection (Sing, Shukla, et al 1995).

1.2 Hypothesis/Research Question.

The central hypothesis investigated in this thesis is that:

- Malnutrition, which also includes inadequate dietary intake, is a consequence and cause of poverty.
- Parasites affect nutrition in several ways including ingestion of blood and other nutrients.
- Some parasites cause the lining of the intestine to change, which reduces the surface membrane available for digestion and absorption.
- Some parasites like Hookworm also cause chronic blood loss.
- During blood loss, iron is irretrievably lost from the body.
- Because of malnutrition and poverty, dietary sources of iron are insufficient to compensate for the loss, and this, coupled with malabsorption of iron, causes iron deficiency.
- Children in developing countries are the most severely affected. (Bellamy, C., 1998)

Some other hypotheses intended to be tested in this research, are :

- People with certain blood group antigens such as AB are less prone to infection, while those with other blood groups like blood group A are predisposed to infection.
- For parasitic infections too, some groups may be predisposed to the infection while others are less prone.
- The total white blood cells count tends to increase with parasites in the system.
- The Packed cell volume of blood is related to Haemoglobin and therefore also to plasma ferritin, and that with parasitic infections all the three haematological parameters tend to decrease.

This research therefore, is aimed at studying parasitic infections and storage iron deficiency in children in impoverished regions of Mauritius, parasitic infections and increase in white cells counts, parasitic infections and reduction in packed cell volume of blood, and also the predisposition of certain blood group type to parasitic infection.

1.3 **Objectives**

The main objectives of the present study were to determine:

- Pockets of poverty in the impoverished regions of Mauritius.
- The prevalence of parasitic infection in school-going children 4-10 years of age in the impoverished regions of Mauritius.
- The prevalence of iron deficiency.
- The relationship between parasitic infections and iron deficiency in these children.
- The degree of success in malaria eradication in Mauritius.
- The relationship between the ABO blood group antigens and the different parameters involved in the test.

1.4 **Parasitic diseases and the biology of the common Human Parasites encountered in Mauritius.**

Humans are hosts to a variety of organisms, not all of which cause disease. All associations in which one species lives in or on another are termed symbiosis, which literally means 'living together'. A parasite is an organism (plant or animal) which lives on or in another organism (the

host), takes nutrient directly from it and which under certain circumstances, may be harmful.

Some parasites are entirely dependent on the host for their reproduction and are therefore incapable of independent existence. These are termed obligate parasites. Whereas those organisms that are capable of existing outside the hosts are called facultative parasites.

A parasite growing in or on a host can cause an infection, but when the infection leads to a disease state, the parasite is referred to as a pathogen. Pathogenicity, therefore, is the ability of an organism to cause disease (Perera, S.A. et al, 1995).

Despite significant advances in the treatment and control of parasitic infections, they remain a major cause of morbidity and mortality in the modern world. The 1995 WHO estimates indicate that 1 400 million people world-wide are infected with *Ascaris lumbricoides, Trichuris trichiura*, or hookworms and that at least 200 million suffer from disease associated with these infections. The adverse effect on growth, nutrition and cognitive function are more evident in children, an age group which harbours the heaviest intensity of infections (WHO, 1995).

Three groups of parasites are recognised. These are protozoa, helminths and arthropods (Jewsbury, J.M., 1995).

Protozoa are single-celled organisms that are larger than bacteria. These are further subdivided into amoebae, which move by extending pseudopodia (e.g. *Entamoeba histolytica)*, flagellates, which move by beating of one or more flagella (e.g. *Giardia lamblia)*, ciliates which move by beating of cilia (e.g. *Balantidium coli*,) and the sporozoa which are all intracellular parasites (e.g. *plasmodium spp.).*

Helminths are the parasitic worms. They are large multicellular organisms and are divided into three groups: the tape worm (Cestoda) (e.g. *Taenia saginata, Taenia solium*), flukes (Trematoda) (e.g. *Schistosoma haematobium*), and the round worms (Nematoda) (e.g. *Ascaris lumbricoides, Trichuris trichiura, Necator spp., Enterobius vermicularis, Strongyloides stercolaris.*)

Arthropods that attack humans are mostly blood feeders (e.g. mosquitoes, ticks and fleas) and reside briefly on the host. Many of these arthropods are vectors for the transmission of other infectious agents (e.g. *Plasmodium spp.*). The scope of this research will be restricted to

protozoa and helminths only, as arthropods are not commonly encountered in Pathology laboratories in Mauritius.

Where and in which groups of people parasitic infection is more likely to occur is determined to a large extent by environmental conditions (housing, water supply and sanitation) and climate (particularly rainfall and temperature). It is also to be noted that individuals may be infected with more than one parasite at any one time.

In the year 1997, the Pathology Services of Mauritius examined a total of 9,097 stool specimens, 1,613 (17.7%) of which were found to contain some form of parasites. The principal parasites found were *Trichuris* ova in 489 specimens, *Ascaris* ova in 233 specimens and amoebae in 282 specimens. (1997 Health Statistics Annual, Report of the Principal Medical Statistician).

1.4.1 Blood parasites (Malaria)

Malaria is the leading communicable disease for a large segment of the population in many developing countries. It is one of the most serious public health problems in Africa as one million deaths from malaria occur

annually in African children less than 5 years of age (Maiga A. Sideye, 1993). Studies carried at Strasbourg, France, have shown that most of the travellers returning from African countries carried malaria with them, diagnosis was confirmed by presence of parasite on blood smears (Hansmann, Y. et al, 1997).In the transmission of malaria, three factors are involved, namely, the parasite, the vector and the host (Mc Gregor, J. D 1974). The parasites affecting man are of four different kinds, and are identified in the peripheral blood as a result of their specific morphologic characteristics, although, any one kind of the parasite may be genetically diverse (Farnet, A et al., 1997)

Plasmodium falciparum is widely prevalent in many tropical areas of the world and transmission is rare or unlikely in temperate regions.

Plasmodium vivax on the other hand, has a wider distribution and may be transmitted in temperate climates. It is absent from most of West Africa and it is believed that this is probably due to the absence of a certain blood group called Duffy blood group in the indigenous population of that region, showing the importance of blood group system in infection (Viqar Zaman 1995).

Plasmodium malariae, and *Plasmodium ovale* are much less common than *Plasmodium vivax* and *Plasmodium falciparum. Plasmodium ovale* is seen mainly in Africa.

Mortality and morbidity occurs in early life, mostly before the age of 5 years. Adults in the endemic areas develop immunity and remain asymptomatic.

Each species has two life cycles (see appendix), one in the host where the invasion of body organs and red blood cells take place, and the other is the vector where the sexual forms of the parasite combine to form a fertilised egg (the sporozoites).

Humans (the hosts) become infected when the sporozoites are introduced into the blood from the salivary secretion of the infected *Anopheles* mosquito, when the latter (the vector) takes a blood meal.

In general, with the exception of *Plasmodium falciparum* the sporozoites, after being inoculated by the infected *Anopheles,* remain in the blood for about half an hour. From the blood they enter the parenchyma cells of the liver where they divide to form the pre-erythrocytic schizont.

The merozoites discharged from the pre-erythrocytic schizonts enter the blood and parasitise red cells.

The entry into the red cells occurs by invagination of the host cell membrane thus forming a vacuole. Inside the vacuole, the merozoite is transformed into a trophozoite, which digests haemoglobin to form the malarial pigment called haemozoin. On maturation, trophozoites undergo schizogony to form daughter merozoites. After a few schizogonic cycles, merozoites develop into sexually differentiated cells, the male and female gametocytes.

Gametocytes continue their development in the mosquito vector. When the appropriate mosquito vector ingests the mature gametocytes, the sexual cycle is initiated within the mosquito with the eventual production of sporozoites that are infective to man (Hildelbrandt, J.P., 1996).

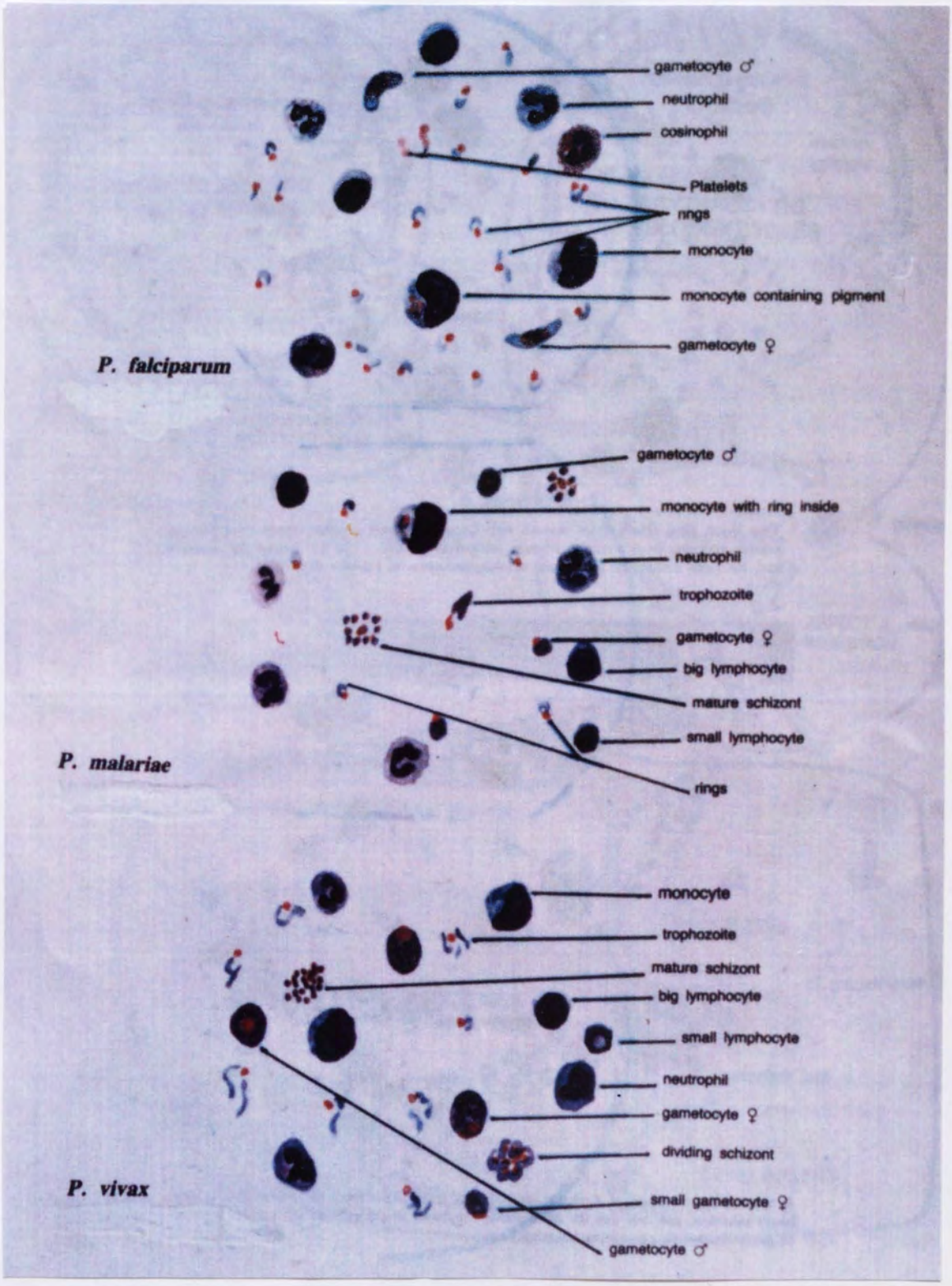
In the case of *Plasmodium vivax* it was previously thought that merozoites liberated from pre-erythrocytic schizonts re-invade fresh liver cells, producing secondary exo-erythrocytic stages and that the entry of merozoites into the blood from secondary exo-erythrocytic stages was the cause of relapses (Bradley, et al 1987). It is now clear that relapses occur because of different rates of development of pre-erythrocytic schizonts

resulting in a portion of parasites to enter a cryptobiotic phase (or the dormant phase (hypnozoites) (Bruce-Chwatt, L. J., 1985).

As regards the case of *Plasmodium malariae*, the persistence of infection (sometime up to 30 years) is not due to hypnozoites (dormant phase) but due to survival of the erythrocytic stages in small numbers. This explains the recrudescences that occur over many years due to small numbers of erythrocytic forms of the parasite viable in the host, possibly as a result of the parasites changing their antigens to remain free from host immune responses. Attacks up to 20 – 30 years after an original one have been reported (Cheesbrough, M. 1987).

The life cycle of *Plasmodium falciparum* differs slightly in that, sporozoites discharged from the salivary gland of the mosquito develop in the liver cells and discharge merozoites into the blood stream. Then some merozoites from the blood are transformed into gametocytes which, when taken up by the mosquito, initiate sexual development in the midgut.

Fig. 1. Sketches of Microscopic Fields of thick blood fillms with Different Plasmodia (See also appendix for outline of the life cycle).



Since relapses do not occur it can be assumed that the sporozoites develop uniformly, producing pre-erythrocytic schizonts at the same time and these schizonts, once formed, discharge all the merozoites simultaneously and that they do not remain dormant.

With *Plasmodium falciparum* infection, exceptionally, as the parasite continues to grow, the red cell membrane becomes sticky. The cells therefore tend to adhere to the endothelial lining of the capillaries of the internal organs. Interference with normal blood flow in these vessels gives rise to additional problems. Biological and antigenic diversity is a characteristic of this parasite and infections can consist of several genetically diverse parasites. (Farnet, A., et al., 1997).

In the midgut of the mosquito the gametocytes ingested with blood become male and female gametes. The male gametes (microgametes) arise by a process of exflagellation. The union of the male (microgametes) and the female gametes (macrogametes) form zygotes.

Within 4 to 6 hours of their formation, zygotes are transformed into motile organisms, the ookinetes. Ookinetes penetrate the wall of the gut and become transformed into a circular body, the oocyst. Sporozoites develop

inside the oocyst, from which they emerge and migrate to the cells of the salivary glands and enter host tissues during feeding.

As already explained, sporozoites entering the circulatory system, soon leave the blood to enter the parenchymal cells of the liver, where they undergo asexual multiplication forming schizonts out of the parenchymal cells. The schizonts eventually rupture, releasing thousands of merozoites into the bloodstream where they invade the erythrocytes. In the red blood cells, the young trophozoites (ring form) continue to grow and feed. They become actively amoeboid within the red cells.

After having grown fully, the chromatin (nuclear material) breaks into fragments, while the cytoplasm divides in such a way so as to arrange each portion with a fragment of nuclear material. Each portion of cytoplasm with a fragment of nuclear material forms a merozoite, while the red blood cell containing the merozoites forms the schizont.

When the infected red cells rupture, the merozoites are released into the blood stream. Production of gametocytes only begins after several generations of erythrocytic schizogony. These forms are derived from merozoites, which do not undergo schizogony, but continue to grow and form the male and female gametocytes that circulate in the bloodstream.

Heavy malarial infection tends to precipitate severe anaemia, by the constant rupturing and repeated digestion of red blood cells. This explains why in infants and children, severe iron deficiency anaemia is manifested particularly in cases of children with low birth weight, and children whose iron store at birth were poor due to maternal anaemia in pregnancy (Anon 1978; Bruce-Chwatt, L.J. 1977; Mc Gregor, J.D, & Avery, J.G, 1974).

Malaria is now known to cause anaemia, some cases requiring transfusion. (Rivera-Matos I. R., et al, 1997). On the other hand Falciparum malaria is a major cause of mortality in children with severe anaemia (Newton, C.R., et al, 1997).

In the diagnosis of malaria, it is of utmost importance to make the laboratory diagnosis, which is based on microscopic examination of blood. However for child survival in malaria-endemic areas, it is also thought to be important for mothers to recognise or suspect malaria in the presence of fever and other physiological and behavioural changes associated with the disease (Lubanga, R.J et al., 1997).

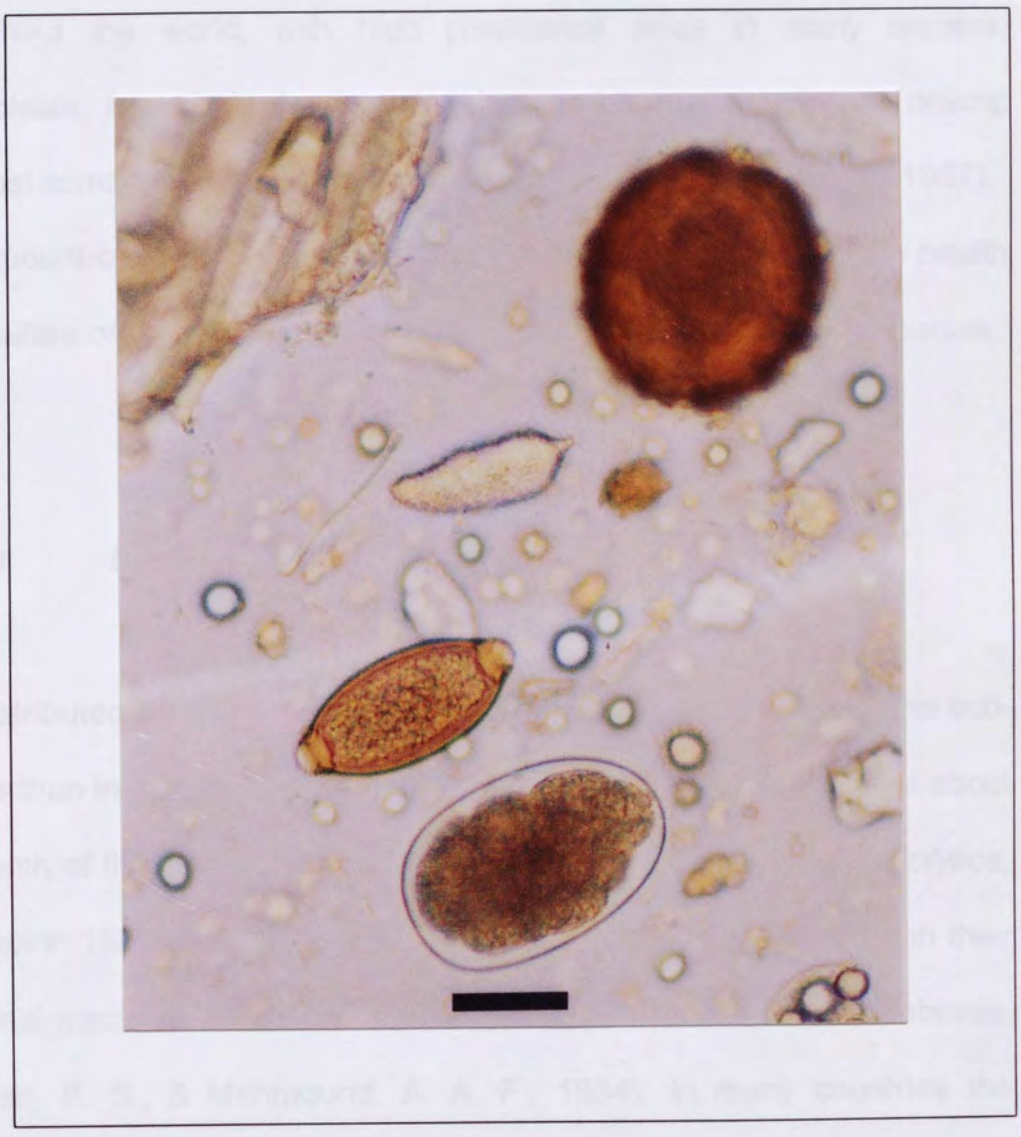
Nowadays, work is also in progress to perfect DNA probes for various species, and may prove valuable. A number of serological tests are available but are used mainly for epidemiological studies. Also a ribosomal ribonucleic acid (rRNA) system for the sensitive and accurate diagnosis of all four species of malaria pathogenic to man has been developed. Although these may well offer advantages for clinical diagnosis, this test too will be invaluable in epidemiological studies (World Health Organisation, 1986) (Waters & McCutchan, 1989; Barker, R.H., Et Al, 1986).

Tests based on the detection of malarial antigens are still at the initial stages of development. The application to the diagnosis of individual cases of malaria remains to be determined, although the required characteristics of such tests have been outlined (World Health Organisation, 1988).

Malaria in Mauritius is now believed to be eradicated, although a few remote cases have been reported particularly from incoming visitors.

1.4.2 Intestinal Parasites

Fig. 2 *Ascaris* (upper), *Trichuris* (middle) and *hookworm* (lower) eggs in the same microscopic Field, illustrating their relative sizes (Measuring bar =10 μm).



Many of the common parasites of man inhabit the gastro-intestinal tract. In the intestine, as a result of parasites, several pathological changes occur.

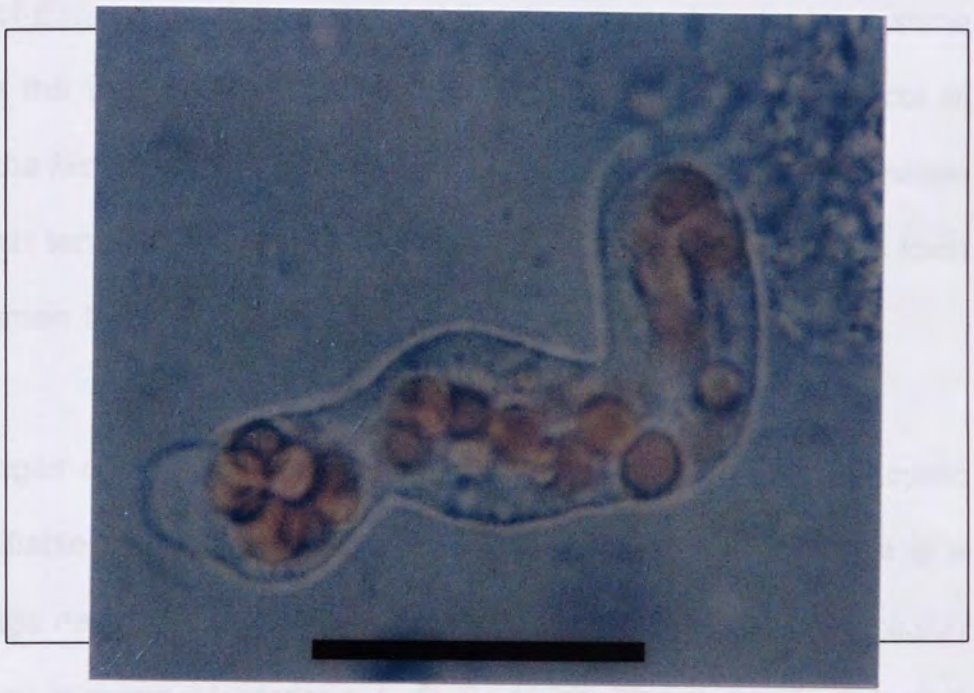
These include loss of body constituents like blood, interference with absorption of nutrients, disruption of the morphological structure of the intestine, tissue injury by the invasive nature of some parasite, and mechanical block (intestinal obstruction) due to heavy infestation of some of the parasites. Intestinal parasitic infections are distributed virtually throughout the world, with high prevalence rates in many regions. Amoebiasis, ascariasis, hookworm infection and trichuriasis are among the most common infections in the world (WHO Expert Committee, 1987). The amount of harm caused by intestinal parasitic infections to the health and welfare of individuals in communities is directly related to the species.

1.4.2.1 ***Entamoeba histolytica* Schaudinn, 1903.**

It is distributed world wide, but is more common in the tropics and the sub-tropics than in the Temperate Zone (WHO, 1987). It is believed that about one tenth of the world's population is infected with *Entamoeba histolytica,* and that in 1981, 480 million people carried *Entamoeba histolytica* in their intestinal tracts and 36 million developed invasive forms of amoebiasis, (Warren, K. S., & Mahmound, A. A. F., 1984). In many countries the prevalence exceeds 30%, and amoebic dysentry and amoebic appendicitis have a fatality rate of 0.5 – 27% (WHO, 1985). The

trophozoites live in the mucous and sub-mucous layers of the large intestine. Tissue invasion is also favoured by Trichuris and Schistosome infection.

Fig 3. *Entamoeba histolytica* live trophozoite containing many red blood cells; unstained wet mount. (Measuring bar = 10 μm)



The virulence of *Entamoeba histolytica* varies considerably. It may live as a commensal in the lumen of the large intestine or it may invade the host tissues. The factors that determine virulence of the parasite are not well understood. However it has been shown that strains isolated from asymptomatic patients are usually less invasive if not non-invasive in laboratory animals. Infections appear to be severe in young children,

pregnant women and elderly. Invasive strains occur more commonly in tropical and subtropical countries (Lucas. A. O., & Gilles, H. M., 1994). The severity of the disease also appears to vary among different ethnic groups.

Recently, viruses have been found to occur in the cytoplasm and the nucleus of *Entamoeba*. It is postulated that these viruses may have some effect on the pathogenicity of the parasites, also Cell free extracts of *Entamoeba histolytica*, produce in vitro a cytopathic effect on mammalian cells which tend to suggest that the parasite could be producing a toxin (Viqar Zaman 1995).

Three stages of the parasite, namely *trophozoites, precystic*, and *cystic* are identifiable by stool examination. . It is also believed that there is a fourth stage namely the *metacystic* stage which exists in the lower region of the small intestine (Macfarlane, L. R. S., 1960). The cyst is the infective form and when ingested hatches in the lower part of the small intestine or upper part of the large intestine. The size of the cyst ranges from 7 – 15 μm and is uniform for any particular strain. Strains can be divided into two groups: those producing cysts over 10 μm and those below 10 μm in diameter. The strains producing small cysts are now held to belong to a

separate species, *Entamoeba hartmanni,* (Lucas, A. O., Gilles, H. M., 1994).

The *trophozoite* is the growing and feeding stage. It varies in size from 12 to 30 μm, although occasionally cysts up to 90 μm may be seen (Viqar Zaman 1995). The trophozoite has no fixed shape, and the cytoplasm demonstrates two portions, a clear translucent ectoplasm, and a granular endoplasm. Red blood cells are always present inside the endoplasm.

The pseudopodia produced by an actively moving trophozoite are broad and finger-like and the parasite generally moves in one direction at a time. The nucleus is spherical and has a delicate nuclear membrane. On the internal surface of the nuclear membrane there are minute granules known as chromatin dots. In the centre of the nucleus is a single dense karyosome or nucleolus. The nuclear pattern of *Entamoeba histolytica* is used to differentiate the parasite from other species of amoeba. This however is not a reliable morphological feature as electron microscope studies show that nuclear structure varies from cell to cell (Viqar Zaman 1995).

Before encysting, the parasite rounds up and become a pre-cyst, which ceases ingesting food and begins to secrete a cyst wall. In its pre-cystic

stage, the parasite is slightly ovoid, with a blunt pseudopodium projecting from the periphery.

The cystic stage is where the parasite becomes rounded and is surrounded by a highly refractile membrane, the cyst wall. Originally, the cyst is uninucleate. The nucleus soon divides by binary fission to form a binucleate and finally a quadrinucleate cyst. It is then known as a mature cyst. The morphology of the nuclei of the cyst is similar to that of the trophozoite, with a row of chromatin dots at the periphery and a central karyosome. The cyst also has two other inclusions, the glycogen vacuole and the chromatoid body. Both of these inclusions become smaller as the cysts ages. The glycogen vacuole is known to act as a food reserve (Garcia, I. S., & Bruckner, D. A., 1993) (Kotpal, R. L., 1978) but the function of the chromatoid body is not known.

Mature quadrinucleate cysts are passed in stools, and can resist heat up to 50^0 C. Also chlorine, at the strength used for bacteriological sterilisation of public water supply does not destroy the cysts.

Transmission of the parasite from man to man occurs through the ingestion of mature cysts of the parasite by the faecal-oral route, usually through contaminated food and water. Faecal contamination of drinking

water, vegetables and food are therefore the primary causes of transmission. Occasionally drinking water supply contaminated with infected faeces gives rise to epidemics (Crewe, W., & Haddock, D. R. W. 1985) (Muller, R. & Baker, J. R., 1990).

Excystation occurs in the lower region of the small intestine and quadrinucleate cysts give rise to a metacyst which rapidly divides to give rise to eight amoebae. These enter the large intestine.

After excystation which may take four to five days, the liberated trophozoites burrows into the wall of the intestine, reaching the submucous coat. They do so by penetrating the columnar epithelium of the mucous membrane by their amoeboid activity and by dissolving the intestinal epithelial cells with a proteolytic enzyme they secrete (Panjarathinam, R., 1990) (Viqar Zaman 1995).

Multiplication takes place in the submucous membrane. They then destroy the tissue in the vicinity utilising the lysed tissue as food. By passing in various directions, dissolving surrounding tissue, large areas of submucosa are destroyed, with underlying complication. In addition, invasion of tissues results in bleeding and the red cells are quickly ingested by the trophozoites.

When invading the intestinal tissues, destruction of the epithelial lining of the large intestine results in ulcers.

The trophozoites from the intestinal tissues may also be carried to the extraintestinal organs via the portal circulation. The most commonly involved extraintestinal organ is the liver. In the liver the parasites may produce localised abscesses due to their cytolytic activity. The parasite may also be carried via the circulatory system, to other organs such as the brain

Intestinal complications also include haemorrhage, and the presenting symptom is dysentery. At times severe intestinal haemorrhage occurs. In either case blood loss occurs resulting in anaemia. When the red blood cells lost during dysentery or intestinal haemorrhage are rebuilt, iron stores are used and hence iron depletion may occur.

Diagnosis is almost exclusively by microscopy of stools or pus from liver abscess, which reveals the presence of either the trophozoites or the cysts, although, sometimes serological examination may prove useful. A novel method for DNA extraction extraction has now been described for the detection of intestinal protozoa (da Silva, A. J., et al 1999).

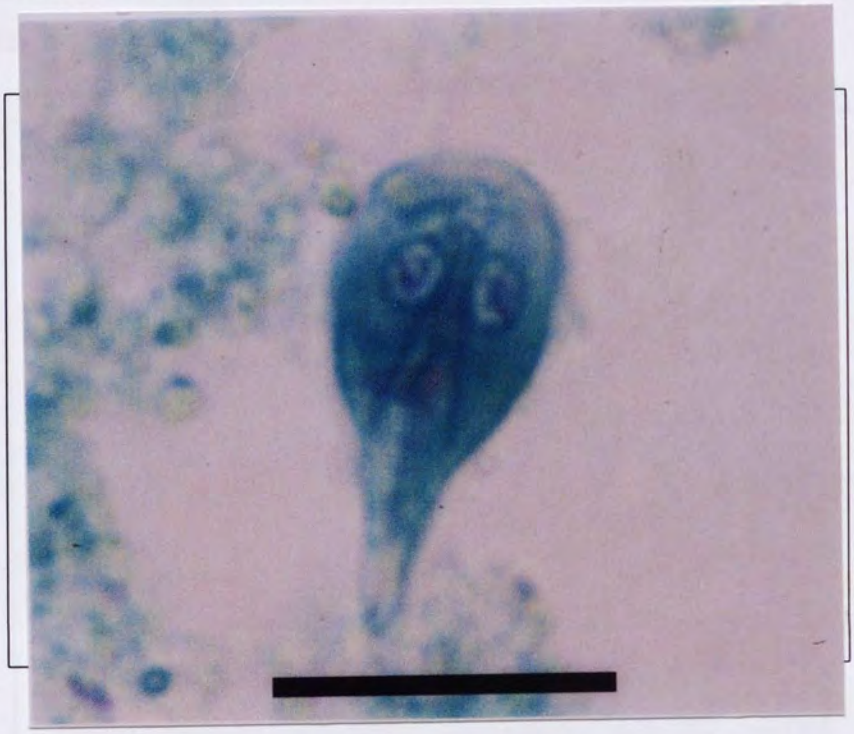
1.4.2.2 *Giardia lamblia/ Giardia intestinalis* (Lambl,1859)

Giardia lamblia is a flagellated protozoan that inhabits the upper small intestine of its vertebrate host and is a major cause of enteric disease worldwide. It is a binucleated flagellated protozoan parasite inhabiting the duodenum and upper part of the ileum of man. It is common in every part of the world and is especially prevalent in children in developing countries causing diarrhoea and weight loss in some (Lujan, H.D. et al 1997). It is a major cause of enteric disease world wide (Lujan, L.D., 1997).

Giardia exist in two phases-the trophozoites and the cystic phase. The trophozoites are symmetrical in shape resembling badminton rackets, when viewed flat. Two oval-shaped nuclei and four pairs of flagella are also clearly visible. When viewed side-on, it is like a longitudinally split pear. On the concave ventral surface, a sucking disk is also visible. It is believed that with the help of this sucking disc, the parasite attaches itself on the convex surface of the epithelial cells of the intestine. It is also believe that host proteases activate a lectin in *Giardia* that promotes attachment to enterocytes. In this way and in so doing, *Giardia* disturbs the intestinal function of the host (Crewe, W., & Haddock, D., R., W., 1985).

During movement, the parasite rolls on itself displaying what is known as the 'falling leaf' movement. Whenever conditions in the duodenum

Fig. 4. *Giardia lamblia trophozoite*, stained with trichome stains (Measuring bar = 10 μm).

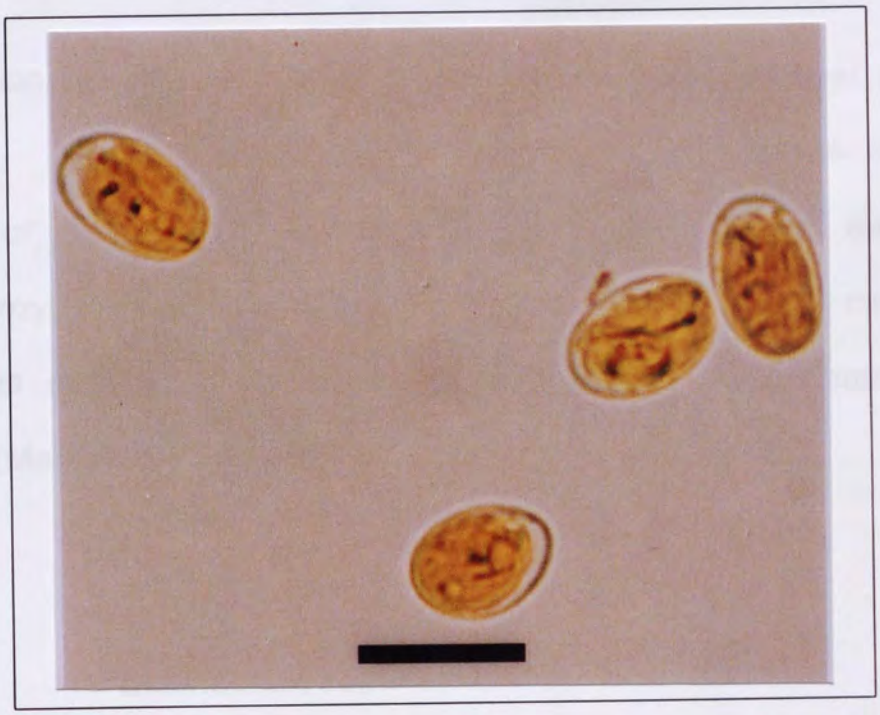


become unfavorable, particularly when the environment becomes acidic, encystment occurs (Panjarathinam, R., 1990) . Cysts of *Giardia lamblia* are ovoid to ellipsoid in shape, measuring 11 to 14 μm.; they have a distinct wall and each contains four small nuclei. The cytoplasm is generally shrunken away from the cyst wall leaving a space.

Transmission occurs from person to person through ingestion of food and water contaminated with cysts. The fact is that the main routes of infection may be contaminated drinking water (Saidi, S.M., et al 1997). Cysts are

distributed homogeneously in contaminated river water. (Payment, P. et al, 1997), while *Giardia* is also found to be one of the contributing factors of waterborne diarrhoeal outbreaks (Steiner, T.S., 1997).

Fig. 5. *Giardia lamblia* cysts, iodine wet mount. (Measuring bar = 10 μm)



Within thirty minutes of ingestion, in the upper region of the small intestine, excystation occurs and the cyst hatches out two trophozoites. These then multiply by binary fission into enormous numbers, and colonise the duodenum. Sometimes, to avoid the high acidity of the duodenum, *Giardia* often localises in the gall bladder, causing cholecystopathy (Chatterjee, K.

D., 1982). Apart from cholecystitis as explained above, the parasite is found to be implicated in the intestinal malabsorption syndrome.

The parasite produces a wide range of gastrointestinal symptoms especially in children. These include vomiting, flatulence and diarrhoea.

Risk of infection with *Giardia lamblia* by age two is nearly 92% (Fraser, D et al, 1997). Diagnosis is made by the examination of stools for trophozoites or cysts, and also duodenal aspiration. Studies have also shown that enzyme immunoassay for the detection of copro-antigens may be almost as sensitive for identifying *Giardia* infection as repeated microscopy (Mank, T.G., et al, 1997)

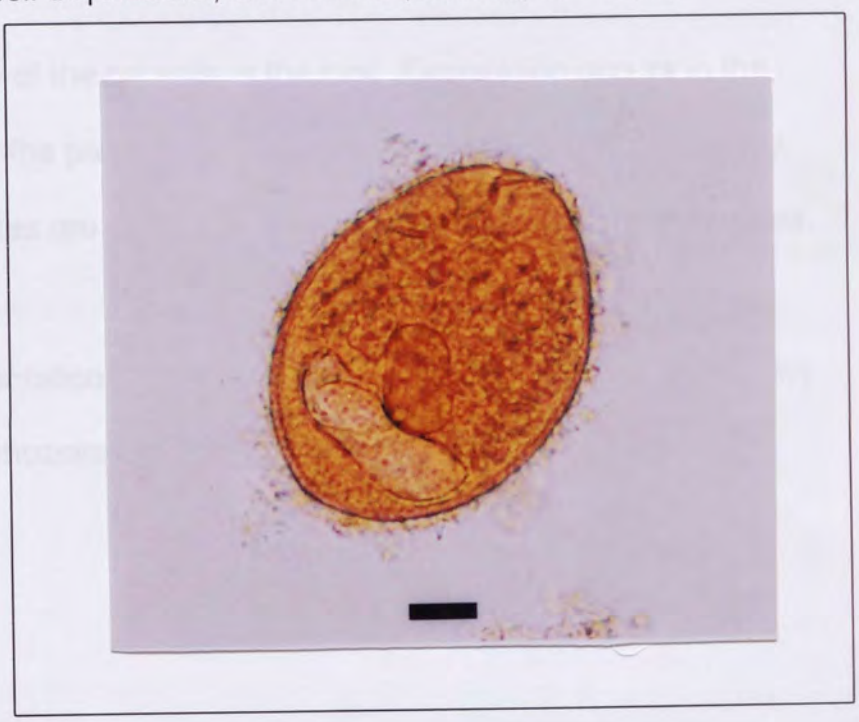
1.4.2.3 *Balantidium coli*

This is the largest protozoan and the only ciliate that infects man (Cheesborough, M., 1987; Finegold, S. M., & Baron, E., J., 1986). The most endemic area is New Guinea, where there is a close association between man and pigs.

The parasite has two stages in its life cycle, the trophozoite stage and the

cystic stage. Trophozoites are 30 to 300 μm in size and have two contractile vacuoles and a large macronucleus often reniform in shape. At the anterior end it has a funnel-shaped depression known as the peristome which leads to the mouth or the cystosome. The cilia, which are organs of locomotion, are embeded in the pellicle in longitudinal rows.

Fig. 6. *Balantidium coli* trophozoite, wet mount. (Measuring bar = 10 μm)



The cyst is spherical and is 40 to 60 μm in diameter. The cyst wall is thick and transparent and the parasite is visible inside the cyst. The contractile vacuoles, the macro and the micronucleus and other internal structures are similar to those of the trophozoites.

Reproduction is by binary fission as well as conjugation.

Human infection occurs from close associations between humans and pigs. Once the infection is established in human, direct human to human transmission also occurs.

The infective stage of the parasite is the cyst. Excystation occurs in the small intestine and the parasite multiplies in the large intestine by binary fission. The parasites are passed in stools both as cysts and trophozoites.

Diagnosis is by isolation of the parasite in faeces. In acute dysentery, actively motile trophozoites are seen.

1.4.2.4 *Ascaris lumbricoides* (Linnaeus, 1758)

Ascaris lumbricoides, L, is the common roundworm. It is distributed worldwide but is especially prevalent in moist and warm climates. In some rural areas in the tropics the infection rates may reach 100 % of the population. It is generally more common in children who also tend to carry

higher worm load. A concurrent socio-economic household survey in southeastern Madagascar showed that *Ascaris lumbricoides* aggregations were associated with gender, housing style, ethnicity, and agricultural factors suggesting that intensity of infection is influenced by gender-related behavioural and environmental factors that contribute to exposure. (Kightlinger, L.K., et al, 1998). If untreated, children tend to retain initial medium infections compared to adults (Peng, W., et al, 1998).

The adult worm lives in the small intestine of man. It is cylindrical with tapering ends resembling an ordinary earthworm. When fresh from the intestine, it is light brown or pink in colour, but gradually changes to white.

The male measures 12 to 31 centimetres in length, with a maximum diameter of 3 to 4 mm. The tail end of the male roundworm is curved. The female is longer and stouter than the male and reaches a length of 40 cm, with a maximum diameter of 5mm. The tail end is straight. The head of the roundworm has three lips at the anterior end which carry minute teeth or denticles along their margins. In cross-section the worm has a thick cuticle. Adjacent to the cuticle is the hypodermis. The hypodermis in turn projects into the body cavity as lateral chords. The somatic muscle cells are large and elongated and lie adjacent to the hypodermis. By the activity of these muscles, the worm is able to maintain

its position in the small intestine.

If the somatic muscles are paralysed by antihelminthics, the worms are expelled by normal peristaltic movement.

The reproductive organs and the digestive tracts of the round worm float inside the body cavity or haemocoel. The male has two broad spicules which may protrude from the cloaca. In the female the vulva opens at the junction of the anterior and middle thirds of the body. This part of the worm is slightly narrow and is known as the vulvar waist.

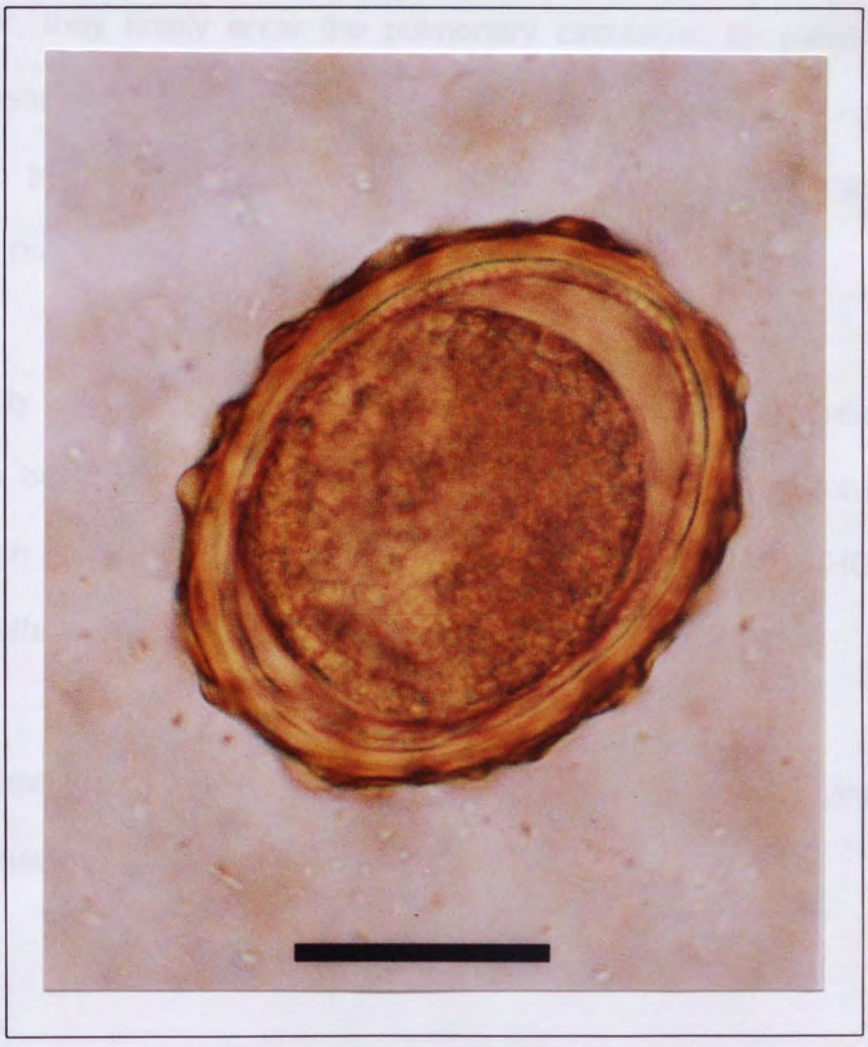
Larvae of *Ascaris lumbricoides* may be seen in infected lungs. They are 2 mm in length and 75 μm in diameter. The larva has a central intestine, paired excretory columns and prominent lateral alae. The life span of the adult worm is six months to one year, and the female deposits an average of 200 000 ova everyday, which pass out in the faeces. The eggs are resistant to various disinfectants and, while they take only three to four weeks to develop to the infective stage, they may remain viable for years in moist soil (Anderson, R. M., 1986; Viqar Zaman 1995).

Continuance of species is maintained by transference from one individual to another when eggs are ingested with food and drink. Nowadays it has

also been observed that transference also occurs through raw sewage (Bouhoum, K., & Schwartzbrod, J 1998).

When freshly passed, *Ascaris* ova are not infective. In 10 to 40 days, when in moist shady soil, the unsegmented ova developed into a rhabditiform larvae. At this stage, the ovum is very infective.

Fig. 7. Normal fertile *Ascaris lumbricoides* egg. (Measuring bar = 10 μm)



When ingested with food, drink or raw vegetables, the embryonated eggs pass down to the duodenum and the larvae are liberated in the small intestine. The larvae then burrow their way through the mucous membrane, and it is understood that in so doing the loss of blood is occasioned in the host.

The burrowing larvae, soon enter the portal vein and are carried by the circulating blood to the liver where the larvae live for another 3 to 4 days. From the liver, they finally enter the pulmonary circulation by passing through the heart, and in the lung they develop further. From the lungs they crawl up to the larynx, where they are swallowed back into the intestine, their normal abode, and the cycle continues.

In the majority of cases infected individuals remain asymptomatic. However, it is believed that the presence of *Ascaris* causes nutritional problems, which in turn hinder the normal development of children (WHO, Informal Consultation on Intestinal Helminth Infection, 9-12 July,1990).

The larval migration of *Ascaris* through the lungs may produce varying degrees of pneumonitis and bronchospasm.

Ascaris may also produce life-threatening disease when large numbers of worms are entangled to each other forming a bolus which blocks the intestinal lumen (de Silva, N.R., et al, 1997). Also in ectopic migration when the worms enters the appendix, the bile duct and the pancreatic duct causing abscesses.

Diagnosis is by detecting *Ascaris* eggs in the faeces. *Ascaris* can also be detected by sonographical studies. (Hoffmann, H., et al. 1997; Khari, A., et al, 1998; Schulman, A 1998).

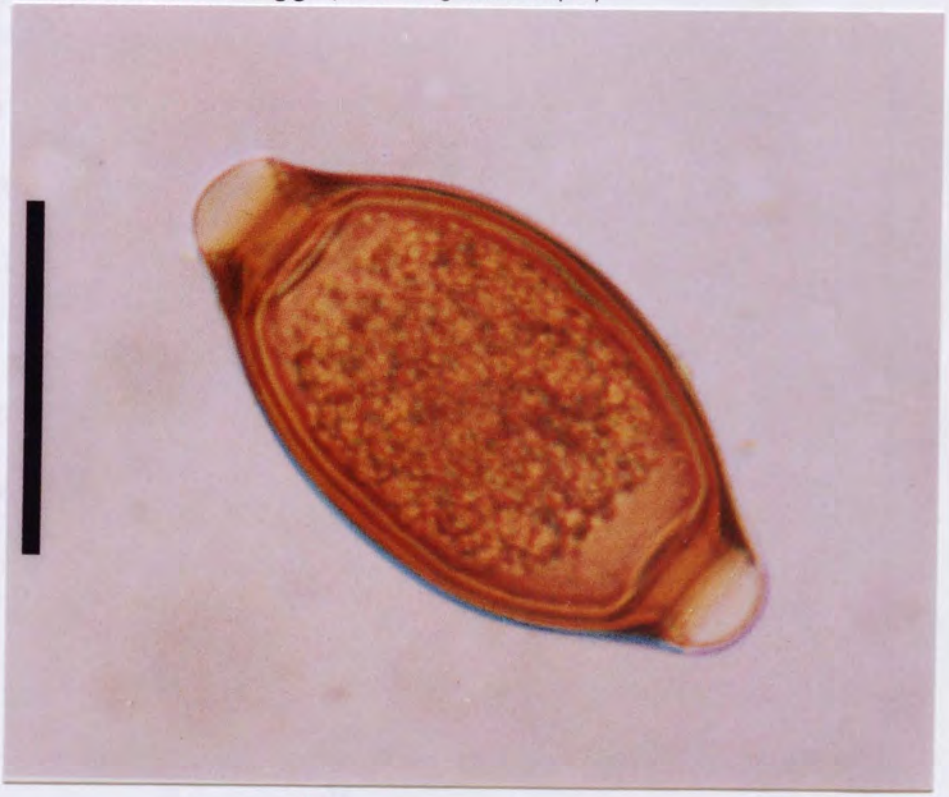
Ascaris remains the most common intestinal nematode in the world followed by hookworm, with significant economic, social, and medical impact (Sarinas, P.S., Chitkara, R.K., 1997). Heavy infestation with ascaris further results in intestinal obstruction (Wasadikar, P.P., Kulkarni, A.B., 1997; Salman, A.B., 1997), causing morbidity and mortality (de Silva, N.R., Guyatt, H.L., Bundy, D.A.,1997). There is also evidence to support the induction of appendicitis as a result of ascariasis, (Singh, P.A., Gupta, S.C., Agrawal, R., 1997). Nowadays it is also known that high intensities of the worm severely affect mental performance in children if they are not dewormed, (Watkins, W.E., Politt, E., 1997).

The effect of worm infestation is significant, it is estimated that at least 13,000 million individuals harbour the parasite, and that approximately 10,000 deaths due to the infection occur annually on a global basis (de Silva, N.R., Chan, M.S., Bundy, D.A 1997). (For general life cycles, see appendix).

1.4.2.5 *Trichuris trichiura* (Linnaeus 1771)

Commonly known as the whipworm, it has a worldwide distribution being the most common nematode in some tropical regions (Panjarathinam, R., 1990; Cheesborough, M., 1987) (See appendix).

Fig. 8. Typical *Trichuris trichiura egg*. (Measuring bar = 10 μm)



The adult male is 30 to 45 mm long and the adult female is 35 to 50 mm long. The anterior three-fifths of the worm is elongated and thin and the posterior two-fifths is fleshy and bulbous, making the worm look like a whip. They are present in the caecum.

One important feature of this group of worms is that they all have a capillary-like oesophagus surrounded by gland cells known as stichocytes.

The eggs are barrel-shaped, brownish, with length varying between 22 to 50μm, with a transparent blister-like plug at the lateral ends. When freshly passed, the eggs are non-infective. The eggs develop further in the soil and become infective in about three week's time. Infection is by the ingestion of embryonated eggs. The larvae penetrate the wall of the gut after which they return to the lumen to mature. The adult worms attach themselves to the large intestine by the anterior part forming a funnel-like structure in the superficial part of the epithelium, while the posterior parts hang freely in the lumen of the bowel. The worms survive for many years.

Heavy infection causes severe colitis with significant blood in faeces (Ananthakrisnan, S., 1997). In children, it is believed that bleeding from damaged and inflamed mucous membrane anaemia associated with

dyspnoea can result in cardiac failure. Occasionally, the worms may lodge in the lumen of the appendix causing acute appendicitis.

Diagnosis is by finding the typicaly barrel-shaped eggs in faeces.

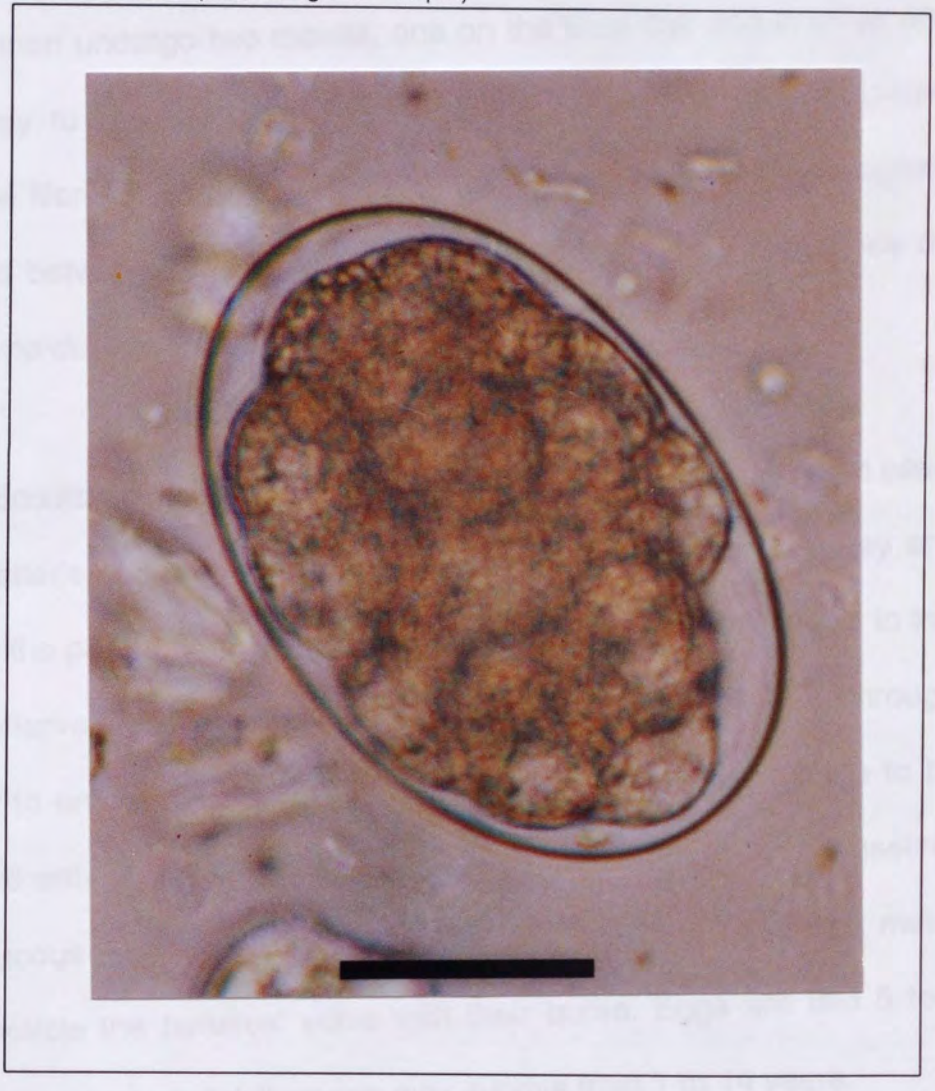
1.4.2.6 Hookworms

The common human species are *Necator americanus* and *Ancylostoma duodenale*. *Necator americanus* is found mainly in moist tropics and *Ancylostoma duodenale* in dryer and cooler areas. They are believed to infect approximately one billion people worldwide (Stoltzfus, R.J. 1997). Recent estimates indicate that hookworms infect approximately 1.3 billion people worldwide with 96 million morbidity (Albonico, M., Savioli, L, 1997).

The adult worms are cylindrical with their head bent sharply backward giving them a hook-like appearance. The males are smaller and possess a bursa at their posterior ends. Their buccal capsule and the arrangement of rays in the bursa mainly differentiate hookworms' species. The eggs are however indistinguishable. They are all ovoid between 74 to 76 μm in length. They have a thin transparent shell, which allows easy exit of the larvae when they are mature.

When laid in the host intestine, they are single celled but by the time they are passed in faeces they are in a 4 to 8 celled stage. This in fact is the diagnostic stage of the parasite.

Fig 9. Hookworm ova. (Measuring bar = 10 μm)



Several hours after having been laid, rhabditiform larvae are formed. These resemble *Strongyloides* larvae except for morphological differences.

The rhabditiform larvae which are the first stage larvae, hatches out after 24 to 48 hours. They feed actively on organic debris and bacteria in the soil. They then undergo two moults, one on the third day and another on the fifth day to become the filariform lavae which are the third stage larvae. The filariform larvae are actively motile. There are morphological differences between the filariforms of *Necator americanus* and those of *Ancylostoma duodenale.*

Infection occurs when filariform larvae penetrate the skin of host and pass into the latter's blood circulation. Once in the blood circulation, they are carried to the pulmonary blood vessel by passing through the heart. In the lungs the larvae leave the pulmonary blood vessels by breaking through the latter to enter the alveoli from where they crawl to the trachea to be swallowed with saliva. In the small intestine, the larvae attach themselves to the mucous membrane to mature into adults. During copulation, males firmly encircle the females' vulva with their bursa. Eggs are laid 5 to 7 weeks after infection. Adult worms may survive from 1 to 14 years.

Ancylostoma duodenale has a second mode of infection. The filariform larvae may be swallowed directly by eating contaminated leafy vegetables and other food contaminated with soil containing the larvae.

Disease caused by hookworm may be in the following form:

- During the stage of skin penetration, larvae may produce intense itching and dermatitis at the site of entry.
- Larval passage through the lungs may cause pneumonitis and bronchitis.
- The fact that blood loss occurs from sucking by the worm, and from continuous bleeding at the site of attachment, anaemia also occurs as a result of the infection.
- Infected children are often oedematous showing the loss of protein (protein-losing enteropathy).

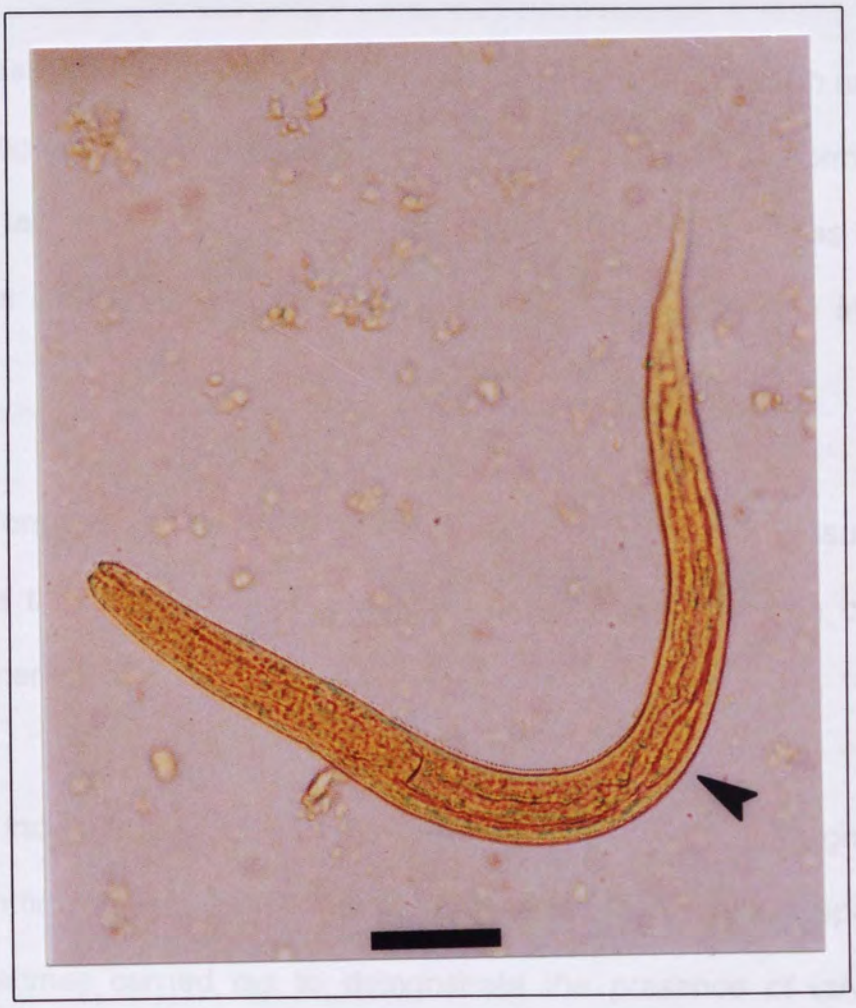
Hookworm infections often begin in childhood. The worm enters the body through the skin and reaches the highest number at the end of adolescence and young adulthood (Santiso, R., 1997).

Diagnosis is by finding eggs in faeces. Adult worms have also been detected and retrieved in upper gastrointestinal endoscopy (Kato, 1997).

1.4.2.7 *Strongyloides stercoralis* (Bavay, 1876)

This is widely distributed in the tropical areas. Its presence in Poland suggests that it may also spread in temperate regions. The adult parasite

Fig 10. Strongyloides stercoralis larvae. (Measuring bar = 10 μm). (Arrow – genital primordium).



resides in the mucosa of the small intestine and is about 2.7 mm long and 30 to 40 μm wide. It reproduces by parthenogenesis. At any time between 10 and 20 eggs are carried in the two uteri. The eggs are thin-shelled, ovoid and transparent. The larvae usually hatch in the intestinal tissues and are passed in the faeces. (For difference in appearance of *Enterobius* and hookworm larvae, see appendix).

The parasite shows three different modes of development, which are: Free-living adults producing eggs, rhabditiform larvae and filariform larvae, with the latter penetrating the skin, to enter the circulation, pass through the lungs and are swallowed to become adult females in the intestinal tissues.

Rhabditiform larvae becoming filariform larvae in the host tissues can penetrate the intestine or peri-anal skin to undergo the same cycle of development.

Disease includes epigastric pain, diarrhoea and vomiting. Diagnosis is based on finding rhabditiform larvae in the faeces. Duodenal aspirations are sometimes carried out to demonstrate the presence of larvae. In severe dissemination *Strongyloides* larvae may be seen in the sputum.

1.4.2.8 *Enterobius vermicularis*

This is one of the few parasites, which is more prevalent in the temperate regions of the world than the tropics. Children are more often involved than adults.

The male is approximately 5 mm long with a diameter varying between 0.1 to 0.2 mm. The female is about 13 mm long and 0.3 to 0.5 mm diameter. The eggs, which are between 50 to 60 mm long and between 20 to 30 mm wide, appear, flattened on one side. They have a thick transparent shell and are embryonated when passed but become infective within a few hours.

The male has a single curved tail and a spicule, and the gravid female has two distended uteri, which almost fill the whole body. Adult parasites are located in the caecal region and the female deposits her eggs on the anus and perianal skin. Direct human to human infection occurs by inhaling and swallowing the eggs. In addition autoinfection occurs by contamination of the fingers. There is no visceral migration and the larva matures in the lumen of the intestinal tract. The life cycle is completed in about six weeks.

The most presenting symptom is pruritus ani, which can be very troublesome and occurs more often during the night. Occasionally, undergo ectopic migration and may enter the female genital tract. Inside the uterus or fallopian tube they may become encapsulated and produce symptoms of salpingitis. The parasite may also become lodged in the lumen of the appendix leading to appendicitis.

Unlike other intestinal nematodes, the eggs are not usually found in the faeces. The best method is to look for them around the anus by taking anal swabs or by using cellophane adhesive tape. Sometimes intact worms are passed out in the faeces.

1.4.2.9 *Taenia saginata*

This forms part of the group called *Cestodes* or tapeworm. Distribution is cosmopolitan and occurs wherever beef is eaten. The adult worm is about five metres long and has about 1000 to 2000 proglottids. The scolex or head is rhomboid and has four circular suckers. There are no rostellar (protruberant anterior part) prominences and there are no hooks. Behind the scolex is the unsegmented neck followed by a row of immature proglottids, mature proglottids and the gravid proglottids.

The eggs are spherical and are between 31 and 43 μm in diameter. They contain a thin transparent outer envelope, which surrounds a thick brown embryophore or eggshell. The eggshell is made up of longitudinal blocks giving the eggs a radially striated appearance. Inside the egg is the hexacanth embryo or oncosphere, which bears six hooks.

Taenia saginata is an obligatory parasite of man. The adults are located in the small intestine and the gravid progglottids migrate out of the anus or are passed in faeces. Cattle become infected when they ingest eggs from the pasture. On ingestion, the oncosphere hatches out and penetrates the intestine. From there it is carried in the circulation and is transformed into a cysticercus (larval form which has a fluid filled bladder) stage in the muscle where it is known as *Cisticercus bovis.* Human become infected when they eat uncooked or improperly cooked beef containing the cisticercus. After ingestion, the scolex of the *Cisticercus,* evaginates in the small intestine, attaches itself to the mucus membrane and develops into adult.

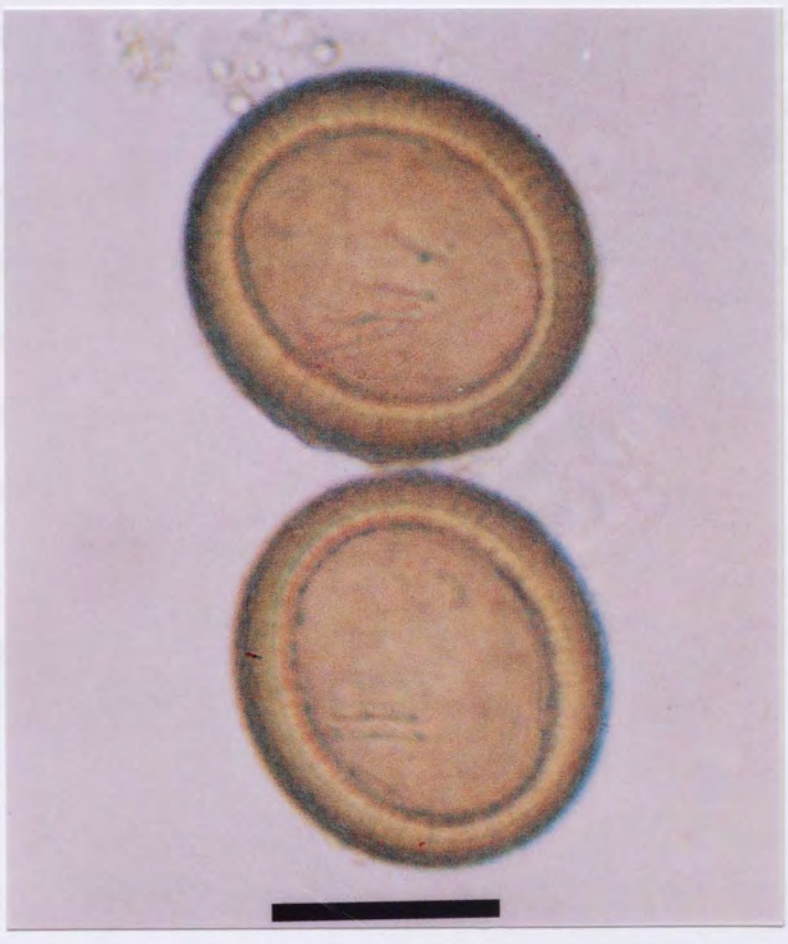
The majority of cases are symptomless. Diagnosis is by detecting eggs or segments (proglottids) in the faeces. Sometimes gravid proglottids disintegrate near the anus where eggs get attached to the anal and

perianal region. Diagnosis therefore is also made by the adhesive cellophane tape technique used for enterobiasis.

1.4.2.10 *Taenia solium*

This is endemic in all parts of the world where pork or pork products are eaten. The species is shorter than *Taenia saginata*, often less than three

Fig. 11. Taenia spp. Eggs. (Measuring bar = 10 μm)



metres. The numbers of proglottids are less than 1000. The scolex has 4 suckers, with rostellar prominence and with hooks.

The life cycle is similar to that of *Taenia saginata*, except that the intermediate host is swine and not cattle, and the larval stage, which is known as *Cysticercus cellulosaae* can also develop in man.

Presence of adult worms is asymptomatic as in *T.saginata.* The larval stage, however may produce disease which includes epileptiform attacks, headache and dizziness, blurred vision etc. Man becomes infected by the accidental ingestion of eggs of *Taenia solium* with contaminated food and water. Retro infection also occurs in persons harbouring the adult worms, when the proglottids are regurgitated into the stomach. In the stomach, when the proglottids get disintegrated large numbers of eggs are released. The oncospheres from these eggs then find their way into various parts of the body and develop into cysticerci.

Diagnosis is by detecting eggs or segments in faeces. In the case of cysticercosis, a CT scan is the most useful diagnostic technique.

1.4.3 Urinary parasites

The only common urinary parasite present in Mauritius is a trematode called *Schistosoma haematobium,* commonly known as Bilharzia. It is widespread in Africa and the Middle East with small foci in India.

Schistosomes are dioecious (a condition where male and female gonads are found in different individuals) and measure 10 to 20 mm in length and 0.5 to 1 mm in width. The male has a deep ventral groove known as the gynaecophoric canal, in which the female lies during copulation. Both sexes have two suckers, an anterior and a ventral sucker. The gut of the female worm appears dark because it is filled with deposit of haematin (breakdown products of Haemoglobin).

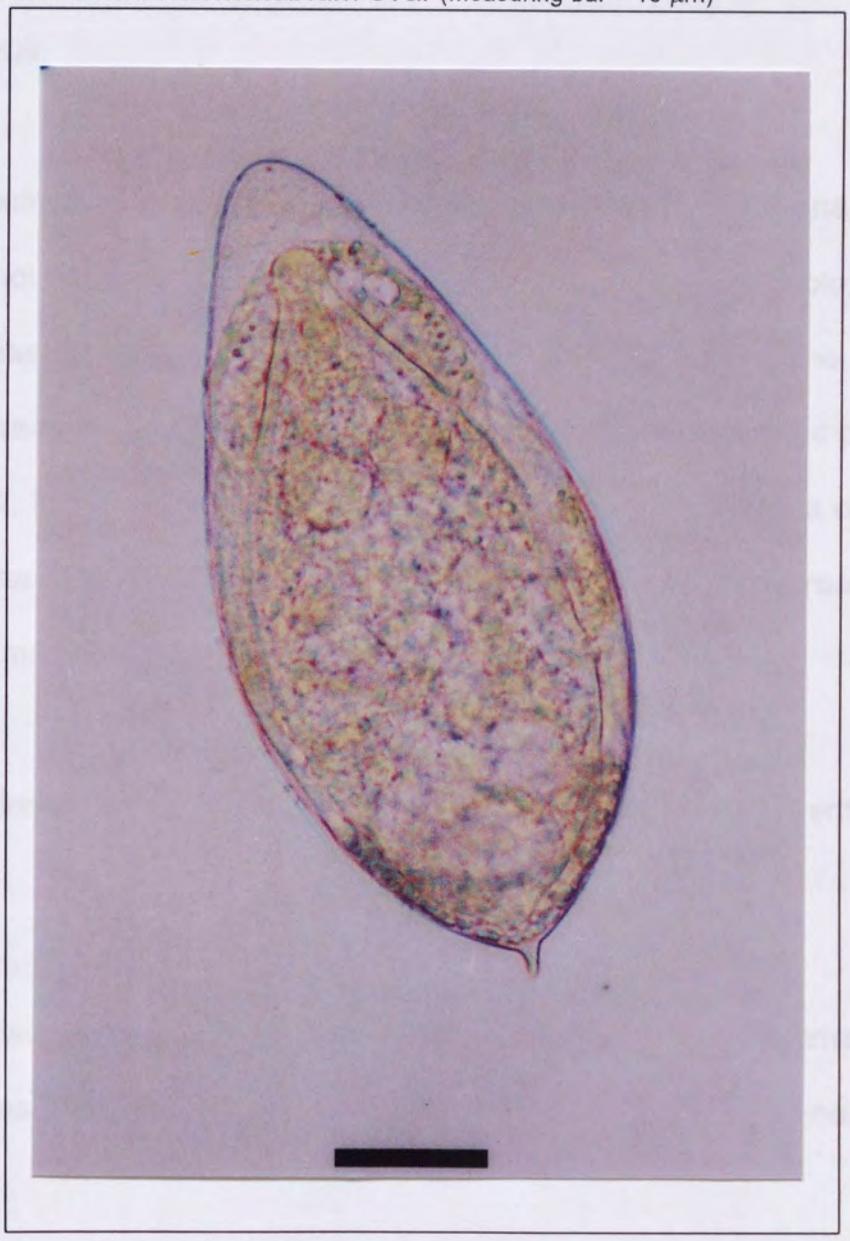
The life span may extend to 30 years but the mean longevity is about 5 years. The schistosomes are called digenetic trematodes as their life cycle has an asexual phase in snails (the intermediate host) and a sexual phase in mammalian host (the definitive host).

The egg contains a fully formed miracidium which, hatches out when the egg is immersed in water. The miracidium swims actively to penetrate an

appropriate snail host. After entry into the snail, it develops through various stages to become cercariae, the infective stage for man.

The cercariae emerge from the snail mainly on exposure to light, and infect the vertebrate host by penetrating the skin.

Fig. 12. Schistosoma haematobium ova. (Measuring bar = 10 μm)



All *Schistosome cercariae* have a bifid tail and do not have a pharynx. The tail of the cercaria is shed during penetration and the parasite is transformed into a schistosomula inside the host tissues.

The schistosomula first enters the systemic circulation and then finds its way into the portal circulation. *S.haematobium* worms generally remain in the systemic circulation and mature in the blood vessels of the uretric and vesical plexus.

Eggs produced by *S.haematobium* are discharged mainly in the urine. The adults do not multiply and the egg is the main cause of pathology in schistosomiasis. The eggs penetrate the blood vessels and the host tissues by secreting proteolytic enzymes through ultra-microscopic pores in their shell. However, many eggs become stranded in the tissues or are carried by the blood stream to other organs of the body. The host reaction to the eggs may vary from small granulomas to extensive fibrosis.

Cercarcial dermatitis often manifests itself with rash at the point of entry of cercariae.

When the worms migrate to the lungs cough fever and asthma-like symptoms may develop. Diagnosis is by the detection of eggs in urine.

1.5 Anaemia

Anaemia is defined as the condition in which the blood, particularly the red blood cell, is deficient in haemoglobin and therefore deficient in oxygen carrying capacity. It is a reduction either in the concentration of haemoglobin in the blood or in numbers of circulating erythrocytes. Usually, if one of these is reduced then the other is reduced also, because haemoglobin is contained within erythrocytes. There are circumstances however, in which haemoglobin concentration may be mildly reduced with a normal red cell count. This occurs particularly in early stages of iron deficiency anaemia (Phillips, J.D., 1995).

Anaemia is estimated to affect one-half of school-age children in developing countries (Stoltzfus, R.J. 1997). Anaemia is not a single disease entity. It is a consequence of one of a number of disorders underlying defective erythrocyte production or excessive loss of erythrocytes from the circulation.

Haemopoiesis (blood cell production) takes place in the bone marrow. All blood cells (erythrocytes, leucocytes and platelets) are derived from a single clone of primitive cells, the pluripotent stem cell. (Hoffbrand, A. V., 1993). Erythropoiesis is a component of haemopoiesis, which produces

mature red blood cells. In the production erythrocytes, protein, iron, vitamin B_{12} , folic acid, vitamin B_6 and trace metals like cobalt and nickel are required. Lack of any one or more of these results in abnormalities in red cell production. Anaemias due to deficiencies in iron is quite common throughout the world. Studies carried in Tanzania have demonstrated that 90% of all severely anaemic subjects were iron deficient (Tatala,S. 1998). On the other hand, it has also been shown that dietary protein has no influence on the degree of anaemia (Katunguka-Rwakishaya, E, 1997).

Haemoglobin is the most important agent in red cells. It is a haemoprotein. The protein part, known as globin, consists of four peptide chains arranged in loops. The haemoglobin molecule has the haem group surrounded by two pairs of polypeptide chains. Deficiency of iron, or abnormalities in globin, causes a decrease in haemoglobin production, or anaemia.

Iron deficiency anaemia is the commonest type of anaemia. It is a symptomatic state, which is always secondary to some underlying causative disorder; generally it represents chronic and usually hidden blood loss. It develops when the supply of iron to the bone marrow is insufficient for the requirements of haemoglobin synthesis. Studies have demonstrated that certain parasitic infections, in particular *Plasmodium*

falciparum, cause suppression of the bone marrow response to erythropoiesis (Khurtzhals , J.A., 1997).

The human body is normally in a state of positive iron balance. When a negative iron balance occurs due to blood loss, increased requirements or impaired absorption, the deficit is made good by iron mobilised from the tissue stores. In this way, an adequate supply of iron for haemoglobin formation is maintained. It is only when the tissue stores are exhausted that the supply of iron to the marrow for haemoglobin synthesis becomes inadequate. Thus iron deficiency develops in two stages which are first the progressive depletion and ultimate exhaustion of the available tissue iron stores, and then the development of anaemia (Bothwell, T. H., 1966; Heinrich, H. C., 1968; Boender, C. A., Verloop, M. C., 1969).

In the pathogenesis of iron deficiency anaemia, three major factors are involved. These are increased physiological demand of the body for iron, loss of blood by haemorrhage and inadequate iron intake. In general, blood loss is by far the most important factor. However very frequently, more than one factor contributes to the anaemia. In children increased physiological demand for iron occurs during the period of growth. Blood loss causing erythrocytes to leave the body results in significant lowering of the total body iron given that 60 to 70 per cent of the total iron content

of the body is contained in haemoglobin. When the iron reserve is exhausted continued bleeding causes iron deficiency.

Inadequate intake arises from nutritional deficiency or impaired absorption. Parasitic infection is one of factors responsible for both malabsorption and malnourishment (Bellamy, C. 1998).

Studies carried out by Dr. Oski have demonstrated that socioeconomic status is an important factor in the development of iron deficiency (Oski, A.F., 1993). There are two broad types of dietary iron. About 90 percent of iron from food is in the form of iron salts and is referred to as non haem-iron. The absorption of this type of iron depends, amongst other factors, on the other components of the diet. Food items like ascorbic acid, meat, fish, and poultry enhance the absorption of non-haem iron (Derman, D.P., 1980).

Although *serum iron concentration* and *total iron-binding capacity* are widely used tests in the diagnosis of iron deficiency, assay of *serum ferritin* concentration is a much more sensitive and reliable means of demonstrating this disorder (Hastka, J., Lasserre, J., Schwarzbeck, A., Reitter, A., Hehlmann, R., 1996; Fairbanks, V. F., Klee, G. G., 1987).

It is to be noted that iron deficiency does not necessarily manifest itself with anaemia. In Lindi, Tanzania, studies have shown that fifty percent of nonanaemic pre-school children are iron deficient (Tatala, S., 1998).

In the diagnosis of iron deficiency, measurement of *serum iron concentration* and *total iron binding capacity* are widely used, however assay of *serum ferritin concentration* is a much more sensitive and reliable means of demonstrating this disorder (Fairbanks, V.F., and Klee, G.G, 1987).

1.5.1 Ferritin

This is the major cellular storage protein for iron in the human body (Theil, E. C., 1987). It is mainly localised in the cytoplasm of cells derived from the reticuloendothelial system, as well as in liver, spleen and bone marrow (Jacobs & Worwood, 1975). Ferritin consists of two components, the apoferritin (protein moiety) and the iron core (Joshi and Clauberg, 1988) (Fe[III]-hydroxyphosphate-micelle). Apoferritin is a globular protein built up of 24 subunits that can bind up to 4500 iron atoms (Jacobs, Path & Worwood 1975). In the human body, ferritin exists in several isoforms that

can be differentiated by their iso electric points and immunological reactivity (Halliday & Powell , 1979; Hazard & Al 1977).

There is a direct correlation between the concentration of ferritin in the serum to that stored in the tissue. This was confirmed in studies with patients who had developed iron deficiency or iron overload (Water & AL, (1973; Cook & Al, 1974; Addison & Al, 1972). From these and other results it was found that a ferritin concentration of 1ng/ml in the serum is equivalent to about 8 mg of stored iron (Walters & Al, 1973). This implies that the determination of ferritin in the serum/plasma is a helpful clinical, non-invasive indicator of the iron stores of individuals Brink & Al, 1982; Beck & Al, 1981; Skikne & Al, 1981).

1.6 Blood group

According to the ABO system, every person can be classified into one of the four major blood groups. These blood groups designated as A, B, AB and O are determined by the presence of specific proteins in the blood plasma and the blood cells. The proteins in plasma are known as agglutinins or antibodies, and the proteins on red cells as antigens or agglutinogens.

Individuals of blood group A have A antigens in their blood cells and Anti - B antibodies in the plasma. Individuals of blood group B have B antigens in their blood cells and Anti-A antibodies in their plasma. Those of group AB have both A and B antigens in their cells and no antibodies in their plasma. Finally those of blood group O have no antigen in their cells but have both Anti-A and anti-B antibodies in their plasma.

Blood group antigens are chemical moieties on the red blood cell membrane. It is not known if blood group antigens have a biological function. Interactions of some parasites and bacteria with human cells have been shown to depend on the presence of certain blood group antigens. *Plasmodium vivax* malarial parasites only enter human red blood cells when certain blood group proteins are present on the red blood cells. Some *Escherichia coli* strans will only attach to the epithelial cells of the urinary tract if certain specific blood group antigens are present in the epithelial cells, so are the *Pseudomonas aeruginosa* (Steuer, Hofstadter et al 1995) and *Candida albicans* (Cameron, B. J., Douglas, L. J., 1996). Statistical correlations with ABO blood groups and disease are not new (Garatty, G., 1995). In fact there are reports on association of blood group antigens with various infections (Hilton, E. 1995). Studies carried in India have shown that people with blood group AB are less prone to malarial

infection (Sing, Shukla, et Al 1995). In another studies carried out in Germany, it was observed that people with blood group A are predisposed to pseudomonal infection of the ear (Steuer, Hofstadter et al 1995). In the UK studies have demonstrated that blood group antigens can act as epithelial cell receptors for *Candida albicans* (Cameron, B. J., Douglas, L. J., 1996). In France, studies of the combined effect of various histo-blood group genetic systems seem to have some relation with breathing difficulties, possibly with reference to susceptibility to infectious agents (Kauffmann, Frette et al 1996). In Zimbabwe, it has been demonstrated that intensity, annual incidence of *Schistosoma haematobium* infection and related organ pathology is significantly higher in children of blood group 'A' and lowest among blood group 'O' children (Ndamba, J., Gomo, E., Nyazena, N., Makaza, N., Kaondera, K. C., 1997).

It is of interest to study if there is any correlation between ABO blood group and parasitic infection.

Mauritius – History, Geography and Poverty

Mauritius, one of the islands in the Mascarenes groups, occupies a choice section of the Indian Ocean. It is situated just north of the Tropic of Capricorn, in latitude 20° south and longitude 57°35' east. The country

includes Rodrigues, an island 560 km to its north-east and other scattered islands such as Agalega and Cargados Carajos (St Brandon).

The island is approximately 855 kms off the East Coast of Madagascar, Africa being the nearest continent, with Mombassa some 1800 kms away. Mauritius is a volcanic island about 10 million years old, only 1865 square kilometres in area. The central plateau reaches 800 metres in altitude with the highest peak, Le Piton de la Rivière Noire, reaching 828 metres. The coastline of 330 kms is almost entirely surrounded by one of the largest unbroken coral reefs in the world. The mountains of the island are not very high but many of them are unusual in shape. It is a tropical Island, with slightly varied climates at different seasons and different places.

Mauritius was known to the early Arab traders as it can be found marked on their maps, but the first visitors from Europe were the Portuguese, who landed in 1510. They used the island as a victualling stop on the way to Goa and Malacca but did not settle. The Dutch, who arrived in 1598 and named the island Mauritius after Prince Maurice of Nassau, made the first attempt at colonisation. They disembarked with a mixed group of Dutch and Indonesians; some of the latter were slaves. They introduced sugar, Malagasy slaves and a herd of Javanese deer. But they were also said to be responsible for the disappearance of the magnificent ebony forests and

the extinction of the famous dodo. They eventually abandoned their settlements in 1710. When they abandoned the Island, the runaway slaves and those marooned in the jungle were left behind (Noel, 1991, Nwulia, 1981).

The French occupied the island between 1715 and 1810, renamed it Isle de France, and many place names are reminders of this period. Mahe de Labourdonnais, who took over as governor in 1735, rebuilt Port Louis and opened the first sugar mill.

The occupation by the French was, followed by slaves brought from the nearby Island of Madagascar. The slave population also included about 6,000 Indians (Mookherji, 1958). By 1803, the total slave population rose to 60,000. In 1810, with the British take-over, the name reverted to Mauritius.

In 1835 the abolition of Slavery Act took effect in Mauritius, when no less than 60,000 emancipated slaves left the sugar estates en masse, thereby exposing the sugar industry to its greatest crisis as a result of shortage of manpower (Hazareesing, 1975). India with its large reservoir of untapped labour came to the rescue. Over 40,000 Indian indentured labourers landed in Mauritius between January 1843 and March 1844 and by the

1860 this number shot up to the colossal figure of 303,000 (Mookherji, 1958).

The abolition of slavery led to the importation of Chinese and Indian indentured labourers, who were followed by traders of their own nationalities.

The movement of people, from the Dutch occupation of the Island to British colonisation, produced the ethnic and cultural pluralism and a model in race relations, which is unique to Mauritius.

In the 50s and 60s many countries gained their independence by wars of liberation and some by concession from the parent country. Mauritius falls in the latter category. Mauritius gained independence from Britain on 12 March 1968, and since then, has been an independent sovereign nation within the British Commonwealth. On 12 March 1992, Mauritius became a Republic.

Independent Mauritius was born in a situation of conflict and disagreement within its own population. Post-colonial Mauritius could have been a recipe of disaster, as independent Mauritius had to face a wide range of social and economic problems. The country had to come to grips with a

stagnating monocrop economy, heavy unemployment, major balance of payments difficulties and a rapidly expanding population. However within less than two decades, Mauritius succeeded in achieving economic and social stability.

By moving rapidly from a struggling monocrop agricultural economy to a highly industrialised one, Mauritius has been able to solve its heavy unemployment problem and balance of payments difficulties (Bheenick, R., Hanoomanjee, E., 1988).

By now, Mauritius has had a remarkable record of nearly 30 years of continuous growth at about 5.6% per year. It has established a broad programme of social security and welfare reaching far into most segments of the populations, and has consolidated a stable democratic framework. (lettre de la Commission européene à Maurice- juillet 1998). Apart from diversified rapid industrialisation the economy has been further strengthened by the expansion of tourism.

Developments in Mauritius have brought unquestionable benefits. While unemployment at the national level has fallen to negligible levels, several thousand workers from China and India are currently employed in the textile industry and in agriculture and telecommunications.

A number of new schools and a few hospitals have been built and equipped. The housing situation has been improved with the construction of houses and apartments for sale to middle and low-income groups. Infrastructural improvements have also been made across the island. Overall, it may be said that these developments have resulted in higher standards of living for a majority of Mauritians (World Bank Country Study – Mauritius Managing Success).

Unfortunately, however, there is another side to this picture. The attentive observer knows that significant numbers of Mauritian are only marginally touched by all this progress. They are being left behind. They are not participators or the beneficiaries; they are the unhappy spectators and often the helpless victims of "progress". They do not fully participate in its process and in its fruits. They are almost "excluded", and so are their children. Many of them have insecure home backgrounds. High proportions of them are illiterate. Those who are not unemployed have either low income or unstable jobs. They generally have poor lodgings and living conditions and are victims of several forms of discrimination. Some such people may be found in most regions of the country but there are concentrations of them in several areas.

Fig. 13. Pockets of Poverty persisting throughout the island. (Top-Anagglumeration at Roche Bois, Middle-an individual residence at Moka, Bottom-a family at La Pipe Tea village).

The child population of the Republic of Mauritius was numbered at 374,158 as at June 1997, with 701 orphans and 2133 cases of reported children in difficult circumstances. Again in the year 1997, 114 cases of abandoned children, 9 case of child labour, 12 cases of child mendicity, and 176 case of neglected children were registered at the Government child Development unit (A Statistical Profile on Children in the Republic of Mauritius, Ministry of Women, Family Welfare and Child Development, June 1998).

There are indicators available as government other publications, which are handy. These indicators include a list of poorest wards ranked in terms of the Relative Deprivation Index developed by the Central Statistical Office (Central Statistical Office, 1994). This is an aggregate index of village council areas. There is also a list of worst performing schools at the Certificate of Primary Education for the three preceding years.

Examination of the above two lists and on the assumption that there is a correlation between levels of economic and social poverty and school performance, as many as 402 poverty stricken areas are identifiable.

Presence of poverty in Mauritius is fully recognised. It is also universally accepted that if left unattended, poverty can give rise to increased crime

and social problems, which would undermine the progress that Mauritius has achieved. Mauritius relies on its social services to redress the problem.

Indeed, Mauritius has a long history of social services dating as far back as the 18th Century. In 1810, Mauritius was taken over by the British who are known to have a long history of social services (Fraser, D., 1973). Existing social services in developing countries, like Mauritius as it is very commonly described, is a 'relic' of the English poor Law transferred to colonies (Jones, H., 1990). Provision for support is made under the Social Aid act 1983. The Social Aid Act operates under the Social Aid Division of the Ministry of Social Security. It devises and implements policies and programmes designed the needs of underprivileged and vulnerable groups of Mauritian citizens. However the aid is restricted to the elderly, the widows, the orphans, and the disabled.

Also there is more to it than the direct effect of income. Certainly, income has material effects, but with poverty, there is a complex of environmental and psychological pressure involved.

While it is true that most of the children from the impoverished areas come to school without their exercise books, pens and pencils, which they

cannot afford, it is also true that poor health increases suffering and reduces alertness and ability to cope with and enjoy life. Also due to poor health, the child has to miss school often. This explains why school performance has been taken as a valid indicator of poverty.

At national level, poor health shackles human capital and undermines socioeconomic environments conducive to entrepreneurial activities. Therefore if the parents of the children are not in good health, morbidity brings the family income further down thereby causing a vicious circle.

As a research ecologist taking all the above in mind, it should prove useful to consider the reciprocal relationship between the child and his total environment. In the search for the causes of disease, it is not sufficient merely to identify the specific agent of a disease, but it is always desirable to identify the influence of environmental factors on the interaction between the child and the specific agent (the parasite and the deficient haematological parameter). Similarly, a specific nutritional deficiency should not be viewed merely as a discrete metabolic defect but it should be seen in the context of the food habits, the level of education and income of the population etc.

It is hoped that from this ecological approach, one can derive a rational basis for the control of disease within the study population. Therefore the relevance in carrying studies to determine the impoverished areas, and the scientific basis of parasitic infections and iron deficiency.

It is hoped that from this ecological approach, interested parties can derive a rational basis for the control of disease within the population and in more complex situations promote welfare services.

It is my opinion therefore, that this study is very timely.

CHAPTER 2

MATERIALS AND METHODS

2.1 **Site selection and site characterisation**.

Mauritius has been hailed an economic miracle by many observers. Its economy successfully overcame the numerous problems of only a few years before. Despite all these, it is reported that pockets of poverty persist. It seems that the rapid transformation of the economy has had a direct and negative impact on the lives of many vulnerable groups. As such, the quality of life of some people has grossly deteriorated. Since there are no published documents on poverty in Mauritius, study on poverty with a view to identifying impoverished regions of Mauritius became a prerequisite of this research.

Poverty is generally characterised by the inability of individuals, households, or entire communities to command sufficient resources to satisfy their basic needs. Thus poverty can be conceptualised as:

- The inability to attain a minimum standard of living (World Bank, 1990).
- The lack of resources with which to attain the type of diet or life-style that is socially acceptable.

For this research the study impacts of changing development framework on socio-economic changes was particularly important. Therefore, vulnerability (having secure and sustainable access to essential

commodities, services, and other conditions for an acceptable life) was found to be most suitable. As such, consideration was particularly given to the social impact of poverty, the assets of the poor and the activities that they perform in order to generate a livelihood.

Based on the World Bank's definition of Poverty (World Bank (1990)) which is "the inability to attain a minimal standard of living" measured in terms of basic consumption needs or income required to satisfy them, a poverty line may be drawn which separates the poor from the 'non-poor'. This can be achieved by calculating 'the expenditure necessary to buy a minimum standard of nutrition and other necessities. It was hoped that by using poverty lines identification of regions of poverty for the purpose of this research could be identified. Poverty lines are used to provide a profile of poverty. Every group falling below the line is considered as poor. For example, an individual drawing a salary of Rs. (Mauritian rupees) 3499 should be considered as poor where as those earning an extra one rupee (household earning 3500), are considered not poor.

Mauritius so far has no official absolute poverty line. Therefore those households which consume less than fifty percent of the mean consumption level as measured by the 1992 budget survey, are considered as poor and those consuming less than twenty five percent as

ultra-poor for this study. In monetary terms, the two relative poverty lines were set at Rs. 3500 and 1750 respectively.

In terms of the poverty profile, it was observed that large households with many dependants are likely to be poor. Children in some regions are far more likely to be poor, compared to the other regions. Poverty rates among female-headed households were much higher than for male-headed households. Poverty and unemployment are closely linked. The poor, more specially, the ultra-poor, are more likely to be squatters living in more crowded conditions. They are less likely to have access to water inside their homes, a modern sanitation system, and electricity.

Since poverty in Mauritius is confined to a minority, a simple random sample of all areas of Mauritius would have been a wasteful methodology. The sample frame was therefore constructed using a combination of the two spatially referred indicators. These were:

- The poorest areas ranked in terms of the relative deprivation index (RDI) developed by the Central Statistical Office (Central Statistical Office, 1994). However, since the index was an aggregate index of

Village council areas, it was felt that some poor areas were being left out. This explains why another two parameters (see below) were included.

- On the assumption that there is a correlation between levels of economic and social poverty and school performance the locality of the 85 worst performing schools at the Certificate of Primary Education during the three preceding years (see appendix) were also added to the list obtained from step 1 above.

When the above was completed, a list of the poorest areas of Mauritius was generated. The sample frame finally included 402 poorest enumeration areas. By a self-weighing multistage cluster sampling methodology, a total of 100 Enumerator Areas were selected. This constituted the Primary Sampling Unit. The number of household in the selected regions was made available from the Central Statistical Office, by one of the two volunteers. Statistical reports are otherwise not for restricted circulation.

Each and every selected enumerator areas (EA) was then visited. A random starting point was selected in the concerned Enumerator Area.

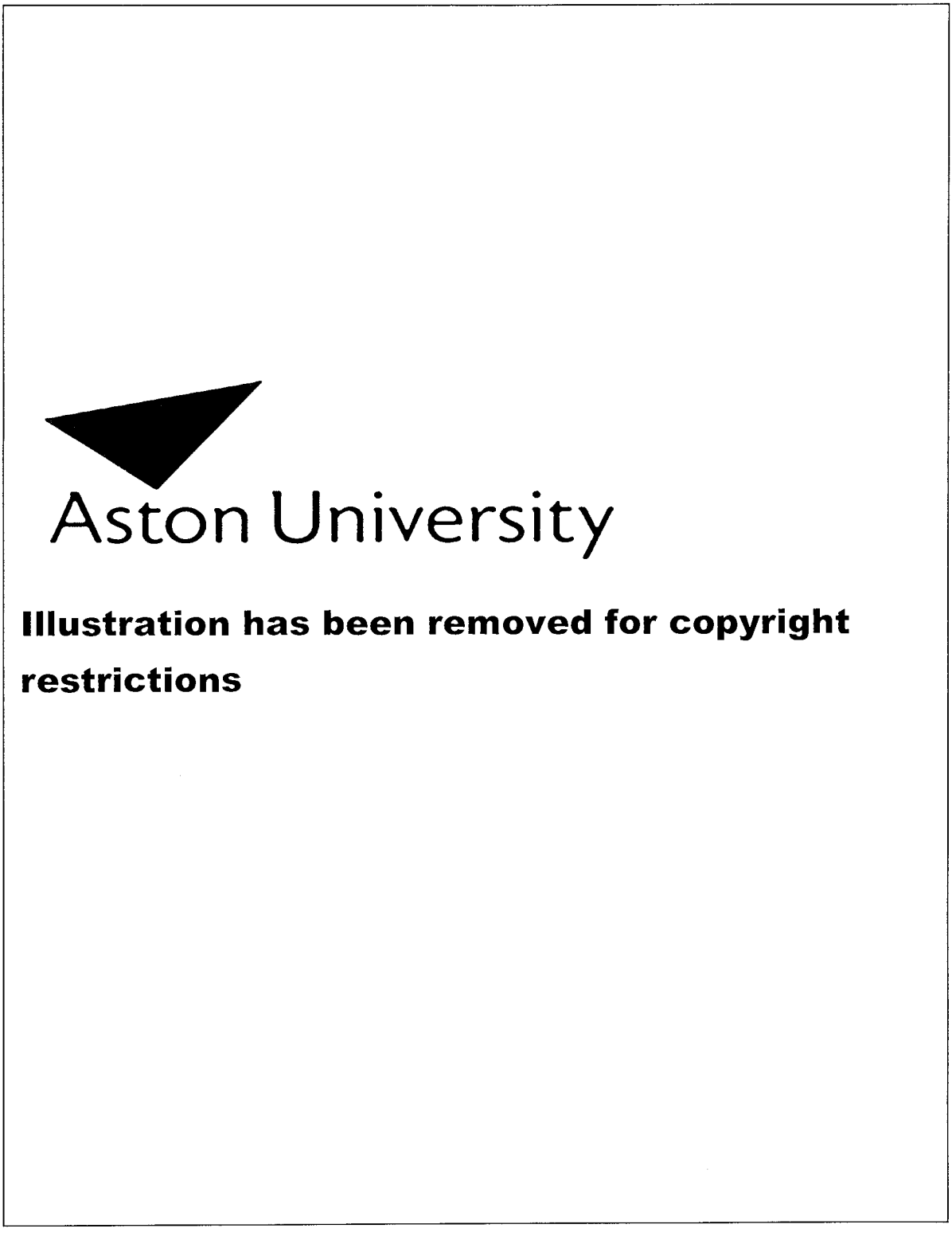
Every 6th household was surveyed. This gave a secondary sampling unit (SSU). With the help of a volunteer from the Central Statistical Office and a volunteer health motivator from to the Ministry of Health, information was obtained by direct interview based on a questionnaire which included employment status and occupational structure, assets, food and non-food spending.

From the information obtained, the 20 poorest areas were identified.

Out of the twenty poverty areas, four were selected as test areas by drawing of lots. These locations which are Pointe Brocus (Petit Bel Air), Mare Chicose, La Pipe, and Mare D'Australia are shown (encircled) in the physical map of Mauritius below.

La Pipe has been earmarked as the suitable site for the construction of the Midland dam as such the houses at the La Pipe Tea Village have now been demolished (so as to prevent new squatters), and the inhabitants re-lodged in new houses with modern facilities at Seiziemme Mille, which is next to the town of Curepipe.

Fig 14. Sites Location. (Sites encircled on physical map of Mauritius)



2.1.1 Pointe Brocus - Petit Bel Air

This is a region near Mahebourg. The site selected is mostly, inhabited by labourers with irregular employment and poor salaries. They live in shacks and the bread earners spend most of their time drinking rum. Although some of these people do own the plot of land they are on, they are unable to build proper houses. Their families are normally large and they do not earn enough money for their basic needs. They do not normally pay attention to their children's education. To make matters worse, the water supply to this village is irregular with regular interruptions.

2.1.2 Mare Chicose

This is a small village situated far away from most of the developed areas. The bread earners are mostly labourers. The standard of living of most of the families is very low. There is no school in the village and parents have to find extra money to send their children/ wards to school.

2.1.3 La Pipe

This place has been earmarked for the construction of the midland dam. As such the houses have been demolished and the inhabitants re-lodged.

At the time of survey, La Pipe was a small and serene village located in the bushes and tea plantations. Originally, the two-roomed houses were built to house tea employees. However, as the tea workers abandoned those houses for houses in better - developed areas, Rodriguan 'immigrants' took illegal possession of those houses. They lived there even in the absence of vital commodities like water and electricity. In those days, the population of La Pipe was composed of about seventy families, all squatters. The females worked on tea plantation with meagre salaries while most of the males worked as fishermen on big vessels, living away from their families for months. In short, the people of La Pipe lived a hard life and had to struggle hard to keep them alive.

As regards to sanitation, in La Pipe, the use of latrine pits was very common. The village was not even connected to a regular water supply. Water tanks (cistern) supply them with water once a week, otherwise the inhabitants had to rely on river water.

2.1.4 **Mare D'Australia**

Most of the bread earners in this village work in the sugar industry, drawing very low salaries. There is no proper infrastructure and the standard of living is very low. Like Mare Chicose, there is no school in the village and the nearest school is at Brisee Verdiere.

2.2 Study population and specimen collection.

As the number of children in those villages was very low, no random selection was required. Each and every child living in the selected villages was recruited.

Along with the two volunteers (Statistical Officer and Health Motivator) the parents were visited individually. The type of studies being undertaken was explained to them. They were encouraged to let their children participate in the study. When they agreed, they were asked to sign a consent form, on which they gave their consent to let us visit the children in their class and collect blood samples by venepuncture. In the consent form they also had to indicate the school and the class in which their

children/wards were. They were then given sterile containers, to enable them to collect stool and urine specimens on six different occasions for each child. The stool and urine specimens were collected, as far as possible, in batches of ten per visit, and taken to the laboratory for thorough examination. Small numbers of specimens were collected at each visit so as to give time to examine the fresh specimens thoroughly.

When the sixth specimens of urine and faeces were collected from each child and examined, arrangements were made for blood collection. This was done on the school premises, and for this a qualified nursing officer experienced at drawing blood from children's veins was recruited on a voluntary basis from the SSR Centre for Medical Research.

Stools/faeces collected were examined for parasites by direct smears as well as by recognised concentration methods.

Urine specimens were centrifuged, and the deposit examined for the presence of *Bilharzia ova*, the only known urinary parasite in Mauritius.

Blood collected was use for determination of the following parameters:

- presence/absence of Malaria and other blood parasite,
- haemoglobin concentration,

- packed cell volume,
- white blood cell count,
- blood group,
- plasma ferritin.

2.3 **Methodology used**

Correct identification of parasites and accurate evaluation of the different, related biological parameters require an intelligent use of laboratory procedures. Investigations, therefore, for this research, were carried using recognised laboratory techniques as described hereunder.

2.3.1 Identification of parasites

The identification of parasitic organisms is dependent on morphologic criteria, these criteria are, in turn, dependent on correct specimen collection and where applicable adequate fixation. Improperly collected specimens may result in failure to find the organisms or in their misidentification.

2.3.2 **Collection of specimens for intestinal parasites**

The ability to detect and identify intestinal parasites, particularly protozoa, is directly related to the quality of the specimen. Certain medications may prevent the detection of intestinal protozoa; these include mineral oil, bismuth, nonabsorbable antidiarrhoeal preparations, antimalarials, and some antibiotics. Care was taken to avoid collection of specimens after the start of medication. Specimens were therefore collected before the start of any medication or several weeks after. Faeces were collected in clean, widemouth containers, and instructions were given so as not to contaminate faeces with water or anything else.

Each specimen collected clearly specified the child's name, age, and residence so as to avoid mixing the specimens up. The six specimens from each child were collected at six different intervals within a period of fourteen days. All the specimens were freshly passed ones and examinations of specimens were started within thirty minutes after collection and completed the same day.

Along with stools, urine specimens were also collected from the same child on the same day. On any day the child did not pass a stool, the urine

specimen was discarded to keep consistency. Microscopic examination of the deposits of the urine specimens was carried out after centrifugation.

On or after the sixteenth day after collection of the first stool specimen, blood was also collected by venepuncture in tubes containing sodium EDTA anti coagulant. Since blood was collected by venepuncture, and since parental consent was also required, only one specimen per child could be collected. The blood samples were tested for haematological parameters in the laboratory where the plasma was also separated and kept at -20^{0} C for the subsequent determination of Plasma ferritin levels.

2.3.3 **Diagnostic procedures for the examination of faeces.**

As stated earlier, the identification of intestinal protozoa and helminth eggs is based on recognition of specific morphological characteristics; these studies require a good binocular microscope with a good light source.

A combination of techniques was used to yield a more accurate number of positive specimens. These techniques included:

2.3.3.1 Direct smears

The direct smear was prepared by mixing a small amount of faeces (approximately 2 mg) with a drop of physiological saline; this mixture provided a uniform suspension under a coverslip. The entire field of each specimen was systematically examined using a low-power objective (10X) and low light intensity. All suspect objects were then examined under high-dry power (43X).

Direct smears are used primarily to detect motile trophozoite stages of the protozoa. Protozoan organisms in a saline preparation usually appear as refractile objects. If suspect objects are seen on high dry power one should allow at least 15 seconds to detect motility of slow moving protozoa. Helminths eggs or larvae and protozoan cysts can all be seen on wet films, although, with the exception of protozoan cysts, these are more often detected after faecal concentration procedures.

After the wet preparation, another wet smear was prepared from each stool specimen using a weak iodine solution instead of physiological saline. With this preparation, protozoan cysts if correctly stained contain

yellow-gold cytoplasm, brown glycogen material, and paler refractile nuclei.

2.3.3.1.1. Isotonic/physiological saline

Isotonic/physiological saline was prepared by dissolving 9g of sodium chloride to make a litre of solution.

2.3.3.2 Concentration methods

Faecal concentration procedures should necessarily be included for complete examination for parasites; these procedures allow the detection of small numbers helminth eggs that may be missed using only the direct mount. Helminth eggs could always be identified when recovered by the concentration method.

Concentration methods that were used for this study were both flotation and sedimentation techniques (often referred to as saline concentration). The flotation technique permits the separation of parasite eggs through the use of a liquid with a high specific gravity. The parasitic elements are

recovered in the surface film, whereas the debris settles down in the bottom of the tube. Since some helminth eggs (dense eggs like unfertilised *Ascaris* eggs) do not concentrate well with the flotation method, sedimentation methods were also used.

The sedimentation procedure using a centrifuge allowed the recovery of all parasites present. It was the easiest to perform and the least subject to technical error.

2.3.3.2.1 Zinc Sulphate flotation methods

Zinc sulphate solution with specific gravity of 1.18 were prepared by dissolving 330gram of dry crystals of zinc sulphate in 670 ml of distilled water. The specific gravity was adjusted by the addition of either distilled water or zinc sulphate crystal as required.

- A faecal suspension of half a teaspoonful of faeces in 10-15 ml of tap water was prepared. This suspension was then filtered through two layers of gauze into a small tube. The latter was then filled with tap water and centrifuged for one minute at 2,300 rpm.

- After decanting the supernatant, the deposit was re-suspended in water and centrifuged again.
- Prepared zinc sulphate solution (s.g. 1.18) was then added to the deposit and the mixture centrifuged again.
- The surface film was then transferred to a slide and the latter examined for the presence of parasites.

2.3.3.2.2 Formol-Ether sedimentation techniques

- A faecal suspension of half a teaspoonful of faeces was prepared in10 ml of formalin.
- After allowing the suspension to stand for thirty minutes, the preparation was filtered through two layers of gauze into a centrifuge tube.
- After addition of physiological saline to the top of the tube, it was centrifuged for two minutes at 1,500 rpm.
- After removing the supernatant the sediment was resuspended with physiological saline, and centrifuged at 1,500 rpm for two minutes.
- The preparation was decanted again and the sediment resuspended in 10% formalin.

- About 3 ml of ether was then added to the suspension above in a tube, shaken vigorously for thirty seconds and then centrifuged for 3 minutes at 1,500 rpm.
- At this stage four distinct layers were observed: a small amount of sediment in the bottom of the tube containing the parasites; a layer of clear formalin; a plug of faecal debris on top of the formalin layer; and a layer of ether at the top.
- The plug of debris was loosened by means of a wooden applicator, and all fluids decanted.
- The sediment was then transferred to slides and examined for the presence of parasites.

2.3.3.3 Permanent stained smears

The detection and correct identification of intestinal protozoa are often dependent on the examination of a permanent stained smear. For this work all specimens whether negative of positive, were stained permanently. This is because the smaller protozoan organisms are often seen on the stained smear and missed with direct smear methods. Besides it is believed that most identification should be considered tentative until confirmed by a permanent stained slide.

Permanent stained slides were prepared as follows:

- Small quantities of stools were smeared onto slides by means of a wooden applicator (applicator stick).
- The slides were then immediately immersed in Schaudinn's fixative for thirty minutes, transferred to a jar containing Trichrome stain, and allowed to stand for twenty minutes. After which the slides were de-stained for two seconds using 90% acid alcohol.
- After de-staining, the slides were quickly rinsed in 100% alcohol, and transferred to another jar containing 100% alcohol for dehydration.

(In correctly stained slides, backgrounds are green and the cytoplasms of protozoa, blue-green to purple, while the nuclei are red.)

2.3.3.3.1 Schaudinn's Fixative

This was prepared by adding 300 ml of 95 % ethyl alcohol and 100 ml of glacial acetic acid to 600ml of saturated solution of mercuric chloride ($HgCl_2$); acetic acid being added just before use.

2.3.3.3.2 Trichrome stain

Formula

Chromotrope 2 R	0.6 g
Light green SF	0.3 g
Phosphotungstic acid	0.7 g
Glacial acetic acid	1.0 ml
Distilled water	100 ml.

Preparation

Acetic acid was added to the dry components, and the mixture was allowed to stand for 30 minutes. 100 ml of distilled water was then added.

Routine

Fresh faecal smears were placed in 70% ethanol for 5 minutes. Then transferred to a mixture containing 70% ethanol and D'Antoni's iodine for 2-5 minutes. Slides were removed and placed in two changes of 70 % ethanol one for 5 minutes and the other for 3 minutes.

The slides were then placed in trichrome stain solution for 10 minutes and differentiated by placing in 90% acid-alcohol (90% ethanol containing 1% acetic acid), for up to a maximum of 3 seconds.

Diped once in 100% ethanol.

Placed in two changes of 100% ethanol for 2-5 minutes each.

Mounted if necessary.

2.3.3.3 D'Antoni's Iodine

Formula

Distilled water	100 ml
Potassium iodide (KI)	1 g
Powdered iodine crystals	1.5 g

Preparation

- Dissolve potassium iodide in water.
- Add iodine crystals.
- Decant into brown-glass bottles for use.

2.3.4 Detection of Malaria, the Blood Parasite

Malaria is one of the few acute parasitic infections that can be life threatening. The definitive diagnosis is based on the demonstration of the

parasites in the blood. Two types of blood films are used. The thick film allows the examination of a larger amount of blood, while the thin film allows speciation of the parasite (Cheesborough, M., 1987) (Finegold, S. M., & Baron, E. J., 1986).

For this study, blood films were prepared out of the sample of blood collected in tubes containing sodium EDTA. Approximately 200 microscopic fields were examined before a film was counted as negative.

For thick films, three small drops of blood were placed on an alcohol-cleaned slide. With the corner of another slide, and using a circular motion the drops were mixed and spread over an area of about 2cm diameter. Formation of fibrin strands, which may obscure the parasites, after staining, was prevented, by stirring the blood drops for another 30 seconds. The films were allowed to air dry at room temperature, and then stained.

For thin films, one drop of blood placed on glass slides was spread into a thin film by the use of the edge of another slide. Thin films were used for specific parasite identification. After air drying, the films were stained.

The following procedures were respected in the preparation of blood films for malaria:

2.3.4.1 Preparation of Thin Blood Films

- Perfectly clean and grease free slides were used
- One drop of blood about the size of a pinhead was collected/placed on the end of the slide.
- The spreader slide was touched to the drop of blood so as to make the latter run along the line of contact between the two slides.
- The spreader slide was gently but firmly pushed along the surface of the horizontal slide to the far end.
- Care was also taken to taper off the blood at the distal end of the horizontal slide.

2.3.4.2 Preparation of Thick Blood Films

- On a clean slide a drop of blood three times as large as those of thin blood films, was placed.
- The corner of a second slide was then applied to the drop of blood, and with a circular motion, the blood was spread to cover an area of about 2 cm. in diameter.
- The slide was then placed in a horizontal position for drying.

2.3.4.3 Staining of Blood Films

For the accurate identification of blood parasite, proficiency in staining is important. As a general rule, blood films should be stained as soon as possible, because prolonged storage results in stain retention.

The Russian protozoologist Romnovsky, first used a combination of dyes that differentially stain the blood elements and protozoa (Viqar Zaman 1995). Romanovsky stain is almost invariably used in the detection and identification of protozoan parasites in blood and tissues, although more than often, various modifications of the stains are used. However the essential components of all these stains are methylene blue, azure A or azure B, and eosin. When blood films are stained with a Romanovsky dye, the various elements are coloured as follow:

- Nuclei of leucocytes — Purple
- Nuclei of protozoa — Red
- Cytoplasm white blood cells and protozoa — Blue
- Eosinophil granules — Purple
- Erythrocytes — Pink /Bluish

This explains its suitability. For this work, Giemsa's stain which is an alcohol based Romanosky stain requiring dilution in pH 7.1 – 7.2 buffered water before use (Cheesborough, M., 1987) was used.

A stock solution of stain was prepared by dissolving the dyes in pure methanol. The alcoholic solutions of the dyes were diluted with distilled water before use. So as to obtain the correct Romanovsky effect, the water used to dilute the stain, was neutral or slightly alkaline (pH 7 to 7.2). For this work buffered water was used using buffer tablets obtained commercially (supplied by BDH GURR) (also available in the market is a concentrated buffer solution which on dilution gives the correct working solution and this is supplied by BAKER).

2.3.4.3.1 Treatment of Films Before Staining

Thin films were fixed in methanol for two minutes before transferring to a tray containing stains.

Thick films were placed directly into the stain. This helps in the lysing of red blood cells. In this way, only white blood cells, platelets, and protozoa took up stains.

2.3.4.4 Preparation of Stock Giemsa's Stain

To 7.5 gram of Giemsa's stain powder in a mortar, 250 ml of glycerol were added. The mixture was ground with a pestle until a paste was obtained. 750 ml of pure methanol was then added to the mixture and stirred. The preparation was then poured into a dark bottle and incubated at 37^0 C for 24 hours.

2.3.4.5 Preparation of Working Solution

A working solution of the Giemsa's stain was obtained by diluting one part of the stock solution with 10 volumes of buffered distilled water (pH 7.2).

2.3.4.6 Microscopic Examination of Stained Blood Films

Dried films were mounted in green euparal and examined with an oil immersion objective. Mounting of films protects the latter from dust and

from deterioration. Green euparal is an excellent mountant as it has a low refractive index, dries quickly and does not shrink.

2.3.5 Practice of Haematology

The increasing demand placed on the haematology laboratory necessitated the development of sophisticated automated equipment that can accurately determine the different haematological parameters. Although for this work a Coulter counter was used, a brief description of the common haematological techniques, with details of the Coulter counter is presented here.

2.3.5.1 Haemocytometry

Blood cells like red cells, white cells and platelets are enumerated in routine practice in clinical laboratories. Indeed for the purpose of this study it was thought appropriate wherever possible to enumerate the leucocytes. Electronic cell counters are always preferred as they avoid human error.

2.3.5.2 Coulter Counter

This equipment is so designed that by means of a mercury manometer, a specific volume containing blood particles in an electrolyte (0.9% sodium chloride) is forced through an aperture of specific dimensions. An aperture current passes between an electrode within the aperture tube and another outside the aperture. As the blood particle passes through the aperture, it lowers electrolytic conductivity between the two electrodes, producing an impulse; the magnitude of which is proportional to the volume of the particle. The voltage pulses are fed into a threshold circuit that discriminates between pulses of different sizes, generating counting impulses for those particles that exceed the threshold level alone and thus counting the number of particles of threshold size in passage.

For the counting of leucocytes, cetrimide counting fluid, available commercially supplied by Coulter Electronics Inc, was used.

The apparatus used for this work was the 'Coulter S', which provides seven parameters including the estimation of haemoglobin concentration.

When using the 'Coulter S', blood sample is presented to the aspirator tip, and the sample button is pressed. In so doing, exactly 1ml of blood is

aspirated, 1.6μl of which is segmented off by the blood sampling valve for other counts while 42.9 μl is segmented off for a leucocyte count and haemoglobin determination. Each volume of blood is directed to its respective analytical bath along with 10 ml of isotonic saline. Lysing agent supplied by the manufacture of Coulter counter is added to the leukocyte bath to lyse the erythrocytes before haemoglobin measurement.

2.3.6 ABO Grouping of Blood

Establishing the ABO group of an individual usually involves so-called forward and reverse grouping. The sera for forward grouping are of human origin, usually collected from individuals whose natural antibodies have been stimulated to high titres. Anti–A (from group B individuala), anti–B (from group A individuals), and anti–A,B (from group O individuals) are normally used in forward grouping. Red cells for reverse grouping are also of human origin, from group A and B individuals.

2.3.6.1 ABO Grouping Technique

On a tile, one group of anti–A, one drop of anti–B, and one drop of anti–A,B are placed at different spots so are two drops (one drop at one spot each) of serum from the individual under test.

1 drop of washed suspension of 10% of red cells from the individual under test is added to the anti–A, anti–B and anti–A,B.

To the two separate drops of serum from the individual under test, one drop of known A and B cells are added respectively.

Each separate cell-serum mixtures are mixed thoroughly using wooden applicator and the tile rotated until agglutinations appear.

For the purpose of research, the tube technique was used additionally as described below.

In a test tube rack five 10 x 75 mm test tube labelled as follows: anti A, anti-B, anti A,B, A-cells, B-cells, are placed in a test tube rack. To tube labelled anti-A, one drop of anti–A grouping serum is added. Similarly, to tube labelled anti B, one drop of anti–B grouping serum is added and to

tube labelled anti– A,B, one drop of anti–A ,B grouping serum is added. To each of the tubes labelled A-cells, and B-cells, one drop of serum from the individual under test is added.

One drop of washed suspension of red cell in saline from individual under test is added to tube labelled anti-A, to tube labelled anti-B and also to tube labelled anti-A,B.

To tube labelled A-cells one drop of washed A-cells suspension is added, so is one drop of B-cells added to tube labelled B-cells.

Contents of all the tubes are mixed thoroughly and the tubes incubated at room temperature for 90 minutes.

Agglutinations (sometimes lysis depending on the presence agglutinin in serum) are noted. Interpretation is as per the table below.

2.3.6.1.1 Interpretation of results

Results of ABO grouping are interpreted as per table1below:

Table 1. Interpretation of ABO grouping results.

Anti–A	Anti–B	Anti–A,B	A–cells	B–cells	Group
-	-	-	+	+	O
+	-	+	-	+	A
-	+	+	+	-	B
+	+	+	-	-	AB

2.3.6.2 Technique for Rhesus 'D' Typing

Like blood grouping, Rh 'D' typing also may be performed on a slide or in tubes. In the slide technique, one drop of anti-D is placed along with one drop of albumin on a slide. To this mixture the test sample is added and the whole is mixed thoroughly. If agglutination appear, the test sample is said to be Rhesus 'D' Positive.

2.3.7 Plasma/Serum ferritin

The quantity of iron in the storage compartment is reflected in plasma ferritin concentration. The plasma ferritin concentration declines very early in the development of iron deficiency (Fairbanks, V.F., and Klee, G, G., (1987). Thus, measurement of serum ferritin concentration is a very sensitive indicator of an iron deficiency.

Serum ferritin assay may be performed by radioimmunoassay (RIA) immunoradiometric assay (IRMA), or enzyme-linked immunosorbent assay (ELISA). For the purpose of this study, serum ferritin assay was performed with commercially available kits supplied by bioMerieux.

Also, because considerable variation in reference values are normally observed with different methods, reference values for children in Mauritius using the ELISA technique was determined using control samples of blood.

The test was performed by standard procedures recommended by the manufacturer based on a sandwich technique using two monoclonal antibodies.

The first step is the immunological test and involves:

- The addition of 250 µl of Conjugate to 25 µl of test sample, control and standards, and incubating at 20^0c for 30 minutes and shaking continuously.
- Aspirating of the liquid from each tube.
- Rapidly dispensing the working wash solution in series of 48 tubes.
- Re-aspirating almost immediately, and repeating the washing procedure.
- Finally thoroughly aspirating all traces of solution.

The second step is the ENZYMATIC STEP and involves:

- Addition of 300 µl of colour working solution (Colour1 + Colour 2) to all tubes (test, control and standard) and incubating for another 30

minutes at 20^0C in the dark, at the end of which the reaction is stopped by the addition of 2 ml of stopping reagent (Colour 3).

- Reading at 492nm against reagent blank after shaking.

Results are calculated on calibration curve of the mean absorbance of the standard using reading of mean absorbance of each sample.

CHAPTER 3

RESULTS AND FINDINGS

3. Results and findings

3.1 Poverty study

This study was undertaken during the first year and assistance in designing the questionnaires and the survey was obtained from an experienced Government Statistical Officer and private survey officers. Although this study was meant to identify and locate suitable sites for the scientific research, its design was based on the Living Standards Measurement Survey (LSMS) that has been used by international organisations like the Word Bank, with slight modifications to better suit the aim, objectives and available resources.

3.1.1 Sample frame

A sample frame was generated using the following indicators:

- Poorest wards ranked in terms of relative deprivation index developed by the Central Statistical Office, supplemented by a list

of low performing school on the assumption that school performance is directly related to economic and social poverty.

In this way a total of 402 enumeration areas were identified and by further selection, a total of 100 enumeration areas were finally obtained. By interviewing every 6th household in the enumeration areas, 2200 households were successfully interviewed and interesting observations made. These included:

3.1.1.1 **Labour market participation**

Every individual between the age of 15 and 64 was regarded as potential worker with the exception of disabled persons, students attending schools and housewives who were termed 'economically not active'. As such there were 6269 people of working age with only 4092 people who could be classified as economically active. The results of the labour market participation are as at table 2.

Table 2. Labour market participation of persons aged 15 - 64

	Population 15 –64	Economically active		Not economically active	
	Number	Number	%	Number	%
Male	3210	2695	84	515	16
Female	3059	1397	46	1662	54
Total	6269	4092	65	2177	35

3.1.1.2 Unemployment

Potential worker comprised those in employment as well as those not in employment. The rate of unemployment is the proportion of the population

of potential worker not in employment. Table 3, shows the incidence of unemployment at the time of survey. Unemployment is far higher among women than men.

Table 3. Employment and unemployment rates

	Economically active	Employed		Unemployed	
	Number	Number	%	Number	%
Male	2695	2202	82	493	18
Female	1397	957	69	440	31
Total	4092	3159	77	933	23

3.1.1.2.1 Working age population distribution

The pie chart below (Fig.15) gives information about labour market participation, employment and employment and how the working age population is distributed.

Fig.15 Labour market participation.

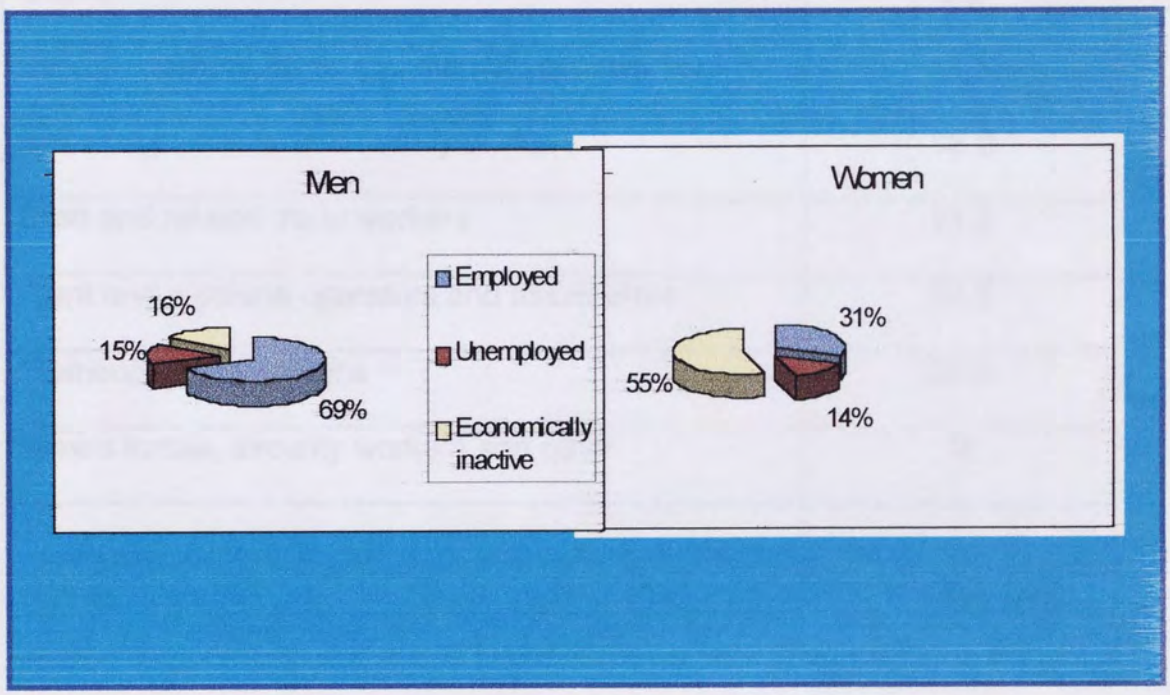


Table 4 shows the occupational breakdown of the workers belonging to household that participated in the survey.

Table 4. Percentage distribution of sample working population by major occupational group

MAJOR OCCUPATIONAL GROUP	%
Senior officials and managers #	0.8
Professionals #	1.1
Technician and related profession	2.6
Clerks	5.2
Service workers, shop and market sales workers	11.3
Skilled agricultural and fishery workers	16.2
Craft and related trade workers	11.3
Plant and machine operators and assemblers	18.5
Elementary occupations	33.0
Armed forces, security workers and other	0
TOTAL	100

#People defined themselves in these categories and do not necessarily reflect educational achievement.

3.1.1.2.2 **Percentage distribution of sample working population by major occupational group and gender**.

From table 5, it can be deduced that workers residing in the areas sampled for poverty study are heavily concentrated in low skilled occupations. Very low numbers are employed in high skilled professional and technical occupation.

Table 5. Percentage Distribution by major occupational group and gender

MAJOR OCCUPATIONAL GROUP	MALE	FEMALE
Senior officials and Managers	0.5	1.6
Professionals	1.2	0.9
Technicians and related profession	3.2	1.4
Clerks	5.0	5.8
Service workers, shop and market sales workers	11.6	10.8
Skilled agricultural and fishery workers	18.5	10.9
Craft and related trade workers	12.5	8.7
Plant and machine operators and assemblers	13.4	29.6
Elementary occupations	34.2	30.3
TOTAL	100.0	100.0

3.1.1.2.3 Distribution of working population by major industrial division and gender

In the impoverished regions workers, in particular women, are concentrated in the agricultural, manufacturing, and various service sectors, while significant numbers of men are also employed in the construction sector.

Table 6. Distributions of working population by major industrial division and gender.

MAJOR INDUSTRIAL DIVISION	MALE	FEMALE
Agriculture, hunting and forestry	21.3	18.8
Fishing	6.4	0.7
Mining and quarrying	2.0	0.5
Manufacturing	15.3	42.9
Electricity, gas and water supply	1.9	1.7
Construction	16.9	2.2
Wholesale and retail trade	8.8	3.9
Restaurants/hotels/entertainment	3.7	4.2
Transport, storage and communication	6.6	0.7

Financial, insurance, real estate and business services	1.5	1.9
Educational services	1.5	2.8
Health and social services	2.5	2.5
Legal services	0.7	0.3
Domestic services	2.5	10.7
Armed forces and security services	1.2	0.5
Other services	7.2	5.7
TOTAL	100.0	100.0

3.1.1.3 Wages

The net monthly salary of workers in the impoverished regions sampled is Rs. 3860. Table 6 shows that professionals, the best paid workers have an average salary of Rs. 5251 while plant and machine operators are the least paid workers with average monthly salary of Rs. 3484. There are not wide disparities in earnings between classes.

Table 7. Mean and median wages

MAJOR OCCUPATIONAL GROUP	NET MONTHLY SALARY (Rs)	
	MEAN	MEDIAN
Senior officials and managers	4133	400
Professionals	5251	4800
Technicians and related professions	4933	4500
Clerks	4250	4000
Service workers, shop and market sales workers	4030	3500
Skilled agricultural and fishery workers	3922	3500
Craft and related trade workers	4924	3800
Plant and machine operators	3484	3000
Elementary occupations	3524	3200

3.1.1.3.1 Wages and gender

Women are seen to earn less than their male counterparts even in the same occupation classes. Table 8 refers only to those workers working between 35 – 45 hours a week.

Table 8. Average monthly wage by major occupational group and gender (Working between 35 and 45 hours a week).

MAJOR OCCUPATIONS	Mean net monthly salary (Rs.)		Median net monthly salary (Rs.)	
	MALE	FEMALE	MALE	FEMALE
Senior officials and managers	3100*	3135*	3000*	4000*
Professionals	6736*	4375*	6000*	2000*
Technician and related professions	5286	4795*	4500	5100*
Clerks	4573	3837	4000	3600
Service workers, shops and market sales workers	4144	2554	4000	2500
Skilled agricultural and fishery workers	4141	2834	3600	2600
Craft and related trade workers	4945	3739	3900	3000
Plant and machine operators/assemblers	4065	2800	3932	2500
Elementary occupations	4161	2743	3800	2500

* Sample size of less than 10 workers in these categories.

3.1.1.3.2 **Household members in paid employment**

The survey showed that in the impoverished regions of Mauritius, there were significant numbers of households with no one in paid employment. This is being presented in table 9.

Table 9. Percentage distribution of household by number of income earners

NUMBER OF INCOME EARNERS	PERCENT
None	14.8
One	47.5
Two	24.6
Three	8.3
Four	3.5
Five	1.2
Six or more	0.1

3.1.2 Access quality and affordability of the surveyed population to health

Access to basic services including water and sanitation is important indicators of well being. The survey shows that access to all health care providers (doctors, area/community health centres and hospital) have improved when compared to the immediately preceding years and is tabulated in table 10.

Table. 10. Access to healthcare

ACCESS	DOCTORS	AREA/ COMMUNITY HEALTH CENTRES	HOSPITALS
BETTER THAN BEFORE	70.9	71.0	61.4
WORSE THAN BEFORE	9.4	8.2	12.5
SAME AS BEFORE	19.8	20.8	26.1
TOTAL	100.0	100.0	100.0

The quality of health care also has been felt as having been improved and this is shown in table 11.

Table 11. QUALITY OF SERVICE OF HEALTH CARE PROVIDERS

QUALITY	DOCTORS	ARE/ COMMUNITY HEALTH CENTRES	HOSPITALS
BETTER THAN BEFORE	70.2	63.4	53.5
WORSE THAN BEFORE	10.8	15.2	21.8
SAME AS BEFORE	19.0	21.4	24.7
TOTAL	100.0	100.0	100.0

3.1.2.1 Affordability of health care

The survey indicates that private medical practitioners are becoming less affordable in contrast to public community health centres. The results are summarised in table 12.

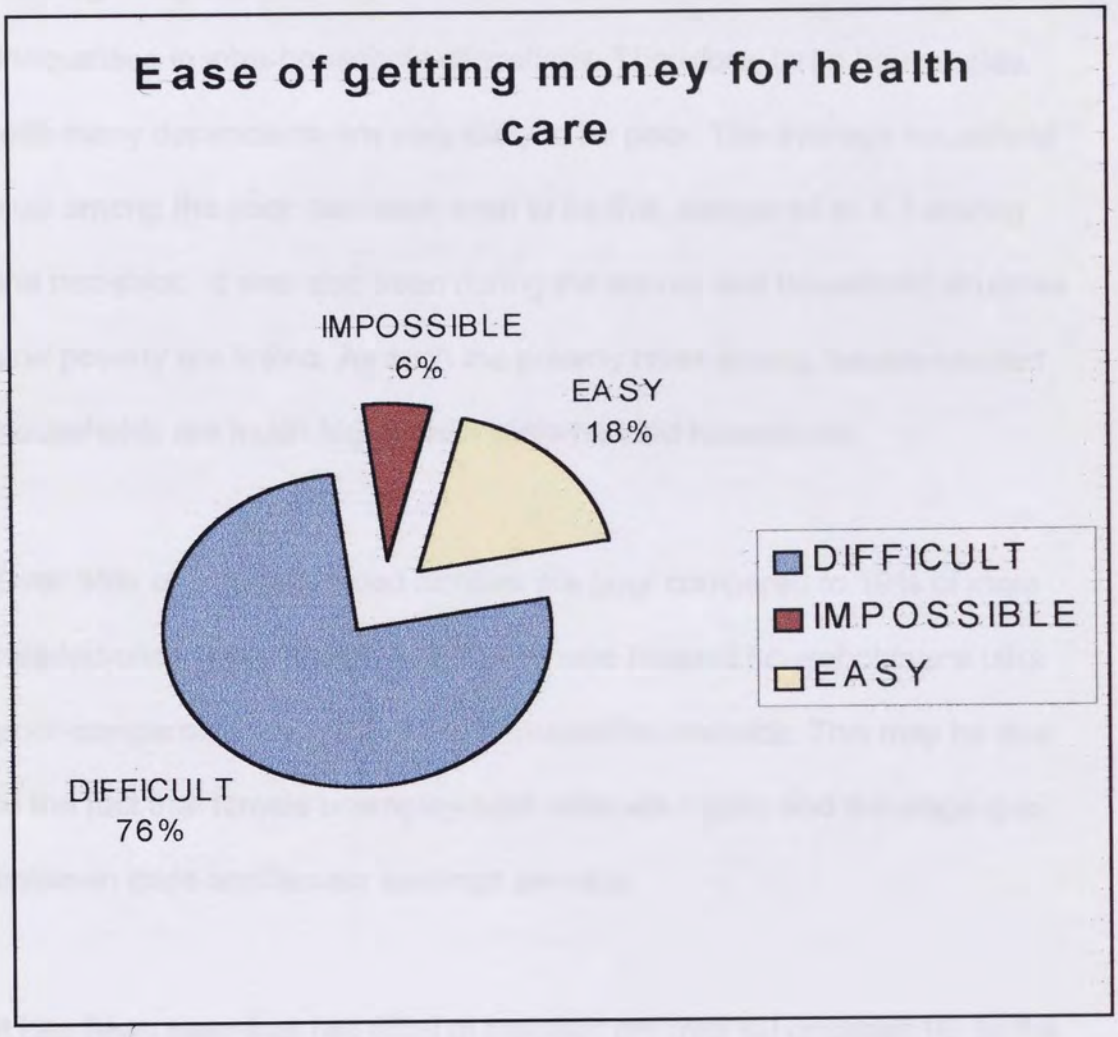
Table 12. Affordability of Health Services

	DOCTORS	AREA/ COMMUNITY HEALTH CENTRES	HOSPITALS
LESS AFFORDABLE	54.8	21.2	20.8
MORE AFFORDABLE	18.9	45.4	42.7
NO CHANGE	12.6	32.6	35.7
NOT AFFORDABLE AT ALL	13.7	0.8	0.8
TOTAL	100.0	100.0	100.0

3.1.2.2 Money to cover medical expenses

About 76 % of the surveyed subjects face difficulty obtaining medical expenses and 6% find it impossible. The results of finding are being summarised in Figure 16.

Figure 16. Ease of Getting Money for Health Care



3.1.3 Household structure and poverty

The household survey gave information at household level mainly. It could show much about the inequalities in resource allocations within households. Poor children for this study were only children who were living in poor households. There may however be many children, who

although they live in non-poor households are still poor because of inequalities in intra-household allocations. Therefore, large households with many dependants are very likely to be poor. The average household size among the poor has been seen to be five, compared to 4.1 among the non-poor. It was also seen during the survey that household structure and poverty are linked. As such the poverty rates among female-headed households are much higher than male headed households.

Over 35% of female-headed families are poor compared to 19% of male headed-ones. Very strikingly, 6% of female-headed households are ultra poor compared to only 3% of male-headed households. This may be due to the fact that female unemployment rates are higher and the wage gap between male and female earnings persists.

It has been seen that two fifths of the poor are over 60 or under 16. In the sampled areas, two children in every five live in poor households. Children in some regions are far more likely to be poor Compared to those in other regions. Table 13 is a summary of findings.

Table 13. Child risk of poverty in sampled areas.

REGION	PERCENTAGE OF CHILDREN THAT ARE POOR	PERCENTAGE OF CHILDREN THAT ARE ULTRA-POOR
Port Louis	15.0	2.7
Riviere du Rempart	26.1	2.4
Pamplemousses	21.5	4.9
Black River	22.8	2.8
Savanne	24.5	1.3
Grand Port	16.9	1.2
Flacq	31.8	1.9
Moka	25.0	3.0
Plaine Wilhems	22.4	3.9

3.1.4 Major findings

Just over 22% of the households and 29% of the individuals in the surveyed areas can be classified as being poor. This is based upon a relative poverty line for Mauritius below which household members consume less than 50% of the mean consumption level. Using a poverty

line of less than 25% of the mean consumption, about 6% of the surveyed population can be classified as ultra-poor.

In terms of the poverty profile of the poor, large households with many dependants are more likely to be poor.

Poverty and unemployment are also closely linked. It is seen that unemployment rate among the poor is 33% compared to only 17% among the non-poor. Alarmingly, the unemployment rate among women from poor households is 43%. To make things worse, labour force participation also differs between the poor and the non-poor, with many working-age poor being out of the labour force due to illness, disability, catching up with education or domestic responsibilities.

The poor and the more especially the ultra-poor are mostly squatters living in crowded conditions. They have less access to modern sanitation system and water inside their home, as was the case with La Pipe.

3.2 Parasitic status and blood profiles

To determine the parasitological and biochemical/immunohaematological status of the child populations in the poverty regions of Mauritius, four villages were selected at random and every child aged 4 – 11 was recruited. This gave a sample size of 301 children. However, due to lack of information on one child who could not be reached subsequently as a result of the demolition of La Pipe Village, that sample was being rejected and the sample size was now being brought down to 300.

Stool and urine specimens were collected from each child on six different occasions giving six pairs of samples from each child. These were examined for the presence of parasites when still fresh. After this, blood was collected from each child for the determination of biochemical and immunohaematological parameters and for the detection of the presence or absence of blood parasites.

3.1.5 Distribution of the surveyed children

There were more males than females and the child populations were distributed as in table14. The Percentage distribution of the children are at table 15 and table 16. As can be seen, males and females are well

balanced except for Mare Chicose where there are twice as many males as females. Hence a slight imbalance. We also note that the sample is not equally distributed between the different regions and also in the control. Mare Chicose and La Pipe are at the lower extreme and Mare D'Australia and Pointe Brocus are at the higher extreme. This observation can prove useful during the analysis while comparing the different regions.

Table 14. Distribution of children by Region and Gender

REGION	SEX		TOTAL
	MALE	FEMALE	
Control	25	26	51
La Pipe	20	16	36
Mare Chicose	16	8	24
Mare D'Australia	59	42	101
Pointe Brocus (Riv des Creoles)	43	45	88
TOTAL	163	137	300

Table 15 Distribution of the children by Region and Gender (Row Percentages)

REGION	SEX		TOTAL
	MALE	FEMALE	
	ROW	ROW	ROW
Control	49.0	51.0	100.0
La Pipe	55.5	44.4	100.0
Mare Chicose	66.7	33.3	100.0
Mare D'Australia	58.4	41.6	100.0
Pointe Brocus	48.9	51.1	100.0

Table 16. Distribution of the children by Region and Gender (Column Percentages)

REGION	SEX		TOTAL
	MALE	FEMALE	COL %
Control	15.3	19.0	17.0
La Pipe	12.3	11.7	12.0
Mare chicose	9.8	5.8	8.0
Mare D'Australia	36.2	30.7	33.7
Pointe Brocus	26.4	32.8	29.3
TOTAL	100.0	100.0	100.0

Distribution of children by age is given in the summary statistics on age of children by Regions at table 17. There seems to be no difference in ages of children between regions. Of course the age group of the test children was already pre-determined.

Table 17. Summary Statistics on Age of children by Regions

REGIONS	MEAN AGE	MEDIAN	MODE	STANDARD DEVIATION
All	8.327	9.000	10.000	2.250
Pointe Brocus	8.466	9.000	10.000	1.971
Mare Chicose	8.792	9.000	10.000	1.793
La Pipe	7.167	7.500	4.000	2.854
Mare D'Australia	8.703	9.000	10.000	1.983
Control	7.941	8.000	10.000	2.634

3.2.2 Distribution of Children with respect to parasitic infection

As many as 51.7 % of the child populations in the impoverished regions do not harbour any parasite or have been freed from parasitic infection.

This is given in table 18. Table 18 also shows that among those children who are infested, the majority harbours only one species.

Table 18. Distribution of children by Numbers of Parasites

NO. OF PARASITES	FREQUENCY (#subjects)	PERCENT
0	155	51.7
1	86	28.7
2	40	13.3
3	18	6.0
4	1	0.3
TOTAL	300	100.0

Table 19 shows that Children living in Mare D'Autralia are the most affected and Mare Chicose, the least.

Table 19. Number of Parasites by Regions

REGIONS	NUMBER OF PARASITES					TOTAL
	0	1	2	3	4	
Control	51	-	-	-	-	51
La Pipe	2	13	11	9	1	36
Mare Chicose	5	15	3	1	-	24
Mare D'Australia	46	34	16	5	-	101
Pointe Brocus	51	24	10	3	-	88
TOTAL	155*	86	40	18	1	300

* 104 excluding control

Table 20 shows that the vast majority of affected children harbour *Trichuris trichiura*. There are indications however that in the majority of case infections occurs along with other parasite/s.

Table 20. Distribution of infected children by types of parasites

PARASITE	TOTAL	PERCENTAGE
Ascaris lumbricoides	70	31.25
Chilomastix mesnili	1	0.45
Entamoeba coli cysts	2	0.89
Entamoeba histolytica Cysts	2	0.89
Entamoeba histolytica trophozoites	1	0.45
Endolinax nana cysts	5	2.23
Endolinax nana trophozoites	1	0.45
Enterobius vermicularis ova	6	2.68
Giardia lamblia cysts	12	5.36
Giardia lamblia trophozoites	2	0.89
Hookworm ova	14	6.25
Precystic amoeba	6	2.66
Strongyloides stercoralis larvae	2	0.89
Trichuris trichiura ova	100	44.64
TOTAL	224	100.0

Figure 17 shows that ascaris and trichuris are among the most prevalent parasites in the impoverished regions of Mauritius.

Fig. 17. Prevalence of intestinal parasites in the impoverished regions of Mauritius

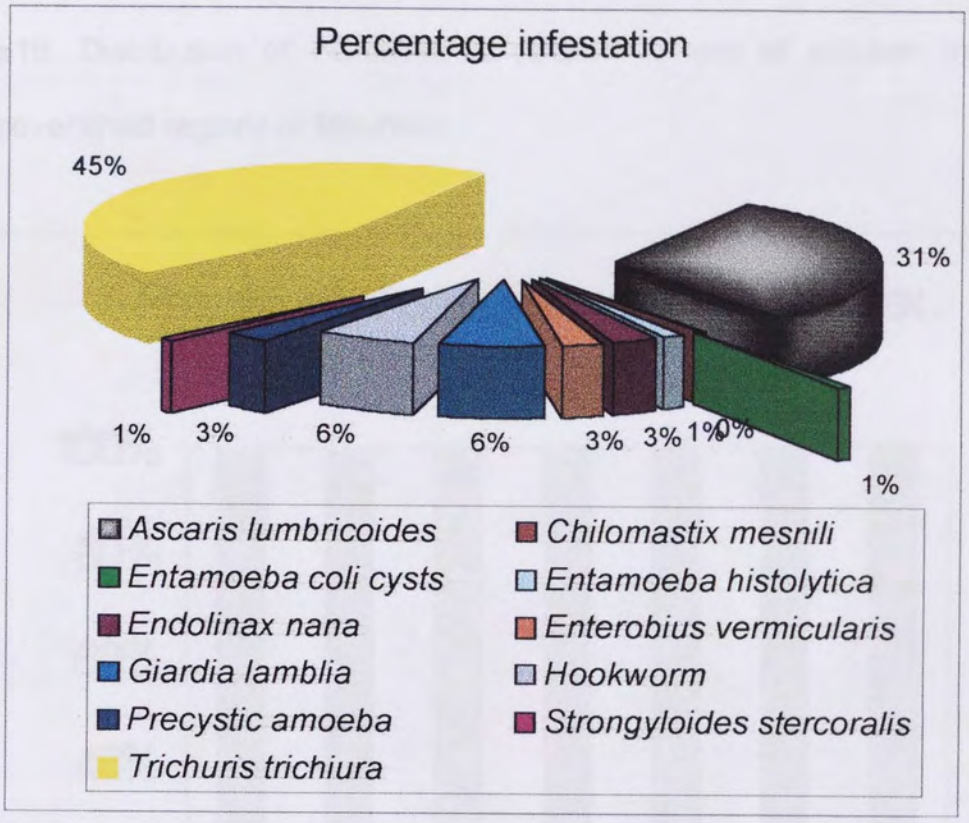
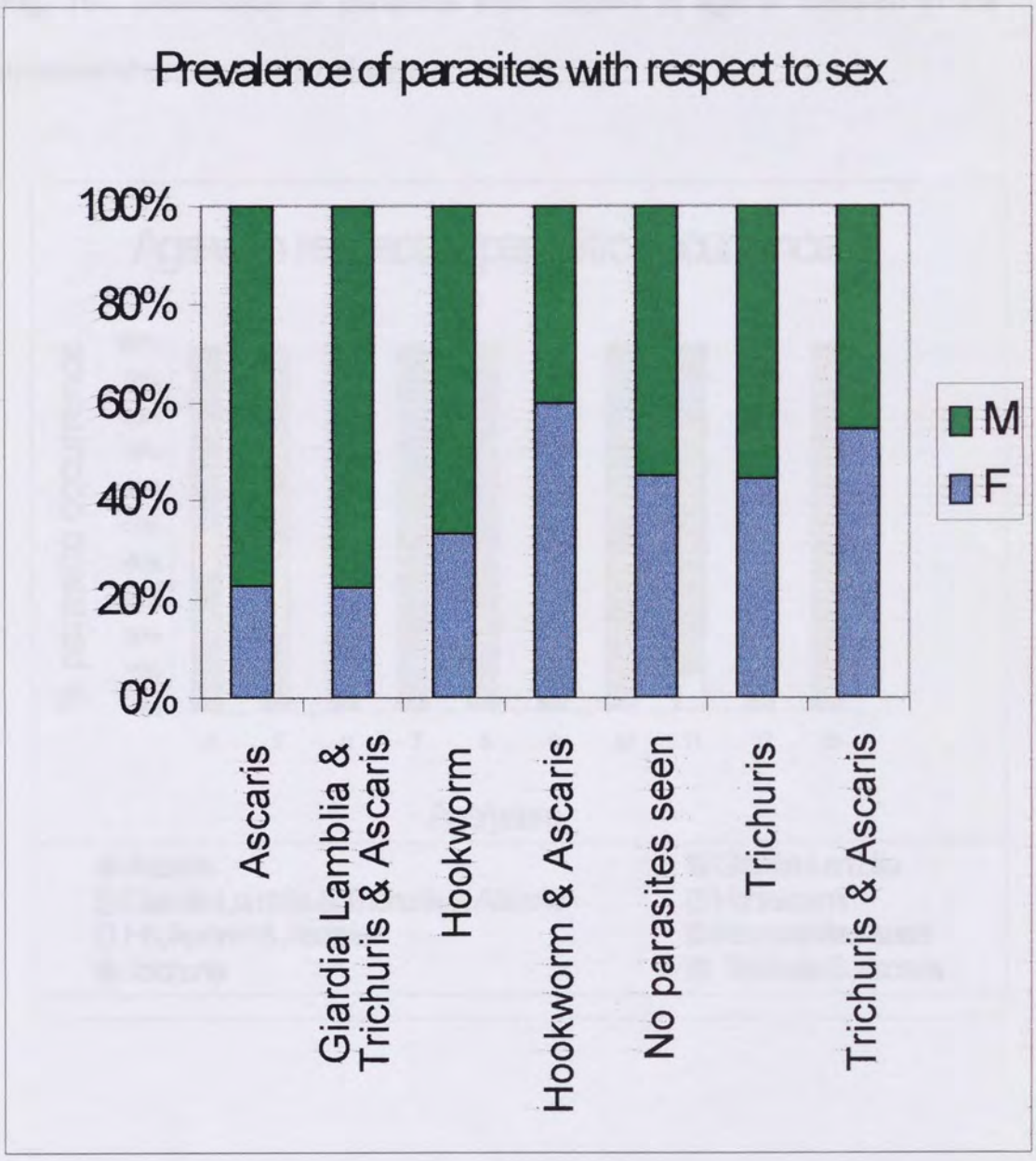


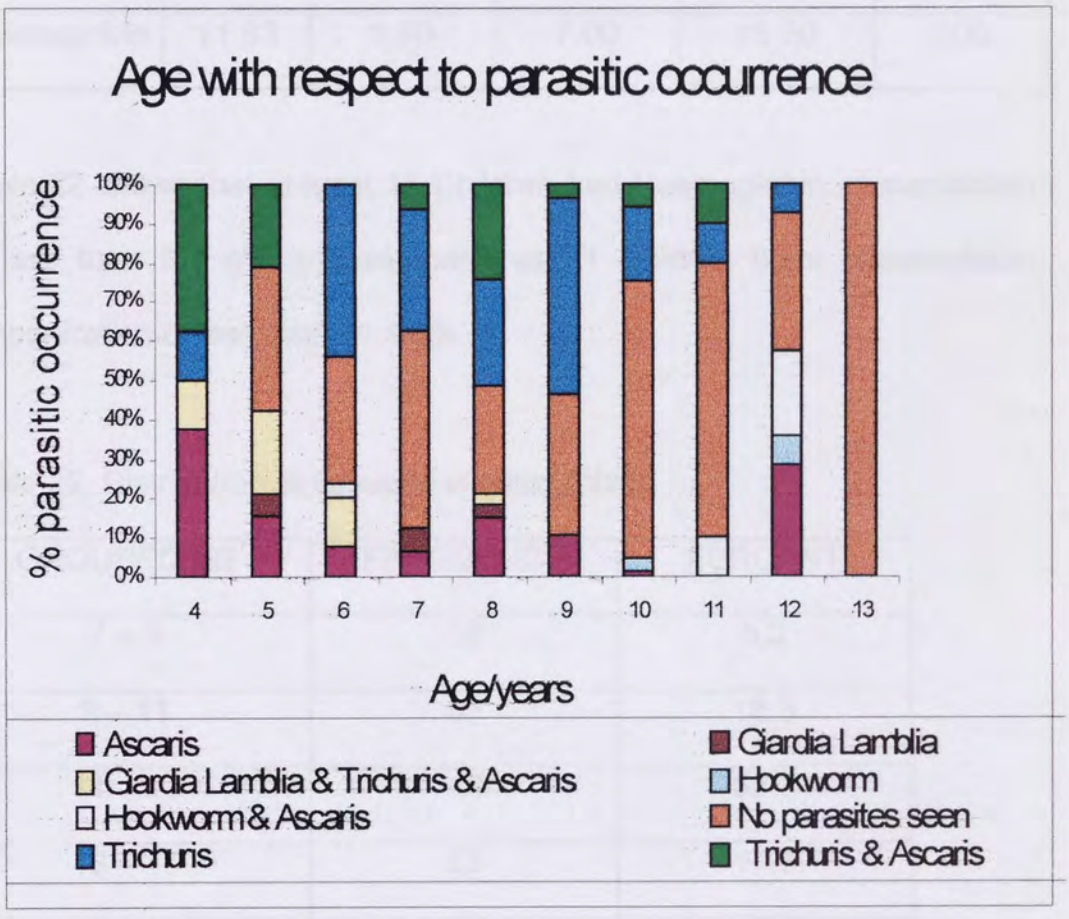
Figure 18 shows that in the impoverished regions of Mauritius, males are more affected than females for almost all the parasires.

Fig.18. Distribution of Parasites in relation to sex of children in the impoverished regions of Mauritius.



In the impoverished regions of Mauritius, prevalence of *Giardia lamblia* in the young children is quite obvious, but there is no age pattern for *Ascaris lumbricoides* and *Trichuris trichiura* as cn be seen in Figure 19. No hookworm is present up to the age of 6.

Fig. 19. Distribution of parasites with respect to age of children in the impoverished regions of Mauritius



3.2.3 Children and the haematological parameters

As regards haemoglobin concentration, table 21 below indicates that some children have haemoglobin concentration as low as 7g%, as compared to others who have haemoglobin concentration of as high as 16.5 gm%.

Table 21. Summary statistics on Haemoglobin

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	N
Haemoglobin	11.83	1.60	7.00	16.50	300

Table 22 shows that at least 16 Children had Haemoglobin concentration of less than 9.1 g% and as many as 71 children have Haemoglobin concentration of less than 11.1 g%.

Table 22. Distribution of Grouped Haemoglobin

GROUPED HB	FREQUENCY	PERCENT
7 – 9	16	5.3
9 – 11	55	18.3
11 – 13	166	55.3
13 – 15	53	17.7
15 – 17	10	3.3
TOTAL	300	100.0

In table 23 it is seen that there are significant differences in average HB between regions. When the 95% confidence intervals are compared, there overlapping is to be seen implying that the differences are significant.

Table 23. Average HB by Region

REGION	HB		CI
Control	12.97	.19	12.6 – 13.34
La Pipe	10.73	.22	10.30 – 11.16
Mare Chicose	11.08	.38	10.34 – 11.82
Mare D'Australia	11.84	.17	11.51 – 12.57
Pointe Brocus	11.79	.13	11.54 – 12.04

In table 24, it can be seen that children from all regions have HB significantly lower than control. It can also be seen that children of La Pipe and Mare Chicose, have similar HB but lower that those of Mare D'Australia and Pointe Brocus. Also, that 50% of children in La Pipe, have HB below 11g% compared to Mare Chicose where it is 1/3 or to Mare D'Australia where it is 29/101, or Pointe Brocus, wher it is 16/88. As regards to control, almost 50% of the children have Hb above 13g%.

Table 24. Grouped HB by Region

REGION	HB (GROUPED)					TOTAL
	7 - 9	9 - 11	11-13	13 - 15	15 – 17	
Control	-	-	26	20	5	51
La Pipe	2	16	18	-	-	36
Mare Chicose	4	4	12	4	-	24
Mare D'Australia	8	21	49	14	5	101
Pointe Brocus	2	14	61	11	-	88
TOTAL	16	55	166	53	10	300

Table 25 shows that there are more male than female with lower HBs while for higher HBs numbers of male and female are equal.

Table 25. HB by Gender

SEX	HB Grouped					TOTAL
	7-9	9-11	11-13	13-15	15-17	
MALE	11	33	83	26	10	163
FEMALE	5	22	83	27	-	137
TOTAL	16	55	166	53	10	300

At table 26, it can be seen that HB increases with age with a sudden drop at age 12.

Table 26. Average HB by Age

AGE	MEAN	STANDARD ERROR OF MEAN
4	12.10	.35
5	11.45	.27
6	12.14	.21
7	11.68	.26

8	11.34	.25
9	12.03	.22
10	12.12	.16
11	12.01	.40
12	11.18	.60
13	12.50	-

Table 27 shows that females tend to have lower HB than male although the confidence intervals (11.79 ± 19.6X0.12) for females is not significantly different from those of male (11.85 ± 1.96x0.14)

Table 27. Average and Standard Error of HB by Gender

SEX	HB	
	MEAN	STANDARD ERROR OF MEAN
MALE	11.85	.14
FEMALE	11.79	.12

Table 28 shows that low HB concentration associates itself with high number of parasites.

Table 28. Relationship between HB and Number of Parasites

No. of Parasites	HB (GROUPED)					TOTAL
	7-9	9-11	11-13	13-15	15-17	
0	3	7	97	40	8	155
1	4	21	48	12	1	86
2	8	14	16	1	1	40
3	1	12	5	-	-	18
4	-	1	-	-	-	1

The above is shown more clearly in table 29 where average HB decreases steadily with increase in types of parasites. It also shows an increase in variability of HB with increase in number of parasites.

Table 29. Relationship between Average HB and Number of Parasites

NO. OF PARASITES	HB	
	MEAN	STANDARD ERROR OF MEAN
0	12.43	.11
1	11.63	.15
2	10.55	.28
3	10.54	.28
4	9.10	-

With regards to Packed Cells Volume as expected, no significant difference is seen between La Pipe and Mare Chicose (A) and also between Mare D'Australia and Pointe Brocus (B). but there is a difference in average PCV between A & B.

Table 30. Relationship between average PCV and Regions

REGION	PACKED CELLS VOLUME (PCV)		
	MEAN	STD. ERROR OF MEAN	
La Pipe	.36	.0072	
Mare Chicose	.36	.0091	A
Mare D'Australia	.38	.0048	
Pointe Brocus	.38	.0032	B

Genderwise, both average and standard error are the same as can be seen from table 31.

Table 31. Relationship between Average PCV and Gender

SEX	PCV	
	MEAN	STD ERROR OF MEAN
Male	.37	.0038
Female	.37	.0036

When the average PCV is compared to age there is no definite pattern. This is probably due to lowering of PCV in children carrying parasites and/ or malnourished children forming part of the test sample and that parasites and/ or malnutrition are randomly distributed among these children.

Table 32. Relationship between Average PCV and Age

AGE	MEAN PCV	STD ERROR OF MEAN
4	.37	.0062
5	.37	.0091
6	.37	.0055
7	.37	.0058
8	.36	.0060
9	.38	.0053
10	.38	.0046
11	.37	.0151
12	.37	.0217
13	.41	-

As expected, table 33 shows a decrease in PCV with increase in number of parasites.

Table 33. Relationship between average PCV and Number of Parasites

NO. OF PARASITES	PCV	
	MEAN	STD ERROR OF MEAN
0	.39	.0033
1	.38	.0041
2	.35	.0081
3	.35	.0086
4	.29	-

Table 34 is a summary statistics on PCV and table 33 is the distribution of grouped PCV.

Table 34. Summary Statistics on PCV

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	N
PCV	.37	.04	.25	.48	249

Table 35. Distribution of Grouped PCV

GROUPED PCV	FREQUENCY	PERCENT
.25 - .30	16	6.4
.30 - .35	33	13.3
.35 - .40	120	48.2
.40 - .45	70	28.1
.45 - .50	10	4.0
TOTAL	249	100.0

Table 36 shows an increasing trend in PCV above 35% from La Pipe to pointe Brocus.

Table 36. Distribution of grouped PCV by Region

REGION	PCV%					TOTAL	PCV >35
	25-30	30-35	35-40	35-40	40-45		
La Pipe	4	7	15	10	-	36	69%
Mare Chicose	3	3	13	5	-	24	75%
M. D'Australia	6	18	35	32	10	101	76%
Pointe Brocus	3	5	57	23	-	88	91%
TOTAL	16	33	120	70	10	249	

Table 37 shows that in the test sample, more females had PCV between 40 and 45 than male.

Table 37. Distribution of Grouped PCV by Gender

PCV %	SEX		TOTAL
	MALE	FEMALE	
25 – 30	10	6	16
30 – 35	20	13	35

35 – 40	65	55	120
40 –45	34	36	70
45 – 50	9	1	10
TOTAL	138	111	249

Table 38 shows that in the test sample children in the age group of 4 – 8 have PCV between 35 – 40 after which PCV increases to the average of 43%.

Table 38. Distribution of Grouped PCV by Age

AGE	PCV%					TOTAL
	25-30	30-35	35-40	40-45	45-50	
4	-	-	10	1	-	11
5	1	5	10	4	1	21
6	1	3	16	6	-	26
7	-	3	12	4	-	19
8	3	8	20	8	-	39
9	-	6	16	14	-	36

10	3	5	30	28	2	68
11	1	1	6	2	-	10
12	7	2	-	2	7	18
13	-	-	-	1	-	1
TOTAL	16	33	120	70	10	249

Table 39 shows that comparatively children without infection are more likely to have higher PCVs.

Table 39. Relationship between Grouped PCV and Number of Parasites

PCV %	NUMBER OF PARASITES					TOTAL
	0	1	2	3	4	
24-30	1	4	8	2	1	16
30-35	7	13	7	6	-	33
35-40	52	41	18	9	-	120
40-45	38	25	6	1	-	70
45-50	6	3	1	-	-	10
TOTAL	104	86	40	18	1	249

Table 40 shows the distribution of WBC between regions. Children of La Pipe and Mare Chicose are seen to have comparatively higher WBCs.

Table 40. Relationship Between Average White Blood cell counts (WBC) and Region

REGION	WBC	
	MEAN	STD ERROR
La Pipe	9780	419
Mare Chicose	9737	575
Mare D'Australia	8487	185
Pointe Brocus	8030	241

In table 41, using confidence interval it is seen that there is no significant difference in WBCs between male and female.

Table 41. Relationship between Average WBC and Gender

SEX	WBC		
	MEAN	STD ERROR OF MEAN	CI
Male	8547	201	8152–8943
Female	8761	211	8346– 9175

When looking at the relationship WBC and age, there seems to be no relation between age and WBC as in table 42. Table 43 shows that the average WBC is around 8642/ml.

Table 42. Relationship between Average WBC and Age

AGE	WBC	
	MEAN	STD ERROR OF MEAN
4	9872	1038
5	9100	450

6	8532	490
7	9473	756
8	8471	267
9	8617	401
10	8156	255
11	9440	676
12	8616	385
13	5400	-

Table 43. Summary Statistics for WBC

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	N
WBC	8642	2283	4100	17800	244

Table 44 shows that as the number of parasite increases, the WBc also increases and that most of the children have WBCs ranging between 8000 – 10 000/ml. This is seen in table 45.

Table 44. Relationship between Average WBC and Number of Parasites

NO. OF PARASITES	WHITE BLOOD CELLS COUNT (WBC)	
	MEAN	STD ERROR OF MEAN
0	8149	192
1	8462	260
2	9158	328
3	10616	589
4	15000	-

Table 45. Distribution of Grouped WBC

VALUE LABEL	FREQUENCY	PERCENT
4000 – 6000	24	9.6
6000 – 8000	72	28.9
8000 – 10000	88	35.3
10000 – 12000	47	18.9
12000+	13	5.2
TOTAL	249	100

For WBC exceeding 10 000/ml, La Pipe and Mare Chicose have a higher percentage of children and this can be seen in table 46.

Table 46. Relationship Between Grouped WBC and Region

REGION	WBC/1000					
	4-6	6-8	8-10	10-12	>12	TOTAL
La Pipe	1	5	17	8	5	36
Mare Chicose	2	4	8	7	3	24
Mare d'Australia	6	36	34	22	1	99
Pointe Brocus	15	27	29	10	4	85
TOTAL	24	72	88	47	13	244

Table 47 shows that there are no significant difference between WBC and sex.

Table 47. Relationship between grouped WBC and Gender

WBC	SEX		TOTAL
	MALE	FEMALE	
4000 – 6000	14	10	24
6000 – 8000	42	30	72
8000 – 10000	49	39	88
10000 – 12000	23	24	47
>12000	8	5	13
TOTAL	136	108	244

Table 48 shows that there do not seem to have any relationship between age and WBC.

Table 48. Relationship Between Grouped WBC and Age

Age	WBC/1000					TOTAL
	4-6	6-8	8-10	10-12	>12	
4	1	1	6	-	3	11
5	1	5	7	6	1	20
6	4	6	8	6	1	25
7	1	7	6	2	3	19
8	4	7	20	8	-	39
9	3	10	10	10	1	34
10	8	29	18	9	3	67
11	-	2	5	2	1	10
12	1	5	8	4		18
13	1	-	-	-	-	1
TOTAL	24	72	88	47	13	244

Out of the 101 children with no parasites the majority have a WBC ranging between 6000 and 12 000, as can be seen in table 49. But then this is also true for children carrying infestations.

Table 49. Relationship between Grouped WBC and Number of Parasites

NO. OF PARASITES	WBC/1000					TOTAL
	4-6	6-8	8-10	10-12	>12	
0	12	37	36	14	2	101
1	11	24	29	15	6	85
2	-	10	18	9	2	39
3	1	1	5	9	2	18
4	-	-	-	-	1	1
TOTAL	24	72	88	47	13	244

Table 50 is the distribution of test children by blood group showing the majority of children to be of blood group B or Blood group O.

Table 50. Distribution of Test Children by blood group (Control excluded).

BLOOD GROUP	FREQUENCY	PERCENT
A Positive	64	25.7
A Negative	2	0.8
B Positive	81	32.5
B Negative	2	0.8
O Positive	80	32.1
O Negative	0	0
AB Positive	20	8.0
AB Negative	0	0
TOTAL	249	100.0

Table 51 shows that there are appreciable differences in the pattern of blood group in the four regions.

Table 51. Relationship between Blood Group and Region

REGION	MAJOR BLOOD GROUP				TOTAL
	A	B	AB	O	
La Pipe	15	3	1	17	36
Mare Chicose	4	12	3	5	24
Mare D'Australia	22	42	7	30	101
Pointe Brocus	25	26	9	28	86
TOTAL	66	83	20	80	249

Table 52 shows that there do not appear to be significant difference in blood groups of the different sex of the children.

Table 52. Relationship between Blood Group and Gender

SEX	MAJOR BLOOD GROUP				TOTAL
	A	B	AB	O	
Male	38	47	12	41	138
Female	28	36	8	39	111
TOTAL	66	83	20	80	249

Table 53 shows that almost two thirds of the children having group A or O are infested with parasites.

Table 53. Relationship between Blood Group and Number of Parasites

NUMBER OF PARASITES	MAJOR BLOOD GROUPS				TOTAL
	A	B	AB	O	
0	22	43	9	30	104
1	32	29	5	20	86
2	8	8	6	18	40
3	3	3	0	12	18
4	1	0	0	0	1
TOTAL	66	83	20	80	249

Table 54 shows that the mean ferritin level of the children is 106 ng/ml.

Table 54. Summary Statistics on Ferritin

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	N
Ferritin	106.98	79.82	10	350	300

Table 55 shows that children of La Pipe and Pointe Brocus tend to have lower ferritin concentration.

Table 55. Relationship between Ferritin and Region

REGION	FERRITIN	
	MEAN	STD ERROR OF MEAN
Control	140.4	13.77
La Pipe	82.5	8.81
Mare Chicose	100.0	16.70
Mare D'Australia	106.8	7.74
Pointe Brocus	99.72	7.92

Table 56 shows that female candidates tend to have lower ferritin level than males.

Table 56. Relationship Between Ferritin and Gender

SEX	FERRITIN	
	MEAN	STD ERROR OF MEAN
MALE	130.2	7.47
FEMALE	79.34	3.58

Table 57 shows that plasma ferritin concentrations tend to increase with age of subjects.

Table 57. Relationship between Ferritin and Age

AGE	FERRITIN	
	MEAN	STD ERR OF MEAN
4	122.4	23.29
5	96.30	13.14
6	90.78	12.63
7	87.20	11.63

8	83.26	9.48
9	108.5	13.05
10	119.9	9.07
11	127.5	23.57
12	133.5	-
13	200.0	-

As number of parasites increases, plasma ferritin concentration decreases and this can be seen in table 58.

Table 58. Relationship Between Ferritin and Number of Parasites

NUMBER OF PARASITES	FERRITIN	
	MEAN	STD ERR OF MEAN
0	127.87	7.04
1	94.07	7.18
2	79.13	10.36
3	53.89	5.95
4	50.00	

In the sample, most of the children have ferritin level between 21 and 70 ng/ml as can be seen in table 59.

Table 59. Distribution of Grouped Ferritin

VALUE	FREQUENCY	PERCENT
< 20	10	3.3
21 – 70	132	44.0
71 – 120	82	27.3
121 – 170	26	8.7
171 – 220	19	6.3
221+	31	10.3
TOTAL	300	100.0

Almost in every region the majority of Children have ferritin level ranging between 21 and 120 ng/ml.

Table 60. Relationship Between Grouped Ferritin and Region

REGION	FERRITIN						
	<20	21 - 70	71- 120	121-170	171-220	221+	TOTAL
CONTROL	0	16	13	7	5	10	51
LA PIPE	2	19	10	2	2	1	36
MARE CHICOSE	1	11	7	2	0	3	24
MARE D'AUSTRALIA	4	45	24	12	6	10	101
POINTE BROCUS	3	41	28	3	6	7	88
TOTAL	10	132	82	26	19	31	300

Table 61 shows that female children tend to have lower ferritin level than male.

Table 61. Relationship between Grouped Ferritin and Gender

FERRITIN	SEX		TOTAL
	MALE	FEMALE	
<20	5	5	10
21 – 70	68	64	132
71 – 120	28	54	62
121 – 170	16	10	26
171 – 220	15	4	19
221+	31	0	31
TOTAL	163	137	300

Table 62 gives the relationship between grouped ferritin and age in the impoverished areas of Mauritius.

Table 62. Relationship between Grouped Ferritin and Age

AGE	FERRITIN						TOTAL
	<20	21-70	71-120	121-170	171-220	221+	
4	-	8	4	1	1	3	17
5	-	17	5	1	2	2	27
6	1	16	10	3	-	2	32
7	-	14	8	1	1	1	25
8	2	24	10	3	2	2	43
9	-	22	8	3	3	5	41
10	1	23	28	10	4	9	75
11	1	4	6	1	2	2	16
12	5	4	3	3	3	5	23
13	-	-	-	-	1	-	1
TOTAL	10	132	26	26	19	31	300

Table 63 shows that when number of parasite increases, plasma ferritin concentration decreases.

Table 63. Relationship Between Grouped Ferritin and No of Parasites

FERRITIN	No. of Parasites					Total
	0	1	2	3	4	
<20	-	2	6	2	-	10
21-70	52	45	21	13	1	132
71-120	53	20	6	3	-	82
121-170	13	10	3	-	-	26
171-220	14	3	2	-	-	19
221+	23	6	2	-	-	31
TOTAL	155	86	40	18	1	300

When compared to control, plasma ferritin concentration decreases with parasitic infections. This can be seen in Figure 20.

Fig. 20. Level of plasma ferritin with different species of parasites.

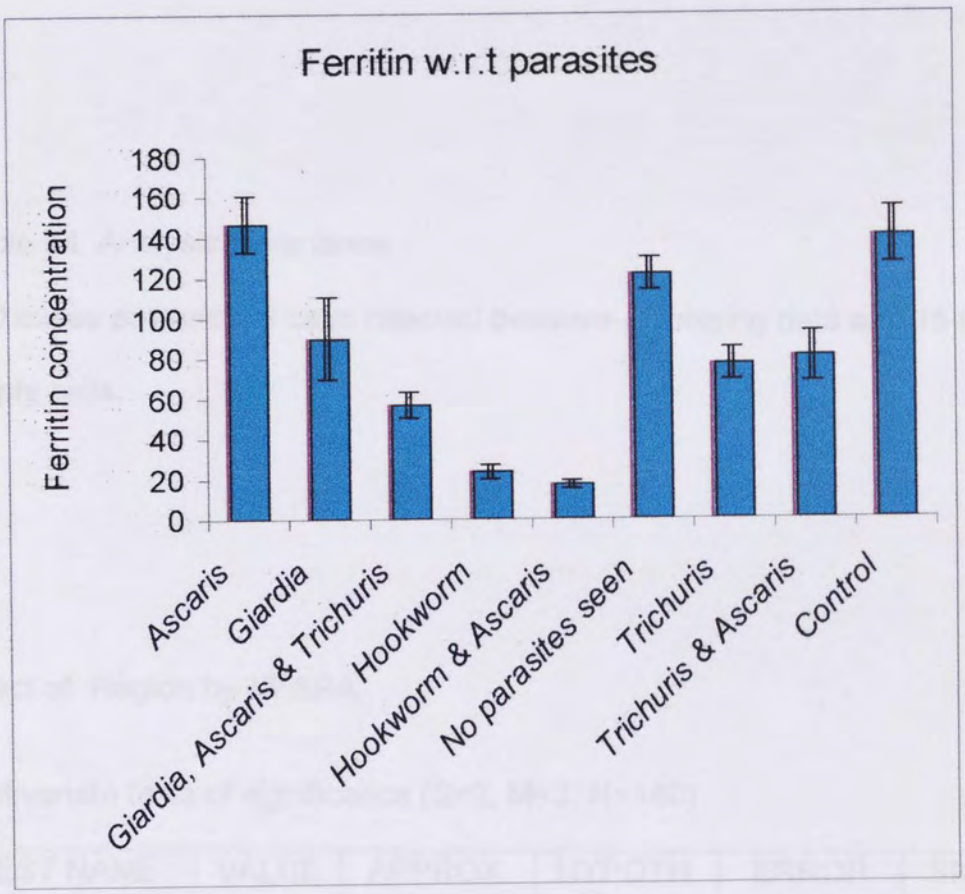


Table 64 is an analysis of variance. When looking at the effects of parasites and region of residence of the children on ferritin and HB, it can be seen that the parasite effects differ across regions, and that regions with high number of parasites is directly related to low HB and low ferritin.

Table 64. Analysis of variance.

300 cases accepted, 1 case rejected because of missing data and 15 non-empty cells.

Effect of Region by NPARA

Multivariate tests of significance (S=2, M=3, N=140)

TEST NAME	VALUE	APPROX. F	HYPOTH. DF	ERROR DF	SIG. OF F
PILLAIS	.15553	2.65150	18.00	566.0	.000
HOTTELLINGS	.17614	2.74969	18.00	562.0	.000
WILKS	.84759	2.70075	18.00	564.00	.000
ROYS	.13187				

NOTE: F-statistic for WILKS' Lambda is exact						
Univariate F-tests with (9,283) D. F.						
VARIABLE	HYPOTH. SS	ERROR SS	HYPOT. MS	ERRO R MS	F	SIG. OF F
FERRITIN	41178.5831	1677347.62	4575.39812	5927.02338	.77196	.643
HB	50.98225	521.51393	5.66469	1.84281	3.07395	.002

3.2.4 Test to determine if Hookworm has a marked effect on ferritin compared to other parasites.

The total number of children with Hookworm is low; only 14 or 4.6% of the sample. Only three children has Hookworm alone the others have hookworm in combination with other parasite/s. It is therefore difficult to say with certainty if any difference in the level of ferritin is due to Hookworm alone or not. Table 65 is a summary of parasites found together with hookworm.

Table 65. Parasites found together with Hookworm.

PARASITES	NO OF CHILDREN
Ascaris and *Trichuris* together	2
Trichuris trichiura	3
Precystic Amoeba	1
Ascaris lumbricoides	4
Strongyloides stercoralis	1
Hookworm alone	3
TOTAL	14

In order to find if ferritin level are affected by the presence of parasites other than Hookworm, a comparison of ferritin level of those children who have *Ascaris* and/or *Trichuris* (possibly with other parasites) but without Hookworm. Table 66 presents the summary statistics for ferritin in children with no worms, Hookworm and other worms.

Table 66. Summary Statistics for Ferritin in Children with no worms, Hookworm and other worms

PARASITES	NO OF CHILDREN	MEAN	MEDIAN	STANDARD ERROR OF MEAN
Non	104	122	100	8
Hookworm	14	23	20	2
Other worm	131	91	70	6
TOTAL	249			

It is clear from the above that children with Hookworm have lower mean and median ferritin, with a very low standard error. One cause of concern however, may be the effect of number of parasites on ferritin level. Earlier

we have seen that increases in the number of parasite lowers the ferritin level errespective of type of parasites. It is therefore relevant to look for differences that may be accounted for by differences in the number of parasites. Table 67 gives the summary statistics, which also includes the variable: number of parasites.

Table 67. Differences in Average Ferritin for Hookworm and other parasites after controlling for number of parasites.

	MEAN	MEDIAN	STD ERROR
Hookworm with No. of Parasites	122	100	8
Hookworm with one other Parasite	23	20	3
Hookworm with Two other parasites	23	20	3
Hookworm with three other Parasites	20	20	0

Other Worm (One Parasite)	97	70	7
Other Worm (2 Parasites)	95	70	12
Other Worm (3 Parasites)	58	50	6
Other Worm (4 Parasites)	50	50	-

From the above table, we see that the mean and the median ferritin levels are considerably lower for the hookworm compared to other worms at each of the three numbers of parasites.

The frequency distribution of the common parasites of Mauritius, in children in the impoverished regions of Mauritius is given in table 68. *Ascaris lumbricoides* and *Trichuris trichiura* are among the commonest parasites encountered in young children aged 5 – 13.

Table 68. Distribution of children by Type of Parasites (impoverished areas only)

Parasite	Percentage
Ascaris lumbricoides	27.8
Chilomastix mesnili	0.3
E.coli cysts	0.7
E.histolytica cyst/trophs	2.1
E.nana cysts	2.1
E.vermicularis	2.3
G.lambia Cyst/troph	5.5
Hookworm	5.6
S.stercolaris	0.7
Trichuris trichiura	39.8
No Parasite	10.7
Total	100

CHAPTER 4

DISCUSSION

&

CONCLUSION

Developing countries carry a heavy burden of disease and death. This is seen mainly in vulnerable groups such as children and women in the reproductive phase of life; in these groups inadequate nutrition, physiologic demands and lack of resistance make the effects of disease more serious. The hostile physical environment manifests itself in heavy infant and child mortality and a low expectancy of life, and these two indices taken together give an indication of the health of a community. Widespread illhealth in the community can produce far-reaching effects on the national economy because the effects of poor health go far beyond physical pain and suffering. Learning is compromised, returns to human capital diminish, and environments for entrepreneurial and productive activities are constrained. Furthermore, in view of the demonstrated importance of human capital to economic progress, it comes as little surprise that no country has attained a high level of economic development with a population crippled by high infant and maternal mortality, pervasive illness of its work force, and low life expectancy (World Bank, 1994). Also, disparity in child health by family income has been shown to have serious consequences for both the child and the society (Montgomery, L. E., Kiely, J. L., Pappas, G., 1996).

All illness in children interferes with the delicate balance between several physiological mechanisms, which determine optimum growth. Nutritional

intake, absorptive processes, cellular metabolism, endocrine function, circulation of blood and supply of oxygen and nutrients, removal of waste products and psychological mechanisms all interact in this highly complex process of growth in which new cells are being added to body tissues and acquire function.

At any given time the individual is a product of the continuing interaction between his genetic endowment and the environment. Nutrition is one of the most important environmental influences affecting the health and growth of children in developing countries. Closely related to nutrition and interacting with it is infection. The undernourished child has less resistance to infection so that even the most trivial illness tends to become severe. On the other hand, each episode of infection causes a further deterioration of nutritional status resulting finally in malnutrition. Nowadays it has also been shown that the economic crisis has positively led to the onset of several infectious diseases among communities with social deprivation (Poinsignon, Y., Marjanovic, Z., Farge, D., 1996).

Another important natural process is that as placentally derived immunity begins to decline, an infant becomes susceptible to a variety of infective hazards in the environment. In the harsh tropical environment with poor standards of hygiene and bad living conditions, infection is more frequent.

In a child, the response to infection may be different from that in adults, and is usually due to different level of immunity. Response to infection is also determined by the nutritional status of the host. It is not new that nursing care during illness, including nourishment offered to the sick, is important for securing early recovery. Also depending upon the main staple and the way it has been processed prior to consumption, a child may or may not be susceptible to deficiency disorders or toxicants. At the same time the body's biological processes are responding to the demands made for adaptation to the microbial, viral and the parasitic agents in the environment.

Children and pregnant and lactating women are the main vulnerable groups in a community. The demands of growth, conception and lactation produce physiological stresses. In addition, immaturity of body systems in young children and lack of acquired immunity to microbial agents make them especially vulnerable to infection.

In developing countries, the commonest illnesses are often called the 'disease of poverty'. Malnutrition, anaemia, tuberculosis, leprosy, worm infestations and many other infective and parasitic diseases are more common among the poorer classes. There is evidence to suggest that

infections like *Helicobacter pylori* are significantly associated with poverty, crowding and the level of education of the head of the household (Staat, M. A., Kruszon-Moran, D., McQuillan, G. M., Kaslow, R. A., 1996).

Poverty rarely exists by itself. Low wages and lack of adequate land resources are usually associated with insecurity of employment, illiteracy, bad housing, large families, exploitation and a host of other factors. The insecurity caused by poverty give rise to fatalism with a characteristic personality on one hand and chronic malnutrition with recurrent ill health on the other. It is now recognised that malnutrition lowers plasma anti oxidant vitamins like alpha-tocopherol, beta-carotene and retinol, although in areas where malaria and malnutrition co-exist, malaria exerts a greater influence on plasma antioxidants than does malnutrition (Adelekan, D. A., Adeodu, O. O., Thurnham, D. I., 1997). The well known overall effect of poverty finally, is that there is a high incidence of illness with little energy or will to change the situation. Characteristically, there is an attitude of hopelessness and despair arising from the awareness of the impossibility of ever achieving success in the prevailing values. "To be poor is not forgivable". This is what nature seems to convey with studies in New York indicating that poverty is directly associated with incidence of cancer (Gorey, K.M., Vena, J. E., 1995), and excess mortality (Geronimus, A. T., Bound, J., Waidman, T. A., Hillemeier, M. M., Burns, P. B., 1996).

Furthermore, malnutrition plays a major role in child mortality in tropical communities (Pelletier, D. L., Frongillo, E. A., Habicht, J. P., 1993) (Pelletier, D. L., Frongillo, E. A., Schroeder, D. G., Habicht, J. P., 1995) and is associated with lasting effects on growth and cognitive development. It has been estimated that in developing countries, 56% of child deaths are attributable to the potential effects of malnutrition, and that 83% of these death are attributable to mild-moderate as opposed to severe malnutrition (Pelletier, D. L., Frongillo, E. A., Schroeder, D. G., Habicht, J. P., 1995). Although in most tropical countries the aetiology is multifactorial, poor nutritional intake and infection appear to play important and synergistic roles (Scrimshaw, N. S., Taylor, C. E., Gordon, J. E., 1968). Inadequate diets have been shown to cause anaemia and nutrient deficiencies in red indians (Diez-Ewald, M., Torres-guerra, E., Layrisse, M., Leets, I., Vizcaino, G., Arteaga-Vizcaino, M., 1997) and also among inhabitants of Indonesian villages where it was seen that malnutrition was evidently caused by food shortage and unsatisfactory traditional feeding practices rather than by disease or other environmental risk factors (Jodjana, H., Eblen., 1997). In Zanzibar, 50% of the non-anaemic preschool children and approximately 90% of all the severely anaemic subjects were found to be iron deficient. Examination of their diet revealed

the presence of low bioavailability iron suggesting inadequate iron nutrition (Tatala, S., Svanberg, U., Mduma, B., 1998). Morbidity and mortality are precipitated when intestinal parasites aggravate this situation (Ighogboja, I. S., Ikeh, E. L., 1997).

Also, in Indonesia again, it has been shown that intestinal helminthiasis significantly lowers nutritional status (Hadju, V., Abadi, K., Stephenson, L. S., Noor, N. N., Mohammed, H. O., Bowman, D. D., 1995). Similar results have been obtained with Zairian children where it was found that infections with *Ascaris lumbricoides* and/or *Trichuris trichiura* cause childhood protein-energy malnutrition (Tshikuka, J. G., Gray-Donald, K., Scot, M., Olela, K. N., 1997).

In India, it has been demonstrated that *Ascaris lumbricoides* causes several intestinal and respiratory symptoms and plays an important role in precipitating protein-energy malnutrition in undernourished children, while hookworm causes anaemia and hypoproteinaemia (Ananthakrishnan, S., Nalini, P., Pani, S. P., 1997).

In Gambia, no significant difference in respiratory quotient between children with low level helminth infections and helminth infection free children has been found suggesting that low level of helminth infection

does not significantly affect the energy metabolism (Stettler, N., Schutz, Y., Jequier, E., 1998). However, mild undernutrition and geohelminth infections do have an effect on school achievements (Hutchinson, S. E., Powell, C. A., Walker, S. P., Chang, S. M., Grantham-McGregor, S. M., 1997).

Intestinal helminths are so named because their life history includes a period of obligatory residence in the human alimentary tract or because they induce pathological changes in that site. Not surprisingly, nutritional impairment is often associated with chronic intestinal helminthiases. Estimates of the global prevalence of the intestinal nematode infections transmitted through soil are as follows: 1000 million cases of *Ascaris lumbricoides*; 900 million cases of hookworm; and 500 million cases of *Trichuris trichiura* (Warren, K. S., Mahmoud, A. A. F., 1984). Ascaris infestation on re-estimation, is found to be 1300 million with 59 million at risk of some morbidity mostly in children and approximately 10 000 deaths (de Silva, N. R., Chan, M. S., Bundy, D. A., 1997). Figures for the percentage prevalence in some of the counties of the world are also available. In Nicaragua in the city of Leon, the overall prevalence of pathogenic parasites has been estimated as being 47.2%, and that of *Entamoeba histolytica/dispar* as 18.6%, *Giardia lamblia* 15.9%, and *Ascaris lumbricoides* 13.4%. Hookworms and strongyloides are at low rate

(Tellez, A., Morales, W., Rivera, T., Meyer, E., Leiva, B., Linder, E., 1997). In Uganda the prevalence of *Trichuris trichura* is 28%, *Ascaris lumbricoides* 17%, and hookworms 12.9%. Other less common parasites found were *Schistosoma mansoni, Strongyloides stercoralis, Taenia spp., Enterobius vermicularis, Giardia lamblia* and *Entamoeba* (Kabatereine, N. B., Kemijumbi, J., Kazibwe, F., Onapa, A. W., 1997). Among the Orang Asli (Aborigines) children of Malaysia, the overall prevalence of *Ascaris lumbricoides, Trichuris trichiura*, and hookworms are 62.9%, 91.7% and 28% respectively (Norhayati, M., Zainudin, B., Mohammod, C. G., Oothuman, P., Azizi, O., Fatma, M. S., 1997). In the city of Abidjan in the Ivory Coast, the prevalence of *Ascaris lumbricoides, Trichuris trichiura, Necator americanus, Stongyloides stercoralis, Hymenolepis nana, Schistosoma mansoni*, and *Enterobius vermicularis* is 15.5%, 23.4%, 6.3%, 1.4%, 1.1%, 0.8% and 0.2% respectively (Menan, E. L., Nebavi, N. G., Adjetey, T. a., Assavo, N. N., Kiki-Barro, P. C., Kone, M., 1997). In School children in Zanzibar, Tanzania, the prevalence of infections has been reported as being 72% for *Ascaris lumbricoides*, 94% for *Trichuris trichiura*, and 96% for Hookworms (Albonico, M., Chwaya, H. M., Montresor, A., Stolfzfus, R. J., Tielsch, J. M., Alawi, K. S., Savioli, L., 1997). In Laos, the prevalence of helminth has been reported as being 75.9% with 43.8% of *Opisthorchis viverrini* (liver flukes), 26.3% of *Ascaris lumbricoides*, 19% of *Trichuris trichiura*, 19% of hookworms, 2.2% of

Strongyloides stercoralis, 0.7% of Taenia spp., and 1.5% of *Schistosoma mekongi* (Chai, J. Y., Hongvathong, B., 1998).

Prevalence of the parasites in Mauritius is not known as no research has been carried out during the recent past and no publications are available except for statistical reports on patients calling at hospitals. This study has shown the prevalence of the gut parasites in children in the impoverished regions of Mauritius as being 27.8% for *Ascaris lumbricoides* , 39.8% for *Trichuris trichiura*, 5.6% for Hookworm, 2.1% for *Entamoeba histolytic*, 5.5 % for *Giardia lamblia*, 0.7% for *Entamoeba coli*, 0.3% for *Chilomastix mesnili*, 2.1 % for *Endolinax nana,* 2.3 % for *Enterobius vermicularis*, and 0.7 % for*Strongyloides stercoralis.* When compared to rich countries like Malaysia, once again we see that 'Mauritius is managing success'. In Malaysia, the prevalence has been reported as being 62.9% for *Ascaris lumbricoides*, 91.7% for *Trichuris trichiura* and 28 % for hookworm in children in one of the tribes (Norhayati, M., Zainudin, B., Mohammod, C. G., Oothuman, P., Azizi, O., Fatma, M. S., 1997). Mauritius also performs better compared to Tanzania (72% *Ascaris lumbricoides*, 96% hookworms, and 94% *Trichuris trichiura*).

While looking at prevalence it is also relevant to note that sex differences in parasite infection rates, intensities, or population patterns are common in a wide range of taxa. These differences are usually attributed to differential exposure to pathogens because of sex-specific behaviour or morphology, and the well-documented association between testosterone and the immune system (Zuk, M., McKean, K. A., 1996). Similarly, in Madagascar it has been found that exposure and infection are ubiquitous and that intensity is influenced by gender-related behavioural and environmental factors (Kightlinger, L. K., Seed, J. R., Kightlinger, M. B., 1998). In this study, it has been seen that for almost every parasite encountered in the children in the impoverished regions of Mauritius, the prevalence is more in males than females (Fig. 18), except for combined infections like hookworm and ascaris and ascaris and trichuris. When we look at the biology of hookworm and at its life cycle, we see that the larvae of hookworm mature in the soils and that man is infected on contact with soil, when the infective larvae penetrate the unbroken skin. Most of the girls tend to remain inside their home playing the role of mothers and teachers. As such they are less exposed to the infection as opposed to males who play outside their home sometimes, bare foot. It is therefore not surprising that more males than females carry the infection. However, when it comes to trichuris, we are not able to explain how the females are less affected bacause both ascaris and trichuris have the same mode of

transmission. One plausible explanation could be the prevalence of the parasite in the environment. If there are comparatively few ova in the environment, then obviously it is going to be the least encountered one. There might be a need to further investigate this theory. There is also a need to investigate if there is any predisposition to the individual parasites and if so, to what extent. We already know that there is a higher prevalence and intensity of infections with ascaris and trichuris in urban districts when compared to rural areas (Curtale, F., Shamy, M. Y., Zaki, A., Abdel-Fattah, M, Rocchi, G., 1998), and we also know that there is no association between parasites like giardia, ascaris and hookworm (Utzinger, J., N'Goran, E. K., Marti, H. P., Tanner, M., Lengeler, C., 1999).

From studies carried out in New York (Gorey, K.M., Vena, J. E., 1995), we have seen that poverty is directly associated with incidence of cancer. In another study in Malaysia, it has been shown that in children with cancer, intestinal parasites precipitate febrile condition with diarrhoea. It has further been shown that at least 42 % of the children with cancer carry one parasite or the other (Menon, B. S., Abdullah, M. S., Mahamud, F., Singh, B., 1999). This type of study was beyond the scope of this present study, it would however be interesting to learn if poverty in Mauritius precipitates cancer and how parasites behave in these subjects.

Studies of intestinal parasites in primary school children are relatively common (WHO 1990b) (WHO 1992a). Accordingg to Bundy et al, studies that have included young people show that the age pattern of prevalence differs between the different gut parasites (Bundy, D. A. P., Hall, A., Medley, G. F., Savioli, L., 1992). However, in the impoverished regions of Mauritius, we see that for parasites like ascaris and trichuris there do no seem to be an age pattern among the children, although there are indications that *Giardia lamblia* seems to be more prevalent in the younger groups where as hookworm starts appearing as from the age of 6 (Fig. 19). If there is a predisposition to infection other than age, then there may not be an age pattern for prevalence. It is going to be quite useful to investigate this aspect of parasitic infections.

Other helminth infections associated with the intestinal tract that are less widespread in man includes *Hymenolepis nana, Taenia saginata,Taenia solium, Faciolopsis buski, Angiostrongylus costaricensis* and *Capillaria philipensis* (Marsden, P. D., 1978). None of these was intercepted during this study.

Infections of the human intestinal tract with pathogenic protozoa are a common cause of diarrhoea and have world wide distribution with potentially fatal complications and giardia causing malabsorption in

children (Martinez – Palomo, A. et al 1986), (WHO 1985). *Giardia lamblia* may be the most common intestinal protozoan in the world with a wider range of symptoms (Garcia, L. S., 1999). Giardia species are very old species. In South America, they are being identified and studied from 3,000 years old mummies (Allison, M. J., Bergman, T, Gersten, E., 1999).

Infections with parasites in general have several implications. For instance, studies in Zanzibari school children have shown that infections with malaria, *Trichuris trichiura, Ascaris lumbricoides*, and hookworms were all associated with worse iron status; the association with hookworms being strongest by far (Stoltzfus, R. J, Chwaya, H. M., Tielsch, J. M., Schulze, K. J., Albonico, M., Savioli, R., 1997). In another study a steady fall in levels of haemoglobin as well as packed cells volume (PCV) level was observed with increasing number of infections (Selvam, R., Baskaran, G., 1996). Placental malaria infection has been found to be the strongest risk factor for anaemia in six-month-old children and are independent of the frequency of parasitaemia (Cornet, M., Le Hesran, J. Y., Fievet, N., Cot, M., Personne, P., Gounoue, R., Beyeme, M., Deloron, P., 1998). To make thing more complicated, strongyloides hyperinfection have been found to be highly associated with human T cell lymphotropic virus (Gotuzzo et al, 1999).

Anaemia in children as such and in particular iron deficiency anaemia, is not only associated with parasitic infections but also with nutritional status, such that 84% of hookworm positive children and 75% of hookworm negative children in an Aboriginal community in the north of Western Australia were anaemic (Hopkins, R. m., Gracey, M. S., Hobbs, R. P., Spargo, R. M., Yates, M., Thompson, R. C.,1997).

Apart from anaemia and iron deficiency, it is believed that parasitic infections may cause a change in the number of white blood cells (WBC) (Dos Santos, J. I., Vituri, C. de L., 1996) and packed cells volume (PCV) (Selvam, R., Baskaran, G., 1996). It is therefore not surprising that *Giardia lamblia* infection has been found to be associated with the secretion of some kind of parasite allergen(s) thereby causing an increase in white blood cells counts particularly of eosinophils (Dos Santos, J. I., Vituri, C. de L., 1996). Nowadays, it is believed that in cases of anaemia as a result of malaria, a robust eosinophilic response shortly after completing therapy has been found to predict a good recovery (Camacho, L., H., et al 1999).

In this study, we have observed a marked increase in the number of WBC /ml with number of types of parasites. This can be seen in table 44. This is contrary to the findings of Dos Santos and Vituri (Dos Santos, J. I., Vituri, C. de L., 1996). Dos Santos and Vituri carried a study with *Giardia*

lamblia and found no change in absolute number of lymphocyte counts when compared to a Giardia free control. When the giardiasis and control groups were separated by paediatric (0-18 years) and adult (older that 18 years) classes, a very significant difference in both relative and absolute number of eosinophils in adult class was observed. With respect to the paediatric class, no differences, either in relative and absolute number of eosinophils could be observed. In this study we were dealing with paediatrics yet we observe an increase in number of leucocyte counts. Increase in number of leucocytes was also found in Germany where they have even recommended the monitoring of helminth infection eosinophil-derived neutrotoxin in the urine (Tischendorf, F. W., Brattig, N. W., Burchard, G. D., Kubica, T., Kreuzpainter, G., Lintzel, M., 1999).

While examining poverty it has been seen that the undernourished child has less resistance to infection so that even the most trivial illness tends to become severe. Also, each episode of infection causes a further deterioration of nutritional status resulting finally in malnutrition thus causing a vicious circle. It is now a fact that the economic crisis has positively led to the onset of several infectious diseases among communities with social deprivation (Poinsignon, Y., Marjanovic, Z., Farge, D., 1996). But then do all the children living in a specific community with the same environmental exposure carry the same infections?

Certainly not! The question is why and how? In fact, overdispersion as has been observed, to be a common feature of population distribution patterns. What about infections then? Only a fraction of the exposed population is affected. This implies that some of the people exposed to the infection do not contract the disease. Can we not conclude that some factors should be responsible for an increased susceptibility to infection or to an increased inhibition of infection.

Indeed, it has been shown that susceptibility may be attributable to genetic factors warranting further investigation (Williams-Blangero, S., Blangero, J., Bradley, M., 1997). One of the genetic factors, which was investigated in this study was blood group.

Nowadays a great deal has been learned about the biologic function of structures bearing blood group antigens. Some blood group antigen-bearing proteins function as major transport channels within the erythrocyte membrane; these include the anion transporter (band 3: Diego and Wright antigens), the water channels (aquaporin: Colton antigens), and the urea transporter (Kidd antigens). At least two erythrocyte blood group antigen proteins have complement regulatory functions: the complement receptor type 1 (CR1, CD35: Knops antigens) and decay accelarating factor (DAF, CD55: Chromer antigens). Some blood group

antigens reside on proteins with known receptor functions, such as the chemokine receptor (duffy) and the hyaluronan receptor (Indian). The Cartwright antigens reside on an enzyme, acetylcholinesterase, and the Kell antigens reside on a protein that belongs to the CALLA-related family of neutral metalloproteinases. Finally, some blood group antigens reside on proteins that serve crucial structural functions necessary to normal erythrocyte lifespan and morphology. These proteins include band 3, glycophorins C/D (bearing the Gerbich antigens), and the Rhesus proteins. Both oligosaccharide and protein blood group antigens may act as receptor for bacterial, viral, and parasitic infectious agents (Mudad, R., Telen M. J., 1996). Similarly blood sub group Lea-b- is believed to have a genetic predisposition to recurrent vulvovaginitis (Hilton, E., Chandrasekaran, V., Rindos, P., Isenberg, H. D., 1995) and blood group A to *Pseudomonas aeruginosa* infections (Steuer, M. K., Hofstadter, F., Probster, L., Beuth, J., Strutz, J., 1995). Other blood group antigens are found to act as epithelial cell receptors for *Candida albicans* (Cameron, B. J., Douglas, L. J., 1996).

It is suspected that blood group does not only predispose humans to certain infections, but there are cases where it inhibits certain infections. Some studies have shown that people with blood group AB are less prone

to malarial infection (Singh, N., Shukla, M. M., Uniyal, V. P., Sharma, V. P., 1995).

In the sampled population, as many as 155 children were free from parasitic infections, or 104 (155-51) children in the impoverished regions were free from parasites (300 children forming part of the samples 51 of whom form part of the control and did not come from impoverished regions leaving us with 249 children from the selected four impoverished regions). This implies that a total of 145 child out of a total of 249 carried one infection or the other (table 18). The percentage of children with parasitic infection therefore is 58.2. This also means that 41.8% of the children in the impoverished regions are free from any parasitic infection. We observed that all the children living in these communities have approximately the same type of exposure to infections and therefore equal chances to carry all of the parasitic infections present in the environment. Also, all the children living in these communities have almost the same type of exposure to food availability and to other environmental factors. There are at least 48% of the children in the community who behaved differently to exposure to the infection. This strenghthens the theory of predisposition whereby where several children are exposed to a certain infection not all of them catch the disease but only a few who have certain predisposition to the infection. Blood group has been found to be a factor

predisposing/inhibiting human to infection (Mudad, R., Telen M. J., 1996; Singh, N., Shukla, M. M., Uniyal, V. P., Sharma, V. P., 1995). We carried additional investigations and our findings are tabulated in Table 53. The observation we made suggests that children with blood group A and blood group O in the sample are predisposed to parasitic infections and those with blood group AB are not susceptible to the infection. When looking at the results of blood group, we find that there are as many as 83 children with blood group B (table 50), and only 66 Children of blood group A. So, although there are more children with blood group B than with blood group A, most of the infections are with blood group A children. There is also another school of thought. Glickman et al believes that geophagia should be another cause and an important risk factor for orally acquired nematode infections (Glickman, L. T., Camara, A. O., Glickman, N. W., McCabe, G. P., 1999).

We now know that parasitic infections cause a number of complications, one of which is storage iron depletion. In fact, the original interest was to investigate if there is any iron depletion as a result of poverty and also as a result of parasitic infections, and ultimately to see if one or the other aggravates the situation. To examine iron stores requires sampling one of two principal storage depots, the bone marrow or the liver. Aspiration and biopsy of marrow are usually preferred because they are safer and the

technique is more familiar to haematologists (Ludin, P., 1964). However, for iron stores, determination of serum ferritin concentration is in fact the method of choice (Finch et. al, 1986). Although it is an indirect measure, it has the advantage over biopsy procedures of being more quantitative as well as non-invasive and therefore less expensive and more acceptable to patients (in this case participants). Immunological methods for measuring the tiny quantities of ferritin in plasma have been available since the early 1970s (Worwood, M., 1979; Alfrey, C. P., 1978). While the mean normal values are 90 μg/L for male and 35 μg/L for female, infants' values are high at birth but rapidly fall to about 30 μg/L where they remain until puberty. Diurnal variations of about 15% are mainly due to methodological factors (Worwood, M., 1979). Normally values of 12 μg/L establish a diagnosis of iron deficiency, but one cannot be certain that iron stores are sufficient unless the value is greater than 50 to 60 μg/L (Finch, C. A., et al 1986; Hansen T. m., et al 1983).

Mauritius has achieved tremendous progress since independence in 1968. The economy has been diversified and strengthened by rapid industrialisation and by the expansion of tourism. These developments have brought unquestionable benefits. Unemployment at the national level has fallen to negligible levels, so much so that several thousands of workers from China, India and Sri Lanka are currently employed in the

textile industry, in agriculture and in the telecommunications. A number of new schools and a few hospitals have been built and equipped. The housing situation has been improved with the construction of houses and apartments for sale to middle and low-income groups. Infrastructural improvements have also been made across the island. Overall, it may be said that these developments have resulted in higher standards of living for a majority of Mauritians.

Unfortunately, however as we saw in this study, there is indeed another side to this picture. We now know that significant numbers of Mauritians are only marginally touched by all this progress. They are being left behind. Many of them have insecure home backgrounds. A high proportions of them are illiterate or near illiterate and certainly has no academic qualifications. Those who are not unemployed have low income or unstable jobs. They generally have poor lodgings and living conditions.

This work focussed on the children living in these areas. The starting point of the inquiry being intestinal parasitic infections is among the most common infections in the poorest communities (Savioli, L., Bundy, D., Tomkins, A., 1992). And that iron deficiency anaemia ranging from mild to life threatening is an inevitable result of inadequate dietary iron intake and this is further aggravated by parasitic infections (WHO, 1990; Cooper et

al., 1990) because parasitic infections also precipitate iron deficiency on its own.

We know that poverty rarely exists by itself. Low wages and lack of adequate resources are usually associated with insecurity of employment, illiteracy, bad housing, large families, exploitation and a host of other factors.

We observed that at least 14.8% of the households in the impoverished regions have no earners, and of the remaining 85.2% as many as 47.5% of the households have only one earner in the family (At table 9). This implies that 61 – 62 % of the households are operating at a very tight schedule with limited resources. This matches perfectly with the above definitions of poverty (Low wages and lack of adequate resources are usually associated with insecurity of employment, illiteracy, bad housing, large families, exploitation etc.). It should therefore be expected that at least 62% of the inhabitants are extremely poor with all the implications of poverty.

In the impoverished regions, the majority of the workers are in elementary occupations (table 4), and this is also true for the women as well (table 5). The mean salary of a person in the elementary occupations group, is Rs.

3524 (table 7). Also, if you are a female in this group, then the average salary is only Rs. 2743 (table 8). Therefore depending upon the number persons in the family, it is very likely that most of the families in the impoverished regions are ultra poor. Fortunately, based on the poverty line for Mauritius, below which household members consume less than 50% of the mean consumption level, as little as 22% of the households and 29% of the individuals in the surveyed areas may be classified as poor. Similarly only 6% of the surveyed population may be classified as ultra poor.

Not surprising at all, most of the poor and more specifically the ultra poor are squatters. They all live in crowded condition and have less access to modern sanitation system and water supply inside their home.

It is therefore expected that almost every person living in these areas should be anaemic and iron deficient and the majority should be habouring at least one of the species of parasites.

Fortunately, for Mauritius, this hypothesis does not hold good. We see that the mean Hb in g/dl for uninfected children in the impoverished regions of Mauritius is 12.43 g/dl (table 29), and 12.97 g/dl for control (table 23) or for children from regions other than the impoverished areas. There does not

seem to be any difference between the two means. This implies that non infected children from poor families enjoy the same haemoglobin concentration as non-infected children from rich families. In the impoverished regions of Mauritius, we see that as the number of types of parasites increases the Hb in g/dl decreases (table 28 & table 29). This is also true for Packed Cells Volume of blood (table 33). We can say that parasitic infection precipitates anaemia in children in the impoverished regions of Mauritius. This result reflects those found by Selvam and Baskaram. Selvam and Baskaram, carried studies to assay the haematological parameters in *Plasmodium vivax* patients with only one infection, two infections, three infections and more than three malarial infections during a period of six months. They observed a steady fall in the levels of haemoglobin as well as packed cells volume (PCV) level with increasing number of infections (Selvam R., Baskaran, G., 1996). This study has demonstrated that this is also true with gut parasites in the impoverished regions of Mauritius.

As regards to storage iron depletion, again we see that as the number of types of parasites increases plasma ferritin level decreases, and this even within the normal limits (table 58). This implies that, when infected with several types of parasites, there are some forms of synergistic effects in

iron depletion, such that the ferritin level decreases as the number of type of infection increases.

While evaluating plasma ferritin concontration level, we saw that, the mean normal values were 90 μg/L for male and 35 μg/L for female, and that for infants, irrespective of sex, the values were high at birth but rapidly fall to about 30 μg/L where they remain until puberty (Worwood, M., 1979). It is believed that normally value of 12 μg/L establishes a diagnosis of iron deficiency, but iron stores are considered insufficient unless the value is greater than 50 to 60 μg/L (Finch, C. A., et al 1986; Hansen T. M., et Al 1983). In the present study, we were able to see that the mean ferritin value for the control is about 140 μg/L. Normally, it would have been best to consider the normal ferritin value for children in Mauritius as being 140 μg/L, which is in line with the studies of Worwood who found that while the mean normal values are 90 μg/L for males and 35 μg/L for females, infants' values are higher. However, iron stores are considered insufficient unless the value is greater than 50 to 60 μg/L (Finch, C. A., et al 1986; Hansen T. M., et Al 1983). For infants and children one should consider values higher than 50 to 60 μg/L as indicative of iron depletion. And for this study restricting us to the recommendations of Finch, C. A., et al; 1986 or 50 – 60 μg/L, the value for children may be extended to 70 μg/L. Table 55 demonstrate that there does not seen to be iron deficiency in any

of the impoverished areas selected so does figure 20. Mauritius is comparatively a small island. While it is surrounded by the sea, even inland, there are many of rivers. The main pastime of many of the adult males, particularly those belonging to poor families, is fishing. This means that where nutrition is concerned, there is an interrupted supply of quality proteins. One of the striking features of the slum-like huts seen in figure13 is the inclusion of a television antaena. In fact, almost all of the houses in the impoverished areas possess a television. We are not sure if people will want to substitute food for television sets. On the contrary, table 54, shows that the mean ferritin level for the children in Mauritius is 106 μg/L and even this increases with age (table57).

So we see that, whether impoverished or not, children in Mauritius in general, do not seem to suffer from iron deficiency. Of course the picture is slightly different for children with parasitic infections (table 58). Those children who carry 3 or 4 different types of parasites do have iron deficiency, which is reflected by plasma ferritin of less that 70 μg/L. With hookworm infection infact there are marked storage iron depletion with a mean mean ferritin of 23 μg/L.

The iron status of the children in the impoverished regions of Mauritius is more clearly depicted in figure 20. The bar chart shows that the ferritin

status for normal children (from rich families) is almost the same as that of poor children with no parasites. Additionally we see that children with ascaris do not have any change in plasma ferritin when compared with children with no parasite and therefore also with children from rich families. Ascaris do not even influence any ferritin depletion when present along with other parasites, suggesting that there is no sinergistic effect either.

There is marked iron depletion in children with trichuris and a relatively marked depletion with giardia when compared to control. Depletion is extremely marked with hookworm infestations, a finding compatible with those of many scientists including including that of Brooker et al (1999). The biology of the parasite demonstrates that the adult attaches to the villi of the small intestine and suck blood. Iron deficiency anaemia occurs when following the feeding activities of fourth stage (L4) larva and adult worms, there is intestinal capillary blood loss (Georgiev, V. S., 1999).

The findings however are far different from those of Tsuyoka et al (1999). Tsuyoka et al carried experiments in Brazil with the Liverpool school of Tropical Medicine, in the UK and found that intestinal were not at all associated with anaemia (Tsuyuoka, R., Bailey, J. W, d'Avila, A. M., Guimaraes, N., Gurgel, R. Q., Cuevas L. E., 1999) and also from those of

Triteeraprapah and Nuchproyoon (1998). Triteeraprapah and Nuchproyoon worked in Thailand. He is of opinion that Hookworm infection is significantly associated with eosinophilia but not anaemia nor microcytosis of red cells (Triteeraprapab, S., Nuchprayoon, I., 1998).

Malaria in Mauritius was certified eradicated by WHO in 1973 after 8 consecutive years of interruption of Malaria transmission. According to Dr Benzerroug, the Regional Malaria Adviser for WHO, the disease was re-introduced in 1975 following a cyclone and relaxation of surveillance mechanism. The situation reached epidemic proportion in 1981/2. After 15 years of continuous effort the transmission was again interrupted and reached a favourable situation in 1990 and 1991. In 1992 a deterioration of the situation was observed and 16 indigenous cases were recorded (Benzerroug, E. H., Ravaonjanaharry, C., 1992).

In Mauritius there is widespread presence of *Anopheles arabiensis*, an efficient malaria vector. Also, visitors are constantly introducing malaria parasites in the country. There are fishermen working in fishing vessel and nobody knows what risk these Mauritian fishermen carry. To make matters worse, nowadays, there are foreign workers particularly from malaria endemic areas like Sri Lanka working permanently in Mauritian textile industries and in construction. We have seen in chapter 1 that

relapses in malaria occur because of different rates of development of pre-erythrocytic schizonts resulting in a portion of parasites to enter a dormant phase (hypnozoites) (Bruce-Chwat, L. J., 1985). For *Plasmodium malariae*, the persistence of infection may reach 30 years due to survival of the erythrocytic stages in small numbers, which explains the recrudescences that occur over many years (Cheesbrough, M. 1987).

It is therefore always very useful to include malaria in this type of research although the latter is very tedious and time consuming. In villages like La Pipe, where most of the heads of household are fishermen working in big vessels and remaining outside Mauritius for weeks, interception of some cases of malaria was expected. Fortunately there were none.

4.2 FINAL CONCLUSION

To conclude, it can be said that this research has demonstrated:

- the presence of pockets of poverty throughout the island of Mauritius,
- that even in poverty striken areas, a proper diet can prevent the development of the most undesirable complication of iron deficiency anaemia

- that blood group antigen precipitates parasitic infections such that in Mauritius, children with blood group "A" and blood group "O" are more prone to parasitic infections
- that when infected with several species of parasites at one time, haemoglobin concentration, packed cell volume and plasma ferritin concentration are significantly lowered suggesting a synergistic effect among the parasites, and that this synergistic effect is absent when one of the parasites is ascaris
- that hookworm infection alone causes marked storage iron depletion, followed by trichuris and giardia,
- that giardia is more prevalent in the younger segment of the population whereas hookworms start appearing at the age of six years
- that giardia causes white blood cell counts to increase, and that for almost every parasite in Mauritius, the prevalence is greater in males than in females.

It is therefore desirable that new research be carried to investigate:

- If parasitic infections are precipitataed as a result of geophagia instead of susceptible blood group factors,
- If, since children in the impoverished regions of Mauritius are not malnourished as demonstrated by their plasma ferrittin level,

children do have their growth hampered as a result of poverty in Mauritius,

- If poverty precipitates cancer as in New York, and if yes, does cancer precipitate parasitic infections as in Malaysia?

Finally, as remedial action, it can be suggested that children should be encouraged to take antihelminthics, and antiprotozoals in dosage sufficient to kill the hardiest parasites, regularly.

REFERENCES

Addison, G. M., Beamish, M. R., Hales, C. N., Hodkins, M., Jacobs, A., Llewellin, P. An immuno radiometric assay for ferritin in the serum of normal subjects and patients with iron deficiency and iron overload. J Clin Pathol 1972;25:326-329.

Adelekan, D. A., Adeodu, O. O., Thurnham, D. I. Comparative effects of malaria and malnutrition on plasma concentrations of antioxidant micronutrients in children. Ann Trop Paediatr 1997;17(3):223-7.

Albonico, M., Chwaya, H. M., Montresor, A., Stolfzfus, R. J., Tielsch, J. M., Alawi, K. S., Savioli, L. Parasitic infections in Pemba Island school children. Aest Afr Med J 1997;74(5):294-8.

Albonico, M., Savioli, L. Hookworm infection and disease:advances for control. Ann Ist Super Sanita. 1997;33(4):567-79.

Alfrey, C. P. Serum ferritin assay. CRC Crit Rev Clin Lab Sci. 1978;9:179.

Allison, M. J., Bergman, T., Gerszten, E. Further studies on fecal parasites in antiquity. Am J Clin Pathol 1999;112(5):605-9.

Ananthakrishnan, S., Nalini, P., Pani, S.P. Intestinal geohelminthiasis in Developing world. Natl Med J India 1997;10(2):67-71.

Anderson, R. M. The population dynamics and epidemiology of intestinal nematode infections. Transaction of the Royal Society of Tropical Medicine and Hygiene 1986;80:686-696.

Anon. Malaria and immunology. (Editorial) Lancet 1978;2:974-5.

Baker, S. J., Nutritional anaemia – a major controllable public health problem. Bulletin of World Health Organisation 1978; 56: 659-75.

Barker, R. H., Suebsaeng, L., Rooney, W., Alecrim, G. C., Dourado, H. V., 7 Wirth, D. F. Specific DNA probe for the diagnosis of *Plasmodium falciparum* malaria. Science 1986;231:1434-1436.

Beck, J. R., Cornwell, G. G., French, E. E., et al. The 'iron screen ': Modification of standard laboratory practice with data analysis. Human Pathology 1981;12:118-126.

Bellamy, C. The State of the World's Children 1998, Unicef Publication, UK.

Benzerroug, E. H., Ravaonjanaharry, C. Report on WHO mission to Mauritius 16 – 23 August 1992. WHO AFRO/Brazzaville.

Bheenick, R., Hanoomanjee, E. Mauritius: Towards an Industrial Training Strategy, 1988 Edition de L'Ocean Indien, Mauritius.

Boender, C. A., Verloop, M. C. Iron absorption, iron loss, and iron retention in man. Br J Haematol 1969;17:45.

Bothwell, T. H. The diagnosis of iron deficiency. NZ Med J (Suppl) 1966;65:880.

Bouhoum, K., Schwartzbrod,J. Epidemiological study of intestinal helminthiasis in a Marrakech raw sewage spreading zone. Zentrabl Hyg Umwelmed 1998;200(5-6):553-61.

Bradley, D. J., Newbold, C. j., & Warel, D.A. Malaria. In Oxford Texbook of Medoicine, Weatherall, D. J., Ledingham, J. G. G., & Warell, D. A., (Eds), 1987, 2nd edition. Oxford: Oxford University Press, pp 5.474- 5.502.

Brinks, S., Van Schalkwyk, D. J., Serum ferritin and mean corposcular volume as predictors of bone marrow iron stores. SA Med J 1982;61:432-434.

Brooker, S., Peshu, N., Warn, P. A., Mosobo, M., Guyatt, H. L., Marsh, K., Snow, R. W. The epidemiology of hookworm infection and its contribution to anaemia among pre-school children on the Kenyan coast. Trans R Soc Trop Med Hyg 1999;93(3):240-6.

Bruce-Chwat, L. J. Prevention and treatment of malaria. Trop Doc 1977;7:17-20.

Bruce-Chwat. L. J. Essential Malariology, 1985, 2nd edition. London: William Heinemann, p 104.

Bryant, J. Health and the developing world, Ithaca & London: Cornell Paperbacks, Cornell University Press, 1969.

Bundy, D.A.P., Hall, A., Medley, G.F., Savioli, L. Evaluating measures to control intestinal parasitic infections. Wld. Hlth. Statist. Quart. 1992;45:168-179.

Camacho, L. H., Wilairatana, P., Weiss, G., Mercader, M. A., Brittenham, G. M., Looareesuwan, S., Gordeuk, V. R. The eosinophilic response and haematological recovery after treatment for *Plasmodium falfiparum* malaria. Trop Med Int Health 1999;4(7):471-5.

Cameron, B.J. Douglas, L.J. Blood group glycolipids as epithelial cell receptors for *Candida albicans*. Infect Imun 1996;64(3):891-896.

Central Statistical Office. Household Budget Survey. July 1991 – June 1992. Volume II Analytical Report. 1994, Port Louis, CSO.

Chai, J. Y., Hongvathong, B. A small-scale survey of intestinal helminthic infections among the residents near Pakse, Laos. Korean J Parasitol 1998;36(1):55-8.

Chatterjee, K. D. Parasitology (Protozoology and Helminthology in relation to clinical medicine). 1982, Calcutta: Chatterjee Medical Publishers.

Cheesborough, M. Medical Laboratory Manual for tropical Countries. 1987, Vol 1. ELBS.

Clegg, G.A., Fitton, J.E., Harrison, P.M., Treffry, A. Ferritin: Molecular structure and iron-storage mechanism. Prog Biophys Molec Biol 1987;36, 56-86.

Cook, J. D. Clinical evaluation of iron deficiency. Seminars in Haematology 1982;19:7-18.

Cook, J. D., Lipschitz, D. A., Miles, L. E. M., Finch, C. A. Serum ferritin as a measure of iron stores in normal subjects. Am J Clin Nutr 1974;27:681-687.

Coop, R. L., Kyriazakis, I. Nutrition-parasite interaction. Vet Parasitol 1999;84(3-4):187-204.

Cooper, E. S., Bundy, D. A. P., Macdonald, T. T., Golden, M. N. H. Growth Suppression in the *Trichuris* dysentry syndrome. Eur J Clin Nut 1990;44:138-147.

Cornet, M., Le Hesran, J. Y., Fievet, N., Cot, M., Personne, P., Gounoue, R., Beyeme, M., Deloron, P. Pravalence of and risk factors for anaemia in young children in southern Cameroon. Am J Trop med Hyg 1998;58(5): 606-11.

Court, S. D. M. Child health in a changing community. Br Med J 1971;2: 125-131.

Crewe, W., & Haddock, D. R. W.. Parasites and Human disease. Edward Arnold, 1985, London.

Crichton, R. R., Ferritin: structure, synthesis and function. New Engl J. Med. 1971;284:1413-1422.

Curtale, F., Shamy, M. Y., Zaki, A., Abdel-Fattah, M, Rocchi, G. Different pattern of intestinal helminth infection among young workers in urban and rural areas of Alexandria Governorate, Egypt. Parasitologia 1998; 40(3):251-4.

Da Silva, A. J., Bornay-Llinares, F. J., Moura, I. N., Slemenda, S. B., Tuttle, J. L., Pieniazek, N. J. Fast and reliable extraction of protozoan parasite DNA from fecal specimens. Mol Diag 1999;4(1):57-64.

de Silva, N.R., Chan, M.S., Bundy, D.A. Morbidity and mortality due to Ascaris: re-estimation and sensitivity analysis of global numbers at risk. Trop Med Int Health 1997;2(6): 519-528.

de Silva, N.R., Guyatt, H.L., Bundy, D.A. Worm burden in intestinal obstruction caused by Ascaris lumbricoides. Trop med Int Health 1997; 2(2):189-90.

de silva, N.R., Guyatt, H.L., Bundy, D.A. Morbidity and mortality due to Ascaris-induced intestinal obstruction. Trans Roy Soc Trop Med Hyg 1997;91(1):31-6.

Derman, D.P., Bothwell, T.H., Mc Phail, A.P., Importance of ascorbic acid in the absorption of iron from infant food. Scand J Haematol 1980;25:193-201.

Diez-Ewald, M., Torres-guerra, E., Layrisse, M., Leets, I., Vizcaino, G., Arteaga-Vizcaino. Prevalence of anaemia, iron, folic acid and vitamin B 12 deficiency in two Bari Indian communities from western Venezuela. Invest Clin 1997;38(4):191-201.

Dos Santos, J. I., Vituri, C. de L. Some haematimetric findings in human Giardia lamblia infection. Rev Inst Med Trop Sao Paulo 1996;38(2):91-5.

Draper, A. 'Child Development and Iron Deficiency: Early action is critical for healthy mental, physical, and social development', The Oxford brief, Opportunities for Micronutrient interventions, Washington, D.C. 1997.

Ebrahim, G. J. Epidemiology of iron deficiency anemia in Zanzibari schoolchildren: the importance of Hookworms. Am J Clin Nutr 1997; 65(1):153-9.

Fairbanks, V. F., Klee, G. G. Biochemical aspects of haematology in Fundamentals of Clinical Chemistry , Tietz (Ed), 1987, pp 789-822.

Farnet, A, Snounou, G, Rooth, I Bjorkman, A Daily dynamics of *Plasmodium falciparum* subpopulation. Am J Trop Med Hyg 1997;56(5): 538-47.

Finch, C. A., Cook, J.D. Iron deficiency. Am J Clin Nutr 1984;39: 471 - 472.

Finch, C. A., et al. Plasma ferritin determination as a diagnostic tool. West J Med 1986;145:657.

Finegold, S. M., Baron, E., J. Bailey and Scott's Diagnostic Microbiology . The C.V. Mosby Company 1986, p. 831.

Forman, D. T., Parker, S. L. Measurement and interpretation of serum ferritin. Ann Clin Lab Sci 1980;10:345-350.

Fraser, D. The Evolution of the British Welfare State, Macmillan Press Ltd., England, 1973.

Fraser, D, Dagan, R , Naggan, L, Greene, V., El-On, J., Abu-Rbiah, Y Deckelbaum, R.J. Natural history of *Giardia lamblia* and *Cryptosporidium* infections in a cohort of Israeli Bedouin infants: a study of a population in transition. Am J Trop Med Hyg 1997;57(5): 544-9.

Garcia, L. Flagellates and ciliates. Clin Lab Med 1999;19(3):621-38vii.

Garcia, L. S., & Bruckner, D. A., Diagnostic Medical Parasitology, 2nd edn. American Society for Microbiology, Washington 1997.

Garratty, G. Blood group antigens as tumor markers, parasitic/bacterial/viral receptors, and their association with immunologically important proteins. Immunol Invest 1995;24(1-2):213-32.

Georgiev, V. S. Parasitic infections. Treatment and developmental therapeutics . 1. Necatoriasi. Curr Pharm Des 1999;5(7):545-54.

Geronimus, A. T., Bound, J., Waidman, T. A., Hillemeier, M. M., Burns, P. B. Excess mortality among blacks and whites in the United States. N Engl J Med 1996;335(21):1552-8.

Gilles, H. M., Watson, J., Ball, P. A. J., Hookworm infection and anaemia – an epidemiological, clinical and laboratory study. Quarterly Journal of Medicine 1964;33:1-24.

Glickman, L. T., Camara, A. O., Glickman, N. W., McCabe, G. P. Nematode intestinal parasites of children in rural Guinea, Africa: prevalence and relationship to geophagia. Int J Epidemiol 1999;28(1):169-73.

Gorey, K.M., Vena, J. E. The association of near poverty status with cancer incidence among black and white adults. J Commun Hlth 1995;20(4):359-66.

Gotuzzo, E., Terashima, A., Alvarez, H., Tello, R., Infante, R., Watts, D. M., Freedman, D. O. Am J Trop Med Hyg 1999;60(1):146-9.

Hadju, V., Abadi, K., Stephenson, L. S., Noor, N. N., Mohammed, H. O., Bowman, D. D. Intestinal Helminthiasis, nutritional status, and their relationship; a cross-sectional study n urban slum school children in Indonesia. Southeast Asian J Trop Med Public Health. 1995;26(4):719-29.

Halliday, J. W., Powell, L. W. Serum ferritin and isoferritin in clinical medicine. Prog Haematol 1979;11:229-266.

Hansen, T. M., et al. Serum ferritin and the assesment of iron deficiency in rheumatoid arthritis. Scand J Rheumatol 1983;12:353.

Hansmann Y., Staub-Schmidt T., Christmann, D. Malaria, Falciparum. Trop Med Int Health 1997;2(10):941-52.

Hastka, J., Lasserre, J., Schwarzbeck, A., Reitter, A., Hehlmann, R. Laboratory tests of iron status: correlation or common sense? Clin Chem 1996;42:718-724.

Hazard, J. T., Yokota, M., Arosio, P., Drysale, J. W. Immunological differences in human isoferritins. Blood 1977;49:139-146.

Hazareesing, K., History of Indians in Mauritius, Macmillan Publishers, England 1977.

Heinrich, H. C. Iron deficiency without anaemia. Lancet 1968;2:460.

Hildebrandt, J.P, Malaria – biological aspects of an infectious disease of importance to human, Naturwissenschaften 1996;83(8):359-369 (English abstract).

Hilton, E., Chandrasekaran, V., Rindos, P., Isenberg, H.D., Association of recurrent candidal vaginitis with inheritance of Lewis blood group antigens. J Infect Dis 1995;172(6):1616-9.

Hoffbrand, A. V., Petit, J. E. Essential Haematology. 3rd edn Blackwell Scientific Publications, Oxford, 1993.

Hoffmann, H., Kawooya, M., Esterre, P., Ravaoalimalala, V.E., Roth, J., Thomas, A.K., Roux, J., Seitz, H.M., Doehring, E. In vivo and in vitro studies on the sonographical detection of Ascaris lumbricoides. Mediator Radial 1997;27(3):226-9.

Hopkins, R. M., Gracey, M. S., Hobbs, R. P., Spargo, R. M., Yates, M., Thompson, R. C. The prevalence of Hookworm infection, iron deficiency and anaemia in aboriginal community in north-west Australia. Med J Aust 1997;166(5):214-4.

Hutchinson, S. E., Powell, C. A., Walker, S. P., Chang, S. M., Grantham-McGregor, S. M. Nutrition, anaemia, geohelminth infection and school achievement in rural Jamaican primary school children. Eur J Clin Nutr 1997;51(11):729-35.

Ighogboja, I.S., Ikeh, E. I. Parasitic agent in childhood diarrhoea and malnutrition. West Afr J Med 1997;16(1):36-9.

Jacobs, A., Miller, F., Worwood, M., et al: Ferritin in serum of normal subjects and patient with iron deficiency and iron overload. Br Med J 19172;4:206-208.

Jacobs, A., Worwood, M., Ferritin in serum: clinical and biochemical implications. New Engl J Med 1975;292:951-956.

Jewsbury, J.M., Parasitic infection. The Biology of Disease. 1995 pp77-86.

Jodjana, H., Eblen. Malnutrition, malaria and intestinal worms in young children. World Health Forum, 1997;18(1):21-3.

Jones, H. Social Work in Third World Development, Macmillan Publishers, England, 1990.

Joshi, J.G., Clauberg, M. Ferritin: an iron storage protein with diverse functions. Bio Factors 1988;I:207-212.

Kabatereine, N. B., Kemijumbi, J., Kazibwe, F., Onapa, A. W. Human intestinal parasites in primary school children in Kampala, Uganda. East Afr Med J 1997;74:311-4.

Kalkofen, U. P. Intestinal trauma resulting from feeding activities of Anchylostoma caninum. Am J Trop Med Hyg 1974;23:1046-1053.

Kato, T., Kamoi, R., Lida, M., Kihara, T. Endoscopic diagnosis of hookworm disease of the duodenum. J Clin Gastroenterol 1997; 24(2):100-2.

Katunguka-Rwakishaya ,E., McKennie, D.,Parkins, J.J., Murray, M., Holmes, P.H. Influence of dietyary protein on live bodyweight, degree of anaemia and erythropoietic responses of Scottish blackface sheep infected experimentally with Trypanosoma congolense. Res Vet Sci 1997;63(3):273-7.

Kauffmann, F. Frette, C. Pham, Q.T. Nafisi, S. Bertrand, J.P. Oriol, R. Association of blood group-related antigens to FEV1, wheezing, and asthma. Am J Respir Crit Care Med 1996;153(1):76-82.

Kemp, C. H., Silver, H. k., O'Brien, D., Current pediatric diagnosis and treatment. Los Altos, California: Lange Medical Publication, 1970.

Khari, A., Parija, S.C., Karki, P., Kumar, N. Sonographic diagnosis of intestinal ascariasis. Trop Doct 1998;28(2):117-8.

Kightlinger, L.K., Seed, J.R., Kightlinger, M.B., *Ascaris lumbricoides* intensity in relation to environmental, socioeconomic, and behavioural determinants of exposure to infection in children from southeast Madagascar, J Parasitol 1998;84(3):480-4.

Kotpal, R. L. Protozoa. Rastogi Publications, Meerut, India 1978.

Kurtzhals J.A., Rodrigues, O., Addae, M., Commey, J.O., Nkrumah, F..K., Hviid, L. Reversible suppression of bone marrow response to erythropoietin in Plasmodium falciparum malaria. Br J Haematol 1997; 97(1):169-74.

Lipschitz, D. A., Cook, J. D., Finch, C. A. A clinlcal evaluation of serum ferritin as an index of iron stores. New Engl J Med 1974;290:1213-1216.

Lubanga , R.J., Norman, S., Ewbank, D., Karamagi,C. Maternal diagnosis and treatment of children's fever in endemic malaria zone of uganda: implications for malaria control programme. Acta Trop 1997;68(1):53-64.

Lucas, A. O., Gilles, H. M. Protozoal infections: in A New Short Textbook of Preventive Medicine for the Tropics, pp52-53, ELBS 1994.

Ludin, P. Comparison of haemosiderin estimation in bone marrow sections and bone marrow smears. Acta Med Scand 1964;175:384.

Lujan, H.D, Mowatt M.R, Nash, T.E. Mechanisms of Giardia lamblia differentiation into cysts, Microbiol Mol Biol Rev 1997;61(3):294-304

Macfarlame, L. R. S. A short synopsis of Human Protozoology and Helminthology. E. & S. Livingstone Ltd 1960.

MacGregor, J. D., Avery, J. G. Malaria Trnasmission and foetal growth. Brit med J 1974;3:433-6.

Mahendra Raj, S., Sein, K.T., Khairul Anuar, A., Mustaffa, B.E. Intestinal helminthiasis in relation to height and weight of early primary school children in northeastern peninsular Malaysia. South East Asian J Trop Med Publ Hlth 1997;28(2):314-320.

Mank, T.G., Zaat, J.O., Deelder, A.M., van Eijk, J. T., Polderman, A.M. Sensitivity of microscopy versus enzyme immunoassay in the laboratory diagnosis of giardiasis, Eur J Clin Microbiol Infect Dis 1997;16(8):615-9.

Marsden, P. D. Intestinal parasites. Clin Gastroent 1978;7:1-243.

Martinez – Palomo, A. et al. Amoebiasis. Amsterdam, Elsevier, 1986.

McGregor, I.A. Tropical aspects of the epidemiology of malaria. Israel J Med Sci 1978;14:523-36.

Menan, E. L., Nebavi, N. G., Adjetey, T. a., Assavo, N. N., Kiki-Barro, P. C., Kone, M. Profil des helminthiases intestinales chez les enfants d'age scolaire dand la ville d'Abidjan. Bull Soc Pathol Exot 1997;90(1):51-4.

Menon, B. S., Abdullah, M. S., Mahamud, F., Singh, B. Intestinal parasites in Malaysian children with cancer. J Trop Pediatr 1999;45(4):241-42.

Montgomery, L. E., Kiely, J. L., Pappas, G. The effects of poverty, race, and family structure on US children's health: data from the HHIS, 1978 through 1980 and 1989 through 1991. Am J Pub Health 1996;86(10): 1401-5.

Mookkerji, S.B. Indians in Mauritius (1842-1870), Indian International Quarterly, 1959;32.

Mudad, R., Telen, M.J. Biologic functions of blood group antigens. Current Opinion in Haematology 1996;3(6):473-479.

Muller, R., Baker, J. R. Medical Parasitology. Lippincott, Philadelphia & Gower Medical, 1990, London.

Ndamba, J., Gomo, E., Nyazena, N., Makaza, N., Kaondera, K. C. Schistosomiasis infection in relation to ABO blood groups among school children in Zimbabwe. Acta Trop (Netherlands) 1997;65(3):181 –190.

Newton, C.R., Warn, P.A., Winstanley, P.A., Peshu, N., Snow, R.W., Pasvol, G., Marsh, K. Severe anaemia in children living in malaria endemic area of kenya. Trop Med Int Health 1997;2(2):165-78.

Noel, Karl. L'Esclavage a L'Isle de France, Editions Two Cities, Paris, 1991.

Norhayati, M., Zainudin, B., Mohammod, C. G., Oothuman, P., Azizi, O., Fatma, M. S. The prevalence of Trichuris, Ascaris, and hookworm infection in Orang Asli Children. Southeast Asian J Trop Med Public Health, 1997;28(1):161-168.

Nwulia, M.D.E. The History of Slavery in Mauritius and the Seychelles: 1810 – 1875, Associated university Press, London 1981.

Oski, F.A. Iron deficiency in infancy and childhood. New Engl J Med 1993;329(3):190-193.

Panjarathinam, R. Textbook of Medical Parasitology. Orient Longman 1990.

Payment, P., Berte, A., Fleury, C. Sources of variation in isolation rate of *Giardia lamblia* cysts and their homogeneous distribution in river water entering a water treatment plant. Can J Microbiol 1997;43(7):687-9.

Pelletier, D. L., Frongillo, E. A., Habicht, J. P. Epidemiologic evidence for a potentiating effect of malnutrition on child mortality. Am J Public Health 1993;83:1130-3.

Pelletier, D. L., Frongillo, E. A., Schroeder, D. G., Habicht, J. P. The effects of malnutriton on child mortality in developing countries. Bull WHO 1995;73:443-8.

Peng, W., Zhou, X., Cui, X., Crompton, D.W., Whitehead, R.R., Xiong, J., Wu, H., Yang, Y., Wu, W., Xu, K., Yan, Y. Transmission and natural regulation of infection with *Ascaris lumbricoides* in a rural community in China. J Parasitol 1998;84(2):258-258.

Perera, S.A., Murray, P.G., General principles of infection. The Biology of Disease 1995:47-9.

Petersen, K.M., Parkinson, A.J., Nobbmann, E.D., Bulkow, L., Yip, R., Mokdad, A. Iron deficiency anemia among Alaska natives may be due to fecal loss rather than inadequate intake. J Nutr 1996;126(11):2774-83.

Phillips, J. et al. The Biology of disease. Blackwell Science Ltd. 1995.

Poinsignon, Y., Marjanovic, Z., Farge, D. Maladies infectieuses nouvelles et resurgentes liées a la pauvreté (New and resurgent infectious diseases associated with poverty). Rev Prat 1996;46(15):1827-38.

Pritchard, D.I. Quinell, R.J. Moustafa, M. McKean, P.G. Slater, A.F.G. Raiko, A.. Dale, D.D.S. Keymer, A.E. Hookworm (*Necator americanus*) infection and storage iron depletion. Transaction of Royal Society of tropical Medicine and Hygiene 1991;85:235-238.

Rivera-Matos, I.R., Atkins, J.T., Doerr, C.A., White, A.C. Jr. Pediatric malaria in Houstom, Texas. Am J Trop Med Hyg 1997;57(5):560-3.

Roche, M., Layrisse, M. The nature and causes of "Hookworm anaemia." American Journal of Tropical Medicine and Hygiene. 1966;15:1031-1102.

Saidi, S.M., Lijima, Y., Sang, W.K., Mwangudza, A.K., Oundo, J.O., Taga,K., Aihara,M., Nagayama, K., Yamamoto,H., Waiyaki, P.G., Honda, T. Epidemiological study on infectious diarrheal diseases in children in a costal rural area of Kenya, Microbiol Immunol 1997;41(10):773-8.

Salman, A.B. Management of intestinal obstruction caused by ascariasis. J Pediatr Surg 1997;32(4):585-7.

Santiso, R. Effects of Chronic Parasitosis on Women Health. Int J Gynaecol Obstet 1997;58(1):129-36.

Sarinas, P.S., Chitkara, R.K. Semin Respir Infect Stanford University Medical Centre, CA, USA 1997;12(2):130-137.

Savioli, L., Bundy, D., Tomkins, A. Intestinal parasitic infections: a soluble public health problem. Trans Roy Soc Trop Med Hygi. 1992;86:353-354.

Schulman, A. Ultrasound appearance of intra- and extrahepatic biliary ascariasis. Abdom Imaging 1998;23(1):60-6.

Scrimshaw, N. S., Taylor, C. E., Gordon, J. E. Interaction of nutrition and infection. World Health Organisation. (Monograph series 57), 1968.

Selvam, R., Baskaran, G. Haematological impairments in recurrent Plasmodium vivax infected patients. Jpn J Med Sci Biol 1996;49(4):151-65.

Sing, N. Shukla, M.M., Uniyal, V.P. Sharma, V.P. ABO blood group among malaria cases from district Mandla, MP. Ind J Malariol 1995;32(2): 59-63.

Singh, P.A., Gupta, S.C., Agrawal, R. *Ascaris lumbricoides* appendicitis in the tropics. Trop Doct 1997;27(4):241.

Skikne, B. S., Cook, J. D. Serum ferritin in the evaluation of iron status. Lab Management 1981;19:31-35.

Staat, M. A., Kruszon-Moran, D., McQuillan, G. M., Kaslow, R. A. A population-based serologic survey of *Helicobacter pylori* infection in children and adolescents in the United States. J Infect Dis 1996; 174(5):1120-3.

Steiner, T.S., Thielman, N.M., Guerrant, R.L. Protozoal agents: what are the dangers for the public water supply, Ann Rev Med 1997;48:329-40.

Stettler, N., Schutz, Y., Jequier, E. Effect of low-level pathogenic helminth infection on energy metabolism in Ghambian children. Am J Trop Med Hyg 1998;58(4):476-79.

Steuer, M.K. Hofstadter, F. Probster, L. Beuth, J. Strutz, J. Are ABH antigenic determinants on human outer ear canal epithelium responsible for *Pseudomonas aeruginosa* infections? ORL J Otorhinolaryngol Relat Spec. 1995;57(3):148 – 152.

Stoltzfus, R.J., Chwaya, H.M., Tielsch, J.m., Schulze, K.J., Albonico, M., Savioli, L. 1997. Epidemiology of Iron deficiency anemia in Zanzibari schoolchildren: the importance of hookworms. Am J Clin Nutr 1997; 65(1):153-9.

Stoltzfus, R.J., Dreyfuss, M.l., Chwaya, H.M., Albonico, M., Hookworm control as a strategy to prevent iron deficiency. Nutr Rev 1997;55(6):223-32.

Stotzfus, R.J., Albonico, M., Chwaya, H.M., Savioli, L., Tielsch, J., Schulze, K Yip, R. Hemoquant determination of hookworm-released blood loss and its role in iron deficiency in African children. Am J Trop Med Hyg 1996;55(4):399-404.

Tatala, S., Svanberg, U., Mdumba, B., Low dietary iron availability is a major cause of anaemia: a nutritional survey in the Lindi District of Tanzania. Am J Clin Nutr 1998;68(1):171-89.

Tellez, A., Morales, W., Rivera, T., Meyer, E., Leiva, B., Linder, E. Acta Trop 1997;66(3):119-25.

Theil, E,C., Ferritin: Structure,gene regulation, and cellular function in animals, plants and microorganisms. Ann Rev Biochem 1987;56:289-315.

Tischendorf, F. W., Brattig, N. W., Burchard, G. D., Kubica, T., Kreuzpainter, G., Lintzel, M. Eosinophils, eosinophil cationic protein and eosinophil-derived neurotoxin in serum and urine of patients with onchocerciasis coinfected with intestinal nematodes and in urinary schistosomiasis. Acta Trop 1999;72(2):157-73.

Triteeraprapab, S., Nuchprayoon, I. Eosinophilia, anaemia and parasitism in a rural region of northwest Thailand. Southeast Asian J Trop Med Public Health 1998;29(3):584-90.

Tshikuka, J. G., Gray-Donald, K., Scot, M., Olela, K. N. Relationship of childhood protein-energy malnutrition and parasite infections in an urban African setting. Trop Med Int Health 1997;2(4):374-82.

Tsuyuoka, R., Bailey, J. W, d'Avila, A. M., Guimaraes, N., Gurgel, R. Q., Cuevas L. E. Anaemia and intestinal parasitic infections in primary school students in aracaju, Brazil. Cad Saude Publica 1999;15(2):413-21.

UNICEF. The State of the World's Children 1998.

Utzinger, J., N'Goran, E. K., Marti, H. P., Tanner, M., Lengeler, C. Intestinal amoebiasis, giardiasis and geohelminthiases: their association with other intestinal parasites and reported intestinal symptoms. Trans R Soc Trop Med Hyg 1999;93(2):137-42.

Viqar Zaman. Handbook of Medical Parasitology, 3rd Edition. KC Ang Publication. 1995.

Walters, G. O., Miller, F. m., Worwood, M. Serum ferritin concentration and iron stores in normal subjects. J Clin Pathol 1973;26:770-772.

Warren, K. S., & Mahmound, A. A. F., Eds Tropical and geographical medicine. New York, McGraw-Hill Book Company. 1984.

Wasadikar, P.P., Kulkarni, A.B. Intestinal Obstruction due to ascariasis, Br J Surg 1997;84(3):410-412.

Waters, A. P., & McCutchan, T. F. Rapid, sensitive diagnosis of malaria based on ribosomal RNA. Lancet,1989;I:1343-1346.

Watkins, W.E., Politt, E. "Stupidity or worms ": do intestinal worms impair mental performance? Psychol Bull 1997;121(2):171-191

WHO (1985) Amoebiasis and its control. Report of the WHO Meeting. Bulletin of the World Health Organisation. 1985;63:417-426.

WHO (1986). The use of DNA probes for malaria diagnosis: memorandum from a WHO meeting. Bulletin of the World Health organisation 1986;64: 641-652.

WHO (1988). Malaria diagnosis: memorandum from a WHO meeting. Bulleting of World health Organisation, 1988;66:575-594.

WHO (1990b). Informal Consultation on Intestinal Helminth Infection, 1990. Document WHO/CDS/IPI/90.1.

WHO (1992a). WHO/PAHO InformalConsultation on Intestinal Protozoal Infections, Mexico, 1991. Document WHO/CDS/IPI/92.2.

WHO (1995). *Intestinal Helminths*. Weekly Epidem. Rec. 4, 1995 pp25-28.

WHO (1985). Amoebiasis and its control. Report of a WHO meeting. Bulletin of World health Organisation 1985;63(3):417-426.

WHO (1987). Prevention and control of intestinal parasitic infections. Report of a WHO Expert Committee 1987;49:86.

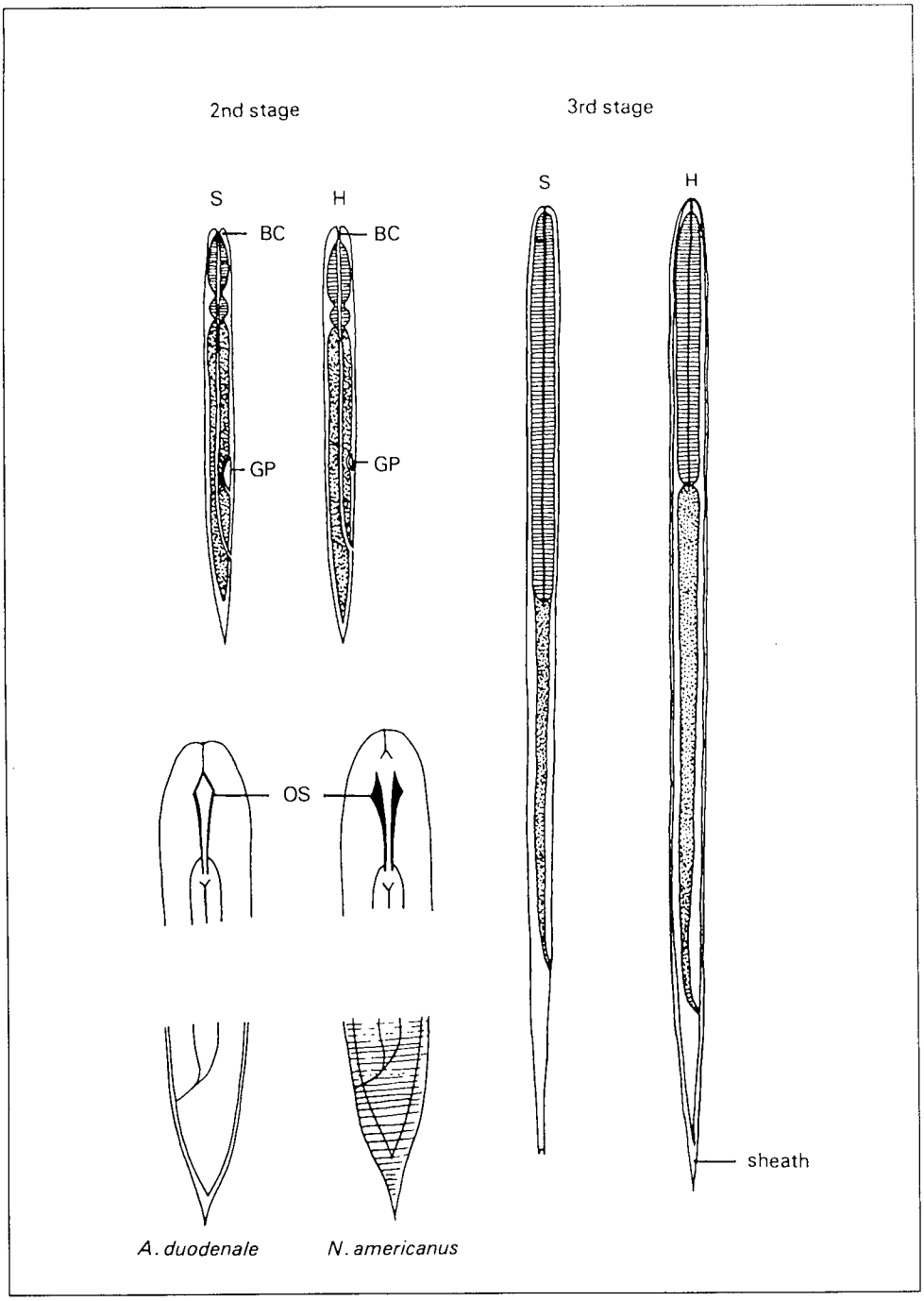
World Bank. World development report 1990:Poverty. New York: Oxford University Press.

World Bank. Development in Practice 1994: Better Health in Africa, Experience and Lessons Learned.

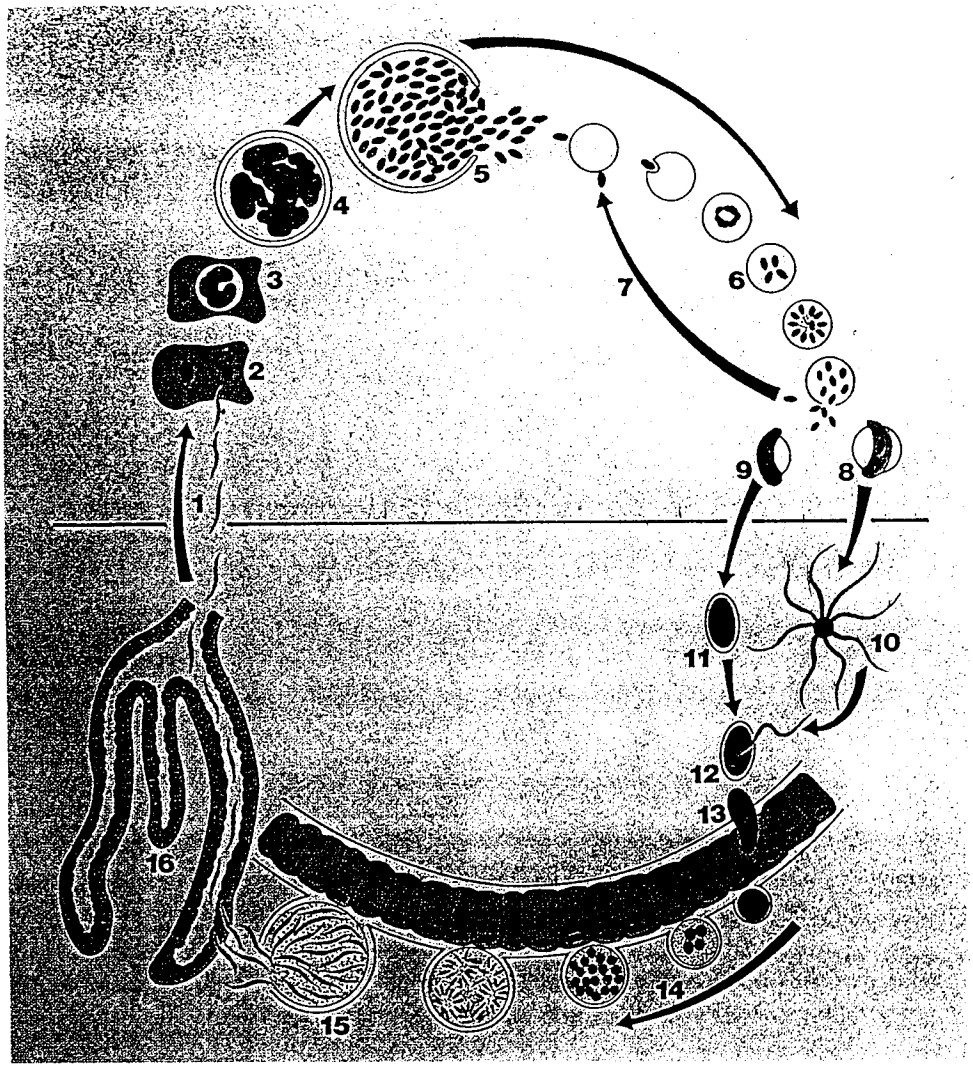
Worwood, M. Serum ferritin. CRC Crit Rev Clin Lab Sci.1979;10:171.

Zuk, M., McKean, K. A., 1996. Sex differences in parasite infections: patterns and processes. Int J Parasitol 1996;26(10):1009-23.

APPENDICES

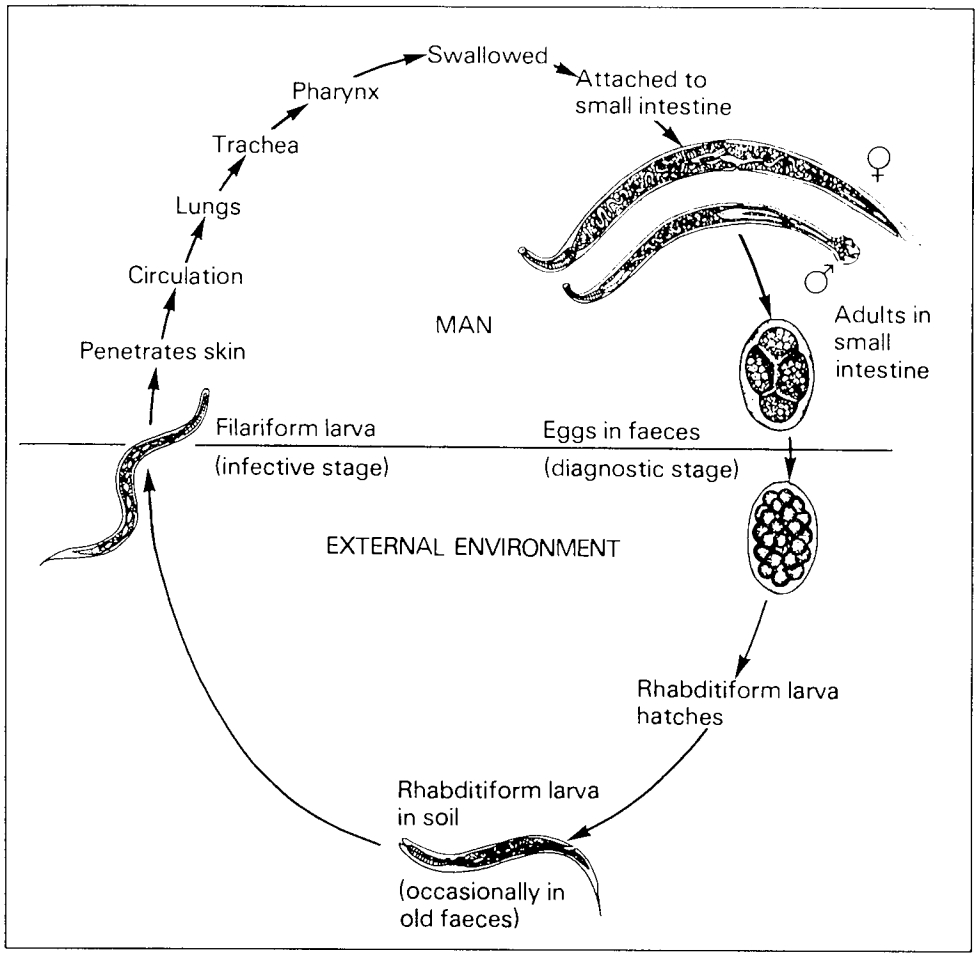


Larval morphology of hookworms and *Strongyloides*. BC = buccal capsule; GP = genital primordium; OS = oesophageal spears; S = Strongyloides; H = Hookworm.

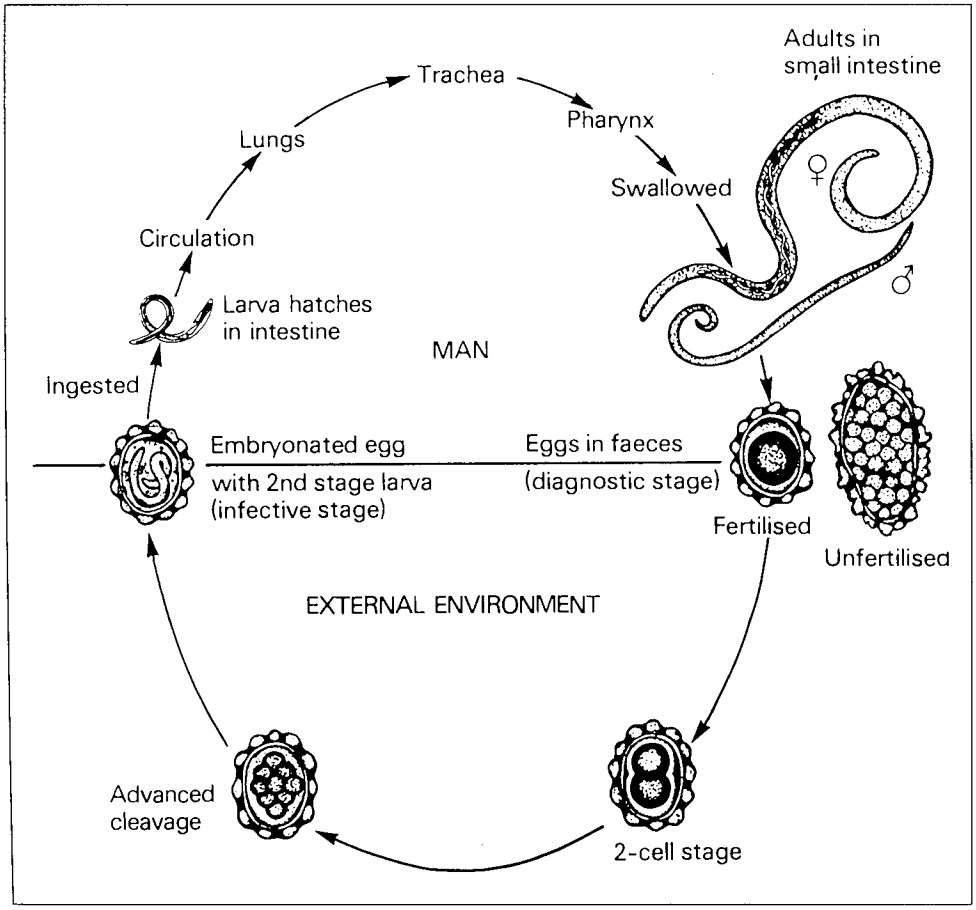


Life cycle of *P. falciparum*.

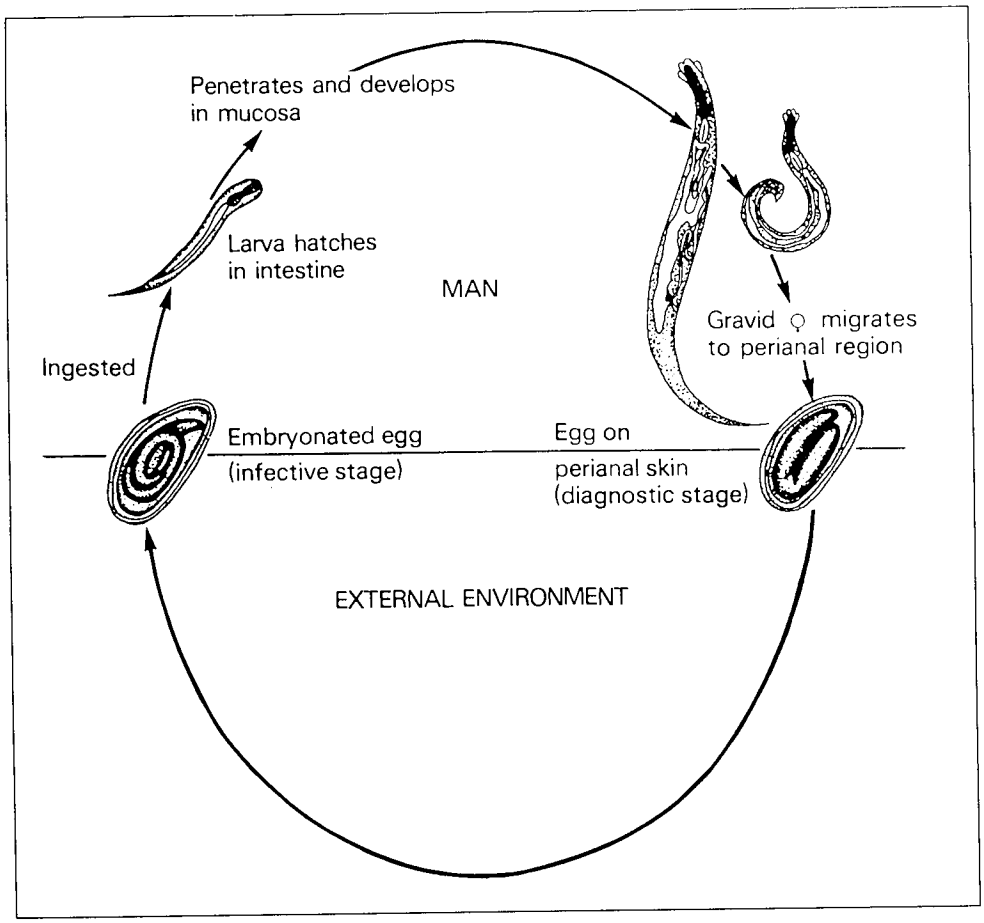
1 sporozoite entering the liver cell
2 developing pre-erythrocytic stage
3 developing pre-erythrocytic stage
4 developing pre-erythrocytic stage
5 mature pre-erythrocytic stage liberating merozoites
6 erythrocytic stages showing schizogony
7 merozoite re-invading fresh rbc
8 male gametocyte
9 female gametocyte
10 exflagellation
11 macrogamete
12 fertilisation
13 ookinete
14 developing oocyst
15 mature oocyst releasing sporozoites
16 invasion of the salivary gland by sporozoites.



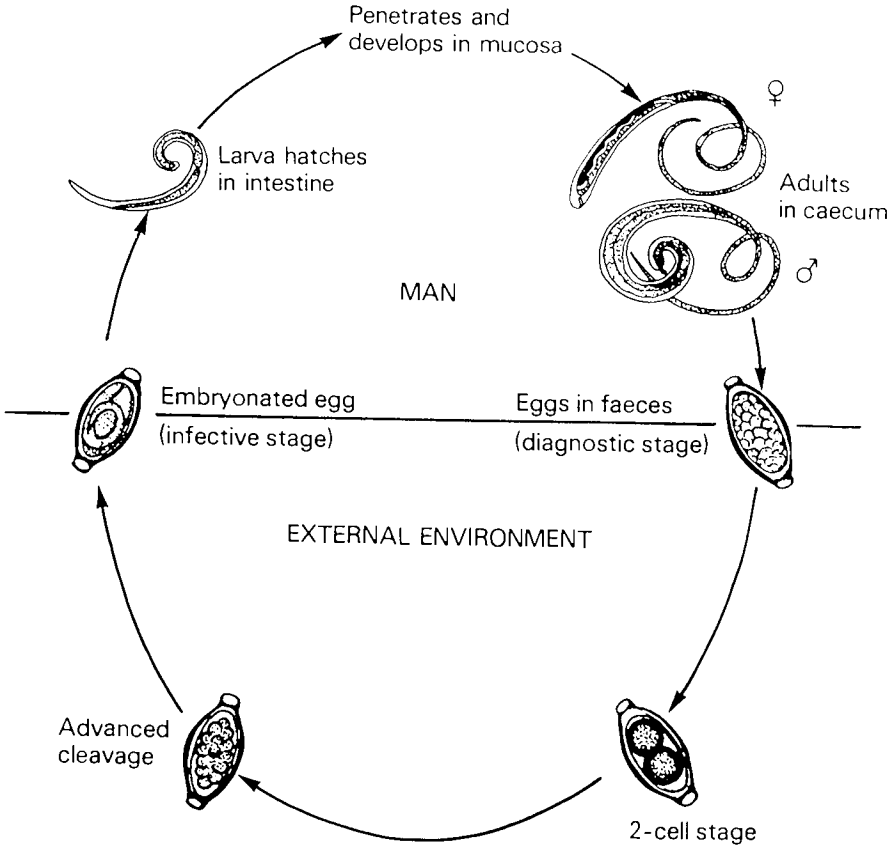
Life cycle of a Hookworm.



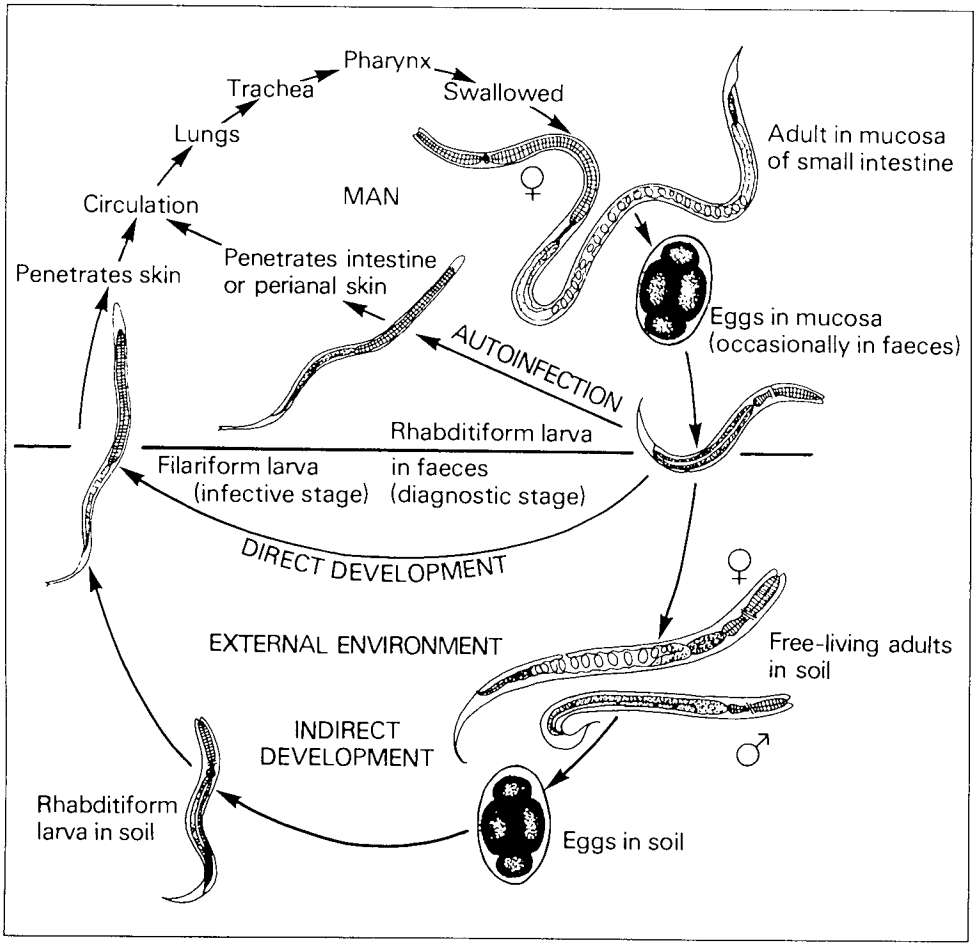
Life cycle of *Ascaris lumbricoides*.



Life cycle of *Enterobius vermicularis*.

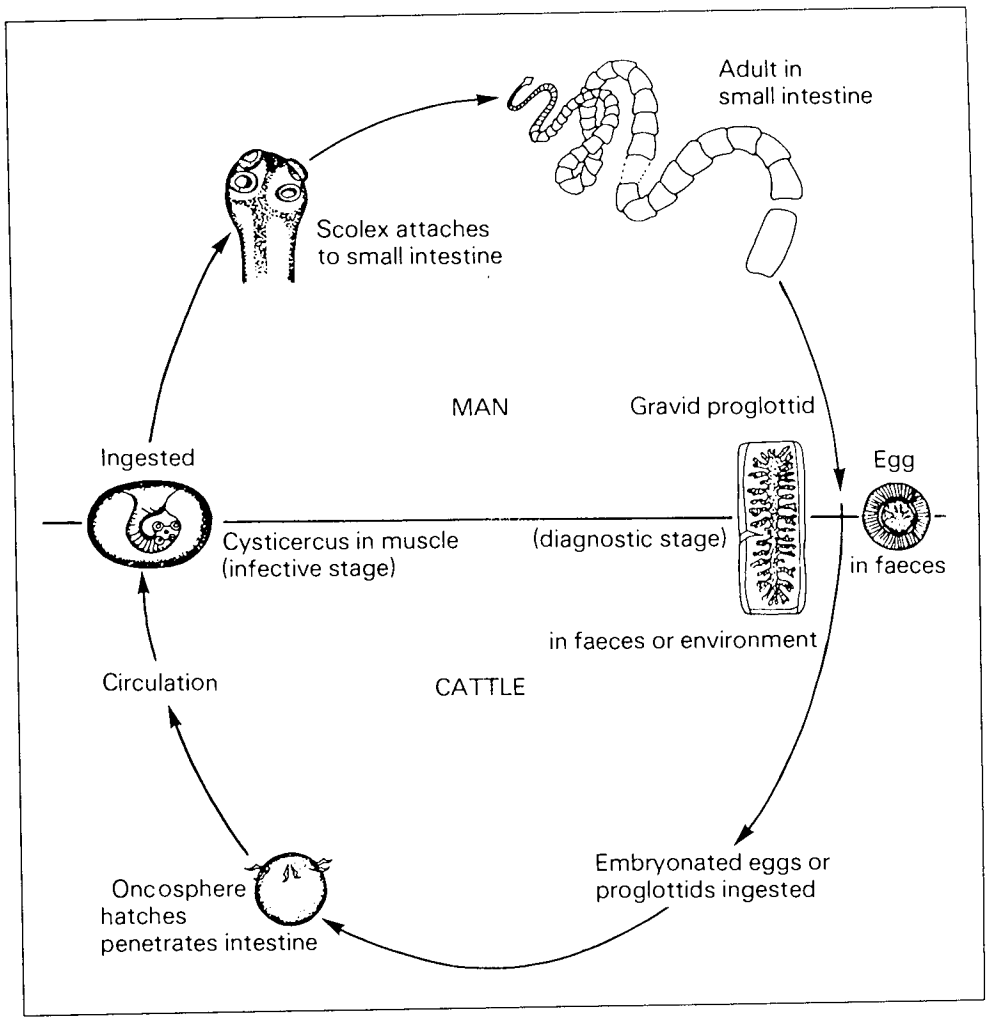


Life cycle of *Trichuris trichiura*.

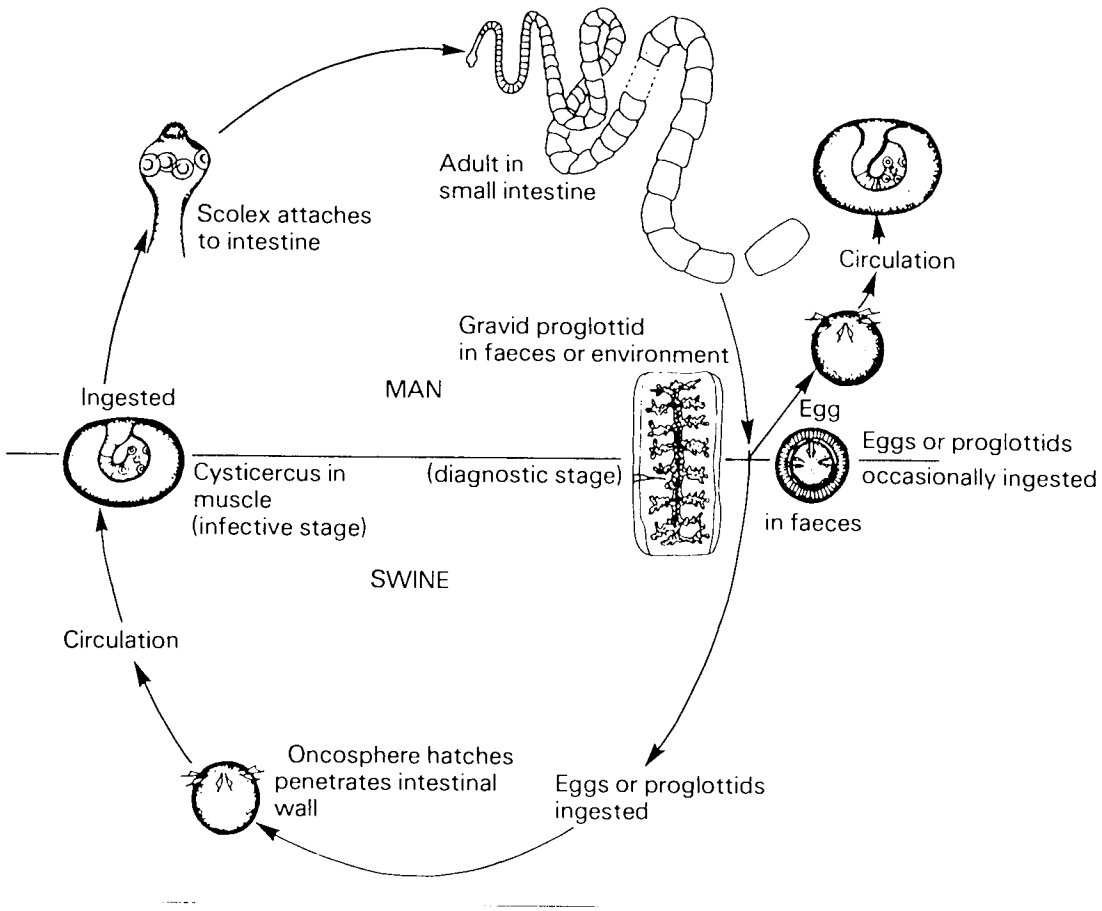


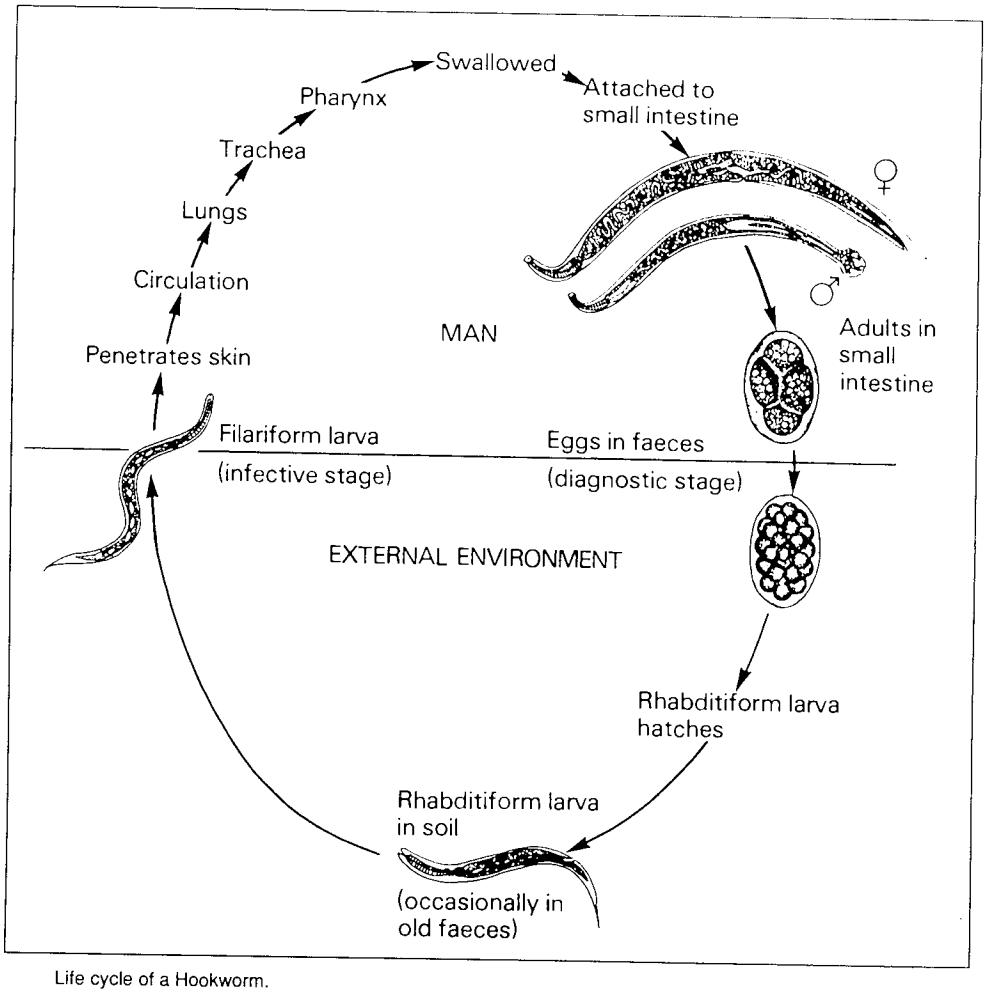
Life cycle of *Strongyloides stercoralis*.

LIFE CYCLE OF TAENIA SAGINATA



LIFE CYCLE OF TAENIA SOLIUM





Life cycle of a Hookworm.