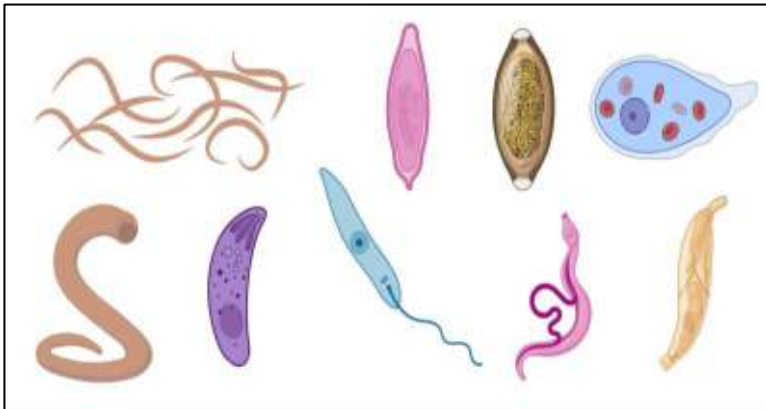




# THE FEDERAL UNIVERSITY OF TECHNOLOGY, AKURE

A PARASITE-FREE WORLD: ACHIEVABLE OR  
WISHFUL THINKING

INAUGURAL LECTURE SERIES 196



Delivered by

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14<sup>th</sup> April, 2026



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# **A Parasite-Free World: Achievable or Wishful Thinking**

## **PROTOCOLS**

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Other Principal Officers of the University,  
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Professors and Members of the Senate,  
My Lords Spiritual and Temporal,  
Distinguished Guests and Friends of the University,  
Ladies and Gentlemen of the Press,  
Distinguished Colleagues, Ladies and Gentlemen,  
Great FUTARIANS!!!

## **PREAMBLE**

It is with a humble heart full of profound gratitude to God Almighty and to Jesus Christ “the author and finisher of my faith” that I stand here before you today the 14<sup>th</sup> day of April 2026, to present the 196<sup>th</sup> inaugural lecture of the Federal University of Technology, Akure.

This is the 9<sup>th</sup> inaugural lecture from the Department of Biology and the second from Applied Parasitology Programme of the Department. The previous lectures from the Department were delivered by Professors S.A Fasuyi (15<sup>th</sup> March, 1994), F.C Adetuyi (9<sup>th</sup> November, 1999), O.O Odeyemi (6<sup>th</sup> December, 2005), C.O Adedire (28<sup>th</sup> October, 2008), M.O Ashamo (5<sup>th</sup> March, 2019), M.O Oniya (21<sup>st</sup> February, 2023), J.A Adeyemi (27<sup>th</sup> August, 2024) and O.E Oladipo (24<sup>th</sup> February, 2026).

Today, I feel deeply honoured by your esteemed presence at my long-awaited inaugural lecture titled “A parasite-free world: achievable or wishful thinking” which had to be rescheduled twice due principally to health reasons.

Madam Vice Chancellor, my entire journey through life thus far, has been a clear testimony of the goodness and faithfulness of the Lord, who has always been my guide. My career journey in academics began sometime in 1978 when I was offered admission to read B.Sc. Zoology at the newly founded University of Maiduguri. Being a single honours course, after graduation, I determined in my mind to proceed for higher degrees whenever I have the opportunity. After the compulsory National Youth Service Corps in 1983, I secured employment with Ondo State Teaching Service and was posted to Owo High School, Owo, as Tutor II. Shortly afterwards, I was promoted to the position of Vice Principal and posted to Eyemojo Comprehensive High School, Osan Ekiti, in the Old Ondo State. The opportunity to join academics presented itself when in 1985 I secured and promptly took up an appointment as Graduate Assistant in the defunct University of Jos, Makurdi Campus, not allowing the allure of Vice Principal and the prospect of becoming a substantive Principal to dissuade me. This was because I saw it as a golden opportunity of fulfilling my most cherished ambition then, of acquiring higher degrees, particularly Ph.D which came to pass in the course of my engagement in the university while enjoying my career progression.

The essence of inaugural lecture is for the presenter to showcase what he or she professes in his or her field of specialization, and to share his or her research and teaching accomplishments with the University community and the public at large.

Parasitism is an interaction between organisms and the product of natural selection, and should therefore be understood as selective response to pressures from the environment. Parasites by definition live in or on a hosts from whom they take something, an attribute that has qualified parasites as the primary enemies of the animal world.

However, not all parasites cause noticeable harm to their hosts and only a small percentage affect humans. Scientists have warned of dire consequences of disregarding the remaining vast majority. Studies have shown that parasitic diseases in association with nutritional deficiencies are the primary killers of humans. Recent summaries of world-wide prevalence of selected parasitic diseases indicate that there are more than enough existing infections for every living person to have one.

Not only is there much we can learn about parasites and the ways we can use them for our own needs (such as the medicinal leech, *Hirudo medicinalis*, still being employed in some surgeries), but we are also beginning to understand that parasites play crucial roles in the ecosystem by keeping some populations in check while helping to feed others.

All said, literature has it that 40% of all known animals are parasites and that these are just the ones that have been described. It is the thinking of scientists that all the described parasites to date, constitute only about 10% of all parasites that exists out there, leaving potentially millions more, yet to be discovered.

## **1.0 INTRODUCTION**

Parasitism is defined as the association between two organisms in which one, the parasite, lives temporarily or permanently in or on the other, the host, deriving benefits from it and in the process causing harm to it. Thus, the parasite gains while the host loses. Parasitism, commensalism and mutualism are the three recognized but not clearly distinguishable types of inter-specific associations between organisms. Symbiosis which is sometimes mistaken for any of the three simply or literarily means “living together”. Although some authors restrict the term to relationships wherein both partners benefit, the original proposer of the term symbiosis, the German Scholar, A. de Barry, 1879 defined it as “Any two organisms living in close association, commonly one living in or on the body of the other, are symbiotic as contrasted

with free-living". The general tendency is to regard all organisms living in or on another as parasites, because it is sometimes difficult to decide if an organism living in or on another actually inflicts harm on its host or not, though, if it can be demonstrated that no harm is done, they must be regarded as commensals.

Similar difficulties also arise when trying to decide whether an association is commensal or mutual. Therefore, for an organism to be regarded as a parasite, it must convincingly be shown or demonstrated that the organism (a) does reside in or on its host, (b) derives any form of benefits from the relationship while the host does not, and (c) the host suffers harm as a result of the association.

**(a) Residing in or on the host:** Parasite-host association can be spatial and temporal.

**Spatial** relates to the part or region of host body being exploited by the parasite. Those living on the surface of host's body are regarded as ectoparasites while others dwelling inside are endoparasites (Plate 1). These latter groups are further subdivided according to how deep into the host body they live. Thus some of such parasites live in the skin or just beneath it, others prefer tissue fluids (intercellular parasites), while some others reside inside individual cell of their host (intracellular parasites) e.g. the malaria parasite, Plasmodium.

**Temporal** relationship defines the length of time the parasite spends in or on its host. While some never leave their host (permanent lodgers), exemplified by the tapeworms (*Taenia saginata* and *T. solium*), some others are ectoparasites that attach to their hosts only when they feed (occasional visitors) e.g. medicinal leech (*Hirudo medicinalis*).

**(b) Benefits derived:** Shelter and food are the two principal benefits the parasite derives from its host. Endoparasites enjoy maximum protections since they inhabit a stable internal environment where they obtain nourishment with minimum effort. On the contrary however, gut parasites are exposed to host's digestive enzymes; consequently they

have developed the ability to secrete appropriate inhibitors (antienzymes) that inactivates these enzymes (Scott et al., 2010; Suleiman et al., 2022).

**(c) Harm suffered:** Relating to harm, parasitism can be viewed as a type of interaction in which one species, the parasite, uses another, the host, in a way that is harmful to the host. Host-parasite relationships are like predator-prey relationships i.e. one population of organisms feeding on another. The difference however is that the parasite rarely kill their hosts quickly because, a dead host is not useful as a continuing source of nourishment.



**Plate 1:** Examples of endoparasites (A-F) and ectoparasites (G-K).

Extreme case of harm is suffered by the host when the parasite is intracellular and feeds on host tissues, the sheer damage of which might even result in death. Example here includes the malaria parasite, Plasmodium, which invades, resides in and destroys host red blood cells (erythrocytes). Some members of this category of parasites like the trypanosomes (that cause African sleeping sickness) liberate toxic substances into host blood stream in addition to exploiting host blood glucose content.

Gut parasites feed on host digested nutrient and therefore pose the least harm to the host. Notwithstanding, cases of about 5000 round worms (*Ascaris lumbricoides*) blocking the intestines and causing death in young children have been documented (Roberts, 1976) (Plate 2).



**Plate 2:** *Ascaris lumbricoides* (round worms) in human intestine occurring in large numbers that it blocks the gut. (Source: Roberts, 1976).

Also the tiny larvae of the filarial worm (*Wuchereria bancrofti*) that invades the lymphatics where they mature and aggregate into such numbers as to block the lymphatic vessels resulting in enormous enlargement of extremities of an infected host, particularly the legs and scrotum known as elephantiasis (Plate 3).



**Plate 3:** Elephantiasis of the lower limb (A-D) and scrotum (E & F).

Parasites most often increase their reproductive potential through production of vast numbers of eggs. For example, the common rat tapeworm, *Hymenolepis diminuta*, produces up to 250,000 eggs per day for the life of its host, and during a period of slightly over a year, a single tapeworm can generate a hundred million eggs. If all these eggs reach maturity in new hosts, they would represent more than 20 tons of tapeworm tissues. Female nematode parasites are also sometimes prodigious egg layers, a single *Ascaris lumbricoides* can produce more than 200,000 eggs each day for several months, and over the course of their lifetimes, members of the filarial worm, *Wuchereria bancrofti*, may release several million young (microfilariae) into their hosts' blood. High reproduction potential, ensures that such parasites become medical and veterinary problem when host population are crowded and condition for transmission are favourable (Schmidt and Roberts, 2010).

## **2.0 ORIGIN AND EVOLUTION OF PARASITISM - in the beginning there were Parasites.**

Parasitism has been found to be an essential part of life to the extent that parasites are found in virtually all of the species of organisms that exist on planet earth (Paulin and Morand, 2000). Little wonder that some ecologists have estimated that at least one-third of all species of organisms on earth are parasites.

Since the beginning of life, protoorganisms (the earliest form of life) adopted parasitism in order to multiply. According to Bremermann (1983), parasitism must have therefore occurred at the early stage of evolution.

All living organisms have a uniform biochemical composition that points to a common origin (Nasmith, 1995), in a common ancestor that lived a billion years ago (Orgel, 1998). Life on earth was only possible as a consequence of parasitism in what was still a molecular world. The appearance of life on earth around 4.4-3.8 billion years ago was thought to be a consequence of molecular parasitism (Chang, 1999; Nisbet and

Sleep, 2001) and the present day life forms still retains the relics of these ancient associations in their genomes (Galtier et al., 1999, Cavalier-Smith, 2001). The later diversifications of life forms and species radiation were also believed to be the consequence of these multiple associations. All these ancient events thus represented the first steps towards a host-parasite way of life, though still at the level of molecules. Worthy of note is the fact that, the very first forms of life in the so-called “RNA world” (Maynard-Smith and Szathmary, 1993; Shapiro, 1999; Poole et al., 1999), along with the other forms that appeared when life started (de Duve, 1998; Shapiro, 1999; 2000) are still an issue under debate.

Associations recognized as host-parasite systems existed since the very first gene association, long before the appearance of the genetic code and gene translation (Maynard-Smith and Szathmary, 1993). Parasitism has been identified as an important promoter of biodiversity, commencing at the molecular level. Molecular parasitism is clearly exemplified by transposable elements of the genome that occur in both eukaryotes and prokaryotes. Indeed, the DNA sequences called transposable elements are actually recognized as molecular parasites (Doolittle et al., 1984; Kidwell and Lisch, 2001).

To date, mounting evidence abound supporting the suggestion that most parasites are products of long time evolution first as symbionts and are thus exquisitely adapted for life within the body of another organism. And the fact that parasites far outnumbered free-living organisms in the world is an indication of the success of parasitism.

## **2.1 EVOLUTION OF VIRULENCE**

Parasites are assessed based on their virulence and pathogenicity. Whereas virulence is the ability of the parasite to multiply, and is under natural selection pressure to increase transmission success (Poulin and Combes, 2000), pathogenicity refers to aspects of the host-parasite association that results in damage to host, thus mediating morbidity and mortality in infected hosts (Levine, 1996).

The question of why some parasites seem to be especially virulent while others are relatively benign has captured the attention of many investigators. A long-established paradigm holds that parasites should evolve into less virulent forms, mainly because the death of a host is expected to have a negative effect on parasite survival. However, according to some theories, parasites should evolve an optimal virulence that maximizes their numbers, with “optimal” depending on a number of factors such as pathogenicity and transmission dynamics (Lenski and May, 1994).

Most, if not all parasites are transmitted both vertically (between generations) and horizontally (among members of the same generation) of hosts. Some theoretical work suggests that vertical transmission tend to select far less virulent parasite strains, whereas horizontal transmission, especially when coupled with high transmission rates, selects far more virulent strains. Not all studies support this idea however, and numerous studies suggest that factors such as genetic diversity of host and parasite, individual host-parasite interactions and different time scales for transmission can also affect the evolution of virulence.

### **3.0 PARASITES AND INFECTIOUS DISEASES**

Parasitic infections and parasitic diseases are two strikingly distinct situations originating from a single process. A parasite is a necessary but not sufficient condition to launch a parasitic disease (Ferreira, 1973). Parasitism does not necessarily result in injury or benefit to the host. Parasitic disease is an eventual outcome of a given parasite in a given host from a given population in a certain environment during a particular life co-evolution period of both protagonists. In essence, it is a unique result of association of parasite and host in a given environment. It does not matter whether a unicellular or multicellular parasite is involved. A system formed by the parasite, the host and the environment where each one interacts and influences the other subsystem in such a way that any change in one

subsystem affects the other two (Ferreira, 1973). A parasitic disease is therefore an expected event whenever one of the variables, hinder or damages the parasite or the host, or when the environment is modified.

#### **4.0 EFFECTS OF PARASITES ON HOSTS**

Parasites affect hosts by causing diseases in them, a process known as pathogenesis. The pathogenic effects of parasites may be so subtle as to be undetectable. A host may harbour hundreds of parasites and still appear to show no clinical symptoms. On the other hand, the host may be so obviously stunted and sick as a result of the infection, e.g. schistosomiasis.

The effects of parasites on infected hosts can be roughly divided into six categories: (i) Destruction of host tissues, (ii) Alteration of host tissues, (iii) Nutrient robbery, (iv) Production of toxins, (v) General upset of body chemistry, and (vi) Immunological complications.

##### **(i) Destruction of host tissues**

There are numerous examples of this in parasitic infections. The larvae of hookworms infect hosts by penetrating through the skin. When a dog hookworm infects man, a condition known as ‘creeping eruption’ sometimes result in the skin. The larvae being in the wrong host fails to penetrate the dermis and crawls around in the epidermis leaving inflamed snake-like tracks of damaged tissues. Both *Ascaris* and hookworms migrate through the body before entering the intestine. This include passing from the capillaries of the lungs into the alveoli before emigrating up the bronchioles and finally being swallowed. Their penetration of the lungs causes haemorrhage and bronchial problems. Another example of tissue destruction is seen in malaria infection when the parasites infect and destroy red blood cell (erythrocytes) as they escape to infect new ones. In amoebiasis, the pathogen *Entamoeba histolytica* feeds directly on the host’s cells, causing ulceration of the gut leading to bloody diarrhoea. In systemic

infections the amoebae invade and feed on other organs, sometimes resulting in death.

**(ii) Alteration of host tissues**

The presence of a parasite in host tissues frequently cause them to change their nature and lose their normal function. This often takes the form of fibrosis i.e. the deposition of collagen fibres as in schistosomiasis. The eggs of the worm become deposited in various parts of the body, especially those which are well vascularized such as the liver, which becomes fibrotic and functions less well, sometimes resulting in cancer of the liver. In filariasis, dead adult filarial worms in the host lymph nodes cause fibrosis there, resulting in blockage of the lymph ducts, accumulation of fluid in the limbs, fibrosis of limb tissues described as elephantiasis (Plate 1). This may be accentuated by secondary infections with bacteria such as Streptococci.

Parasites living in the gut may alter the structure of the gut. This has been shown in the case of *Nippostrongylus brasiliensis*, a hookworm which is found in rats and which is frequently used as an experimental model. In infected rats, the villi are atrophied and irregular in shape, the microvilli are shorter and less dense, resulting in a reduction of surface area which may contribute to reduced efficiency in food absorption by the host (Symons, 1976). The pathology associated with hookworm infections is not due to blood loss caused by the feeding of the worms but also to impairment of food absorption by changes in the gut mucosa.

An interesting example of host tissue being altered by parasites is seen in the case of *Trichinella spiralis* where the larvae become encysted in the muscle of the infected host. Experiments have shown that such infected muscles can go into tetany sooner and become fatigued more quickly than uninfected muscles. When the larva invades the tissues and encysts, it causes the nearby muscle fibres to transform to new cell type whose function is to protect and nurture the developing parasite. These 'nurse cells' although physiologically active, are

incapable of contraction. In addition there is upset to the normal calcium pump of such muscles. High levels of free calcium are found even in recently contracted muscle. This provides an uninterrupted stimulus for contraction, resulting initially in the observed tetany, and eventual fatigue, as the muscles use up their energy reserves.

### **(iii) Nutrient robbery**

The importance of this particular mode of pathology is a little controversial since most parasites divert only a relatively small proportion of their host's food to their need. Tapeworms for example only half metabolize the glucose they take up, excreting lactate which can be used by the host as a source of energy. In addition, when times are hard and food is scarce, they destrobilate (i.e. lose their entire tapes), leaving the small scolex behind to survive until times improve. People are able to harbour heavy infection of *Ascaris* without clinical symptoms.

Most documented evidence of food robbery involves malnutrition of the host caused by the parasite absorbing a large proportion of a particular nutrient that is of great importance to the host. A classic example of this is seen in tapeworm pernicious anaemia caused by infections with the great tapeworm of man *Diphyllobothrium latum*, prevalent in temperate countries where fish harbouring the parasite is consumed raw or undercooked. This has strong affinity for Vitamin B<sub>12</sub> which it absorbs from the intestinal wall of the host. Since this vitamin is essential for the formation of red blood cells (RBCs) through a process known as erythropoiesis, it results in chronic anaemia in the host. *Ascaris* has a similar affinity for vitamins A and B<sub>12</sub> which it can only synthesize to a limited extent. The balance must therefore be acquired from the host.

### **(iv) Production of toxins**

Several parasites are known to produce toxins which damage their hosts' tissues.

- *Trypanosoma cruzi*: develops clusters of cells in the smooth and cardiac muscles of the host. When they degenerate, they release a neurotoxic substance that attacks the autonomic ganglion cells, ruining nervous control of peristalsis and heart contraction respectively.
- *T. brucei gambiense*: produces a neurotoxin that causes severe brain damage resulting in the sleeping sickness syndrome.
- *Plasmodium*: toxins released from burst red blood cells cause the chills and fever that characterizes malarial infections.

**(v) General upset of body chemistry**

This results from any of the above categories of causes of pathology and can manifest itself in a number of ways. Mice infected with *Plasmodium* show a low blood cholesterol. This is due to hyperactivity of the thyroid gland, which is supposedly stimulated by some substances produced by the parasite. Such hormone changes can have profound effects on the host. Trypanosome infections in mice cause reversal of diurnal body rhythms and a decrease in water consumption and urine outflow. This is thought to be due to hormonal imbalance.

The picture is sometimes quite complex as shown in the case of *Trypanosoma congolense* which causes severe anaemia in cattle. This is due to decreased protein anabolism, and consequently production of red cells; and an increased protein catabolism. Parasitic infections may also cause the loss of unborn young. Trypanosome infections in rats though they may not be pathogenic to the host, cause resorption of the foetus. If the foetus is well developed, the mother may die due to combined anaemia of the disease and birth. *Toxoplasma gondii* infection can also cause abortion in pregnant mothers (Olusi, 2008).

**(vi) Immunological complications**

The importance of this cause of parasite pathology has been appreciated with the advent of world-wide growing interest in

immunology. Being large and biochemically complex organisms, some parasites evoke strong and diverse immune responses in the host. Many of these reactions are not of protective importance (e.g. reaction with the metabolic waste of the parasite) but may cause severe side effects. An excellent example of this is seen in human infections with *Plasmodium malariae* (common in Africa). The parasites circulate in the blood for a fairly long time and produces a considerable amount of antigen in the blood. These are 'mopped up' by the host immune response frequently forming soluble immune complexes of Ab/Ag, under certain circumstances, which may be deposited in the kidneys causing a nephritic syndrome sometimes called 'chronic soluble complex disease'. The kidneys malfunction and protein is excreted in the urine. This can be particularly debilitating to malnourished persons whose immune response is poor and are therefore prone to this condition. They develop a worse protein deficiency than before. A similar situation is seen in *Schistosoma haematobium* infection.

Malaria and trypanosomiasis are also known to cause immunosuppression. This may sound paradoxical in view of the fact that the parasite does stimulate the immune response. However it has been suggested that the parasites act by somehow breaking the control link between the host's B cells and T cells, resulting in some B cells getting out of control, and going on to produce a heterologous collection of antibodies. The parasites may also produce a substance which stimulates the B cells to reproduce (mitogen). Most of these antibodies are functionless and the immune system is kept busy producing them. While the immune response is 'distracted' in this way other infections can proliferate. Malaria has been shown to act in conjunction with a virus to give rise to tumors.

Another pathological condition caused by the host immune response to blood parasites is the lysis of red blood cells. The antigen in the plasma binds to the uninfected RBCs, the host antibody therefore recognizes the cells as being foreign and destroys them.

Immune problems also occur in helminth infections. This is especially noticeable when certain drug therapy is being used. In onchocerciasis and elephantiasis, the death of the worms resulting from the application of drugs, causes the release of large amounts of worm's antigen. This causes a violent immune response leading to unpleasant side effects. In onchocerciasis, the patient suffers an intense itching all over the body due to the death of microfilariae in the skin.

## **5.0 EPIDEMIOLOGY OF PARASITIC INFECTIONS - My journey with Parasites**

The epidemiology of infectious diseases attempts to describe the patterns and processes by which diseases are distributed in the host population. In a parasitological context, epidemiology is the study of infectious diseases and disease-causing agents such as parasites (protozoans and helminthes), at a population level. It seeks to characterize the patterns of distribution and prevalence of the diseases, and the factors responsible for these patterns. In a more applied context, epidemiology also strives to identify and test prevention and treatment measures. The transmission or movement of parasites from one host to another is a crucial factor in understanding the epidemiology of diseases (Elbert, 2005).

The displacement of people due to conflicts and wars, increased international travels, and shifting patterns of immigration, have further increased the importance of raising awareness of these parasitic infections. As at 2016, the World Health Organization (WHO) estimated the global burden of parasitic infections at approximately three billion reported cases and one million deaths per year (Piperaki and Tassios, 2016). In a survey carried out in 2024, Jin et al. (2024) reported that one and half million people worldwide, are infected with soil transmitted helminthes (STHs), primarily from tropical and subtropical regions of the world.

Parasitic infections are most prevalent in developing nations where they constitute a major public health concern, with dire consequences for individuals with weakened immune system (immune-compromised). Infections are transmitted directly from person to person (horizontally, indirectly through an intermediate host (vector-borne), agricultural lands contaminated with human and animal faeces (faecal-oral route) consumption of vegetables infested with parasite eggs or larval stage (food-borne) or contaminated water (water-borne) (Ahmed, 2023).

Disease-causing parasites have varied life cycles, consequently, the infections they cause, whose consequences are varied and sometimes fatal are known to be associated with geographical and socio-economic factors, as well as natural disasters and wars, poverty, malnutrition, personal and community hygiene, high population density, unavailability or absence of potable water, low health status or compromised immune system, outright absence or poor sanitation, growing anti-parasitic resistance and climate change. The combination of these conditions greatly enhances the growth, transmission and dissemination of parasites and increase in the prevalence and incidence of the diseases they cause in human communities (Smith and Conn, 2015; Norman et al., 2015).

## **5.1 Mechanisms of Parasite Transmission**

Transmission is the indispensable aspect in the life of parasites that refers to the means by which they are transmitted from one host to another. This has been shown to occur through four major routes, including direct contact, contaminated food and water, vectors, and fomites. Parasites are able to continue to thrive and exert their insidious and treacherous influence on countless organisms across the biological spectrum through the above listed routes of transmission. Other less prominent routes include vertical (mother-to-foetus) transmission (as in *Trypanosoma cruzi*, *Toxoplasma gondii*), blood transfusion (as in *Plasmodium falciparum*, *T. gondii*) and organ transplantation (as in *T.*

gondii). Transmission through direct contact can occur either between individual hosts, as they come into contact with each other's vulnerable surfaces or with environmental surfaces infested with parasites. This, according to Lopez and Duffy (2021), is a clear testament to the fact that parasites have become so adapted and resourceful that they have mastered the art of transmission via direct contact to such a fine degree.

The marvels of parasitic transmission do not stop at the above, as, in the realm of ecto-parasitism, fascinating mechanisms come into play. The ecto-parasites, such as blood-feeding creatures like ticks and fleas have developed an astonishing repertoire of 'mechanosensory' triggers to initiate their transfer from one host to another. They are attuned to an unbelievably wide range of stimuli that allow them to seamlessly transit between hosts and continue their parasitic pursuits with ease (Hawley et al., 2021).

The world of parasitism becomes even more intriguing when we delve into the realm of endoparasites. These insidious organisms take up residence within their hosts, establishing an intimate connection that is both fascinating and destructive. Endoparasites draw sustenance from their unfortunate hosts, causing damage and siphoning off vital nutrients from a multitude of vertebrate tissues. The intricate complexity of this relationship between host and parasite is a testament to the perfect balance that has evolved over millennia. Endoparasitic transmission can occur through direct means, wherein parasites are transmitted in their infective stages via coprophagy or by infiltrating internal tissues (Milotic et al., 2020).

The plot thickens even further when parasites employ an indirect transmission method. This involves the engagement of a reducing or intermediate host, where a critical stage of development takes place before the final transmission to a suitable host site occurs. This intricate dance between hosts and parasites highlights the adaptability and resourcefulness that underpin the success and proliferation of parasitism in the natural world. As our understanding of the intricate web of parasites transmission grows, so does our

appreciation of the astonishing ways in which these stealthy organisms ensure their survival. The complexities and mechanisms involved in parasites transmission are not simply elusive curiosities, but rather a vital field of study that sheds light on the delicate balance that exists within ecosystems (Lopez and Duffy, 2021).

According to Hawley et al. (2021), unraveling the secrets of parasitic transmission is a critical requirement in gaining a deeper understanding of the intricacies of life itself. Some parasites have the capability to alter the behavior of their hosts, making infected organisms more vulnerable to predation. This is a well-documented phenomenon, particularly among protozoan parasites.

There is another intriguing occurrence that has not been extensively studied which is the manipulation of intermediate hosts to increase their chances of being consumed by the final host after being infected by a specific parasite. This process has been most commonly observed among various trematodes, endowed with intricate life cycles that involve infecting multiple intermediate hosts before reaching maturity within a single final host, such as cormorants or herons. In addition to manipulating hosts' behavior, certain parasites, particularly those with pathogenic attributes, can also render their hosts more susceptible to other diseases. This phenomenon plays a significant role in the growing concern surrounding “emerging diseases”, especially within wild and domestic animal populations. It is important to note that paradoxical situations exist whereby parasites are intentionally introduced into a host population, with the aim of gaining a commercial advantage (Dubois and Binning, 2022).

## **5.2 Risk factors for Parasitic infections**

Several factors are known to increase the likelihood of acquiring parasitic infections, these according to WHO (2020) include:

- Poor sanitation: inadequate waste disposal, lack of access to potable water, and poor hygiene can increase the risk of parasitic infections.
- Poverty and low socioeconomic status: People living in poverty and low economic status are more likely to be infected with parasites owing to their limited access to healthcare, clean water and sanitation.
- Travel to endemic areas: Traveling to areas where parasites are common can increase the risk of acquiring an infection.
- Weakened immune system: People with weakened immune systems, such as those living with HIV/AIDS, suffering from malignant diseases or taking immunosuppressive medications are usually more susceptible to parasitic infections.

### **5.3 Epidemiological studies of some parasitic infections**

Defined as the science that evaluates the occurrence, determinants, distribution and control of health and diseases in a defined human population (Lucas and Gilles, 2003), epidemiology is an indispensable area of parasitology. This stems from the fact that it facilitates a thorough understanding of the extent of disease spread and the development of effective control strategies. In other words, it provides the foundation for control programmes by identifying the sources of parasite spread in populations and environments, which guarantees targeted interventions. Epidemiology assists in the analysis of field data, and identify patterns of diseases and designing of intervention programmes that prevents infection and mitigate the impact.

Madam Vice Chancellor, permit me to state at this juncture that my interest in the epidemiology of parasitic diseases date back to my undergraduate days at the University of Maiduguri, when in my final year, I was allocated to a Briton and seasoned epidemiologist, Dr. Christen Betterton-Jones for the supervision of my project work. She

was the one that stimulated my then young mind in epidemiological research.

To date, majority of my over three scores of journal publications and contributions to books were on different aspects of epidemiological studies of various diseases caused by parasites of medical and veterinary importance. Permit me to mention here that my modest contribution to scientific research and by extension the world of science is primarily in the core area of epidemiology of parasitic diseases. Diseases covered during my nearly four decades of research include **toxoplasmosis** (Olusi et al., 1994; Olusi et al., 1996; Olusi, 1998; Olusi, et al., 2000a&b; Olusi et al., 2001; Olusi et al., 2002a,b&c; Olusi et al., 2008; Olusi et al., 2018; Olusi et al., 2023); **malaria** (Olusi and Abe, 2014; Abe and Olusi, 2014; Ayoade et al., 2014; Olusi et al., 2015; Ibukunoluwa et al., 2015; Olusi et al., 2019; Olusi et al., 2021; Awosolu et al., 2022; Akeju et al., 2022a&b; Olalere et al. 2023a&b; Olusi et al., 2024; Olaniran et al., 2024a,b&c; Olalekan et al., 2024; Olusi et al., 2025); **schistosomiasis** (Olusi et al., 2016; Ajakaye et al., 2016; Awosolu et al., 2020; Aniaguya et al., 2024; Olusi et al., 2024a&b); **trypanosomiasis** (Olusi, 1987; Nwagwu et al., 1988; Olusi, 2004); **fascioliasis**, (Olusi, 1982; Olusi, 1996; Olusi and Amuta; 2001; Akeju et al., 2024) and **onchocerciasis** (Amuta and Olusi, 2000, 2001; Adeshina et al., 2017) among many others. For the purpose of this lecture I will be limiting my discussion to the six diseases mentioned above.

### 5.3.1 Toxoplasmosis

The etiologic agent of the zoonotic disease, toxoplasmosis is *Toxoplasma gondii*, a ubiquitous, single-celled, cyst-forming, obligate intracellular protozoan parasite discovered in 1908 by Nicolle and Manceaux, and named after the North African rodent *Ctenodactylus gundii* in whose tissues it was first recovered. Domestic cats and sister felines are the only known natural hosts of the parasite and are responsible for spreading infective oocysts in their faeces through their

habit of “burying” their faeces after defecation. Tissue cysts containing the bradyzoites stage of the parasite have been recovered from all the mammalian species examined, which explains the worldwide distribution of the disease in nearly all vertebrate animals.

Common routes of infection in humans are: ingestion of undercooked or raw meat containing the parasites’ cysts, ingestion of contaminated vegetables containing infective oocysts, direct oral contact with the faeces of cats shedding oocysts and trans-placental foetal infection with the bradyzoites of the parasites from actively infected pregnant mothers. There is a growing body of literature suggesting that in humans, latent *Toxoplasma* infection is responsible for many neuropathological effects exemplified by psychiatric disorders such as anxiety, schizophrenia spectrum disorders, depression, self-directed violence and/or suicidal behaviour (Rostami et al., 2020). While acute toxoplasmosis is usually asymptomatic, the disease is reputed to have devastating consequences, in pregnant women and individuals whose immune system has been compromised either as a result of HIV infection or malignant infections.

In pregnant women, the effect of congenitally acquired toxoplasmosis which depends on the trimester of pregnancy during which infection was contracted (Olusi, 2008), include spontaneous abortion/stillbirth, hydrocephaly/microcephaly and ocular/neurological complications in childhood or early childhood manifesting in birth of mentally retarded or blind child (Rostami et al., 2020). In individuals immunocompromised as a result of HIV/AIDS, toxoplasmosis have been ranked among the most lethal opportunistic infections (Brun-Pascaud et al., 1994). Studies have suggested that as high as 26% of HIV co-infected patients will develop the AIDS- defining-condition of toxoplasmic encephalitis.

Considering the potential long-term complications and public health importance of toxoplasmosis, a comprehensive understanding of the global prevalence of toxoplasmosis could help shape health policies for international bodies such as WHO and for individual countries. The

prevalence of toxoplasmosis varies from country to country, however, higher prevalence are recorded in communities with high feline proximity and habit of consuming raw or undercooked meat. Significantly higher prevalence of latent toxoplasmosis was associated with countries with low and middle income populations and low human development index in Africa and South America.

Studies have shown that toxoplasmosis is highly prevalent in all human communities infecting nearly one-third of the global human population. In a recent publication, Sengupta et al. (2025) recorded a high global prevalence rate of 31% from a comprehensive meta-analysis synthesis of global sero-prevalence estimates of human toxoplasmosis from 320 studies covering 658,172 individuals across 113 countries and six continents, spanning the period from 1959 to 2020. Continental distribution was Australia - 54%, South America - 45%, Africa - 42%, and Asia - 25%. These findings further highlights the widespread nature of *Toxoplasma gondii* infection with notable regional disparities influenced by environmental, dietary, and diagnostic factors. Reports from Tropical Africa indicate that in spite of the fact that infections are widespread in large parts of human populations, fairly little is known about the epidemiology of *T. gondii* infections (De Rover-Bonnet, 1972). The first report of *Toxoplasma* infection in Nigeria was by Ludlam (1965) who carried out a dye test serological survey of 84 men from the Niger Delta area and found 83.3% and 52.6% seropositive men from the Northern and Southern parts of the study area respectively.

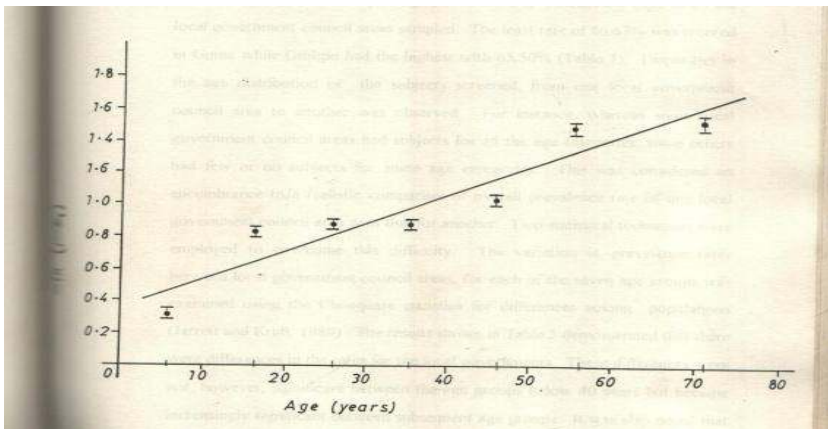
It has been suggested that infection acquired during pregnancy represent a risk of congenital transmission, this underscores the significance of the findings in our study (Olusi et al., 1996), in which 43.7% of 606 women of childbearing age from Benue River Basin had serological evidence of toxoplasmosis. In the same study, we applied a previously published epidemiological model  $-\ln(1-x_t) = \lambda t$  (Blewett, 1983) to our data (Table 1, Figure 1) and we were able to generate an equation for the prediction of the expected incidence

(seroconversion) of *Toxoplasma* infection during pregnancy in any given human population.

**Table 1:** Estimates of Statewide Prevalence Rates for the Different Age Groups.

Age group (years)	% of Statewide population in age group	Prevalence (%pos.)	Corrected* prevalence (% pos.)	Standard error (%)
≤ 10	33.64	27.72	26.1	3.51
11 - 20	18.48	51.40	56.12	3.90
21 - 30	22.64	56.78	58.11	3.18
31 - 40	12.01	59.80	58.12	4.15
41 - 50	6.27	63.10	64.14	3.73
51 - 60	2.50	70.00	77.10	4.20
≥ 61	4.46	73.86	78.35	3.56
Estimated overall prevalence for population			52.39	1.54

\* see statistical addendum



**Figure 1:** Age-specific prevalence of toxoplasmosis in Benue State.

In separate studies, Olusi et al. (1994), Olusi (1998), Olusi et al. (2000a), Olusi et al. (2000b), Olusi et al. (2002), Olusi et al. (2018), and Olusi et al. (2023) recorded 57.00%, **55.6%**, 26.30%, 32.50%, 25.90%, 30.44% and 34.68%, respectively, among different human populations surveyed from Benue, Ondo and Osun States of Nigeria, immunopositive for *T. gondii* infection. Reasons adduced for the widespread infection ranged from chance consumption (Beattie, 1982) of raw/undercooked rat meat (in Benue State where meat from rats is a delicacy), given the fact that consumption of raw meat is not a common practice in Nigeria in general, to close proximity with cats, among others.

### **5.3.2 Malaria**

Human malaria also known as “laziness disease” is caused by any of the following pathogenic plasmodial species, *Plasmodium falciparum*, *P. vivax*, *P. malariae* and *P. ovale*. Additionally, a hitherto known parasite of monkeys, *P. knowlesi*, has now been reported to be infecting humans in Asian countries particularly Malaysia and Indonesia. The disease is transmitted strictly by the female of any of the about 30 to 40 species of *Anopheles* mosquitoes. As mentioned earlier in this lecture, *Plasmodium* parasites induce significant devastating changes in blood cell components causing enormous destruction of red blood cells.

Recently, the impact of global warming on the distribution of vectors of parasitic diseases have been documented. The resultant increase in temperature, heat waves and consequent constant flooding have impacted the distribution of malaria globally since mosquitoes are sensitive to climate change. According to Otubanjo (2013), the following is the global burden of malaria according to species: *P. falciparum* accounts for 40% of global malaria cases, *P. malariae* accounts for 70% of global malaria cases, *P. vivax* accounts for 10% of global malaria cases, while *P. ovale* is very rare.

Malaria remains a global health problem with enormous devastating consequences particularly in tropical countries, including Nigeria, where it has been a menace to the health conditions of both rural and urban populations. When it is not treated promptly, malaria infection could progress to complications such as multi-organ failure in adults, severe anaemia, respiratory distress or cerebral involvement in children and eventual death if not properly managed (Olusi et al., 2024).

Despite the remarkable achievements in the reduction of malaria mortality, the disease had remained the most common cause of morbidity and mortality (Olaniran et al., 2024b).

In the last decade plus, I have lead or jointly conducted research activities on the various aspects of the epidemiology of malaria including the prevalence of the disease among different groups of individual, including those co-infections with other diseases (such as HIV and hepatitis). Some of our researches were also focused on the antimalarial activities of plant extracts (botanicals) and vector control. In one of our studies (Abe and Olusi, 2014), nearly all (96%) of the pregnant women attending a major health facility in Akure harboured the parasite. These malaria-positive women recorded very low parasitaemia and PCV, attesting to the endemicity of the disease and suggesting its involvement in maternal anaemia. Correlation analysis after these same set of women were screened for HIV (Olusi and Abe, 2014), revealed that pregnant women positive for HIV were more likely to be infected with malaria ostensibly due to their immunocompromised state.

Olusi et al. (2019), in a related study recorded a 52.6% prevalence rate of malaria among 93 HIV-positive mothers reporting for routine antenatal examination in Ondo City. The implication of this finding is underscored by the suggestions of previous studies that coinfections of HIV and malaria in pregnancy is associated with various unpleasant outcomes including anaemia, low birth weight, premature births, maternal and neonatal death.

Coinfections of other viruses such as Hepatitis B and C with malaria parasites have been reported to be widespread (Olaniran et al., 2024 a,b & c; Dada et al., 2024) among blood donors screened from Ife Central LGA and Modakeke in Osun State.

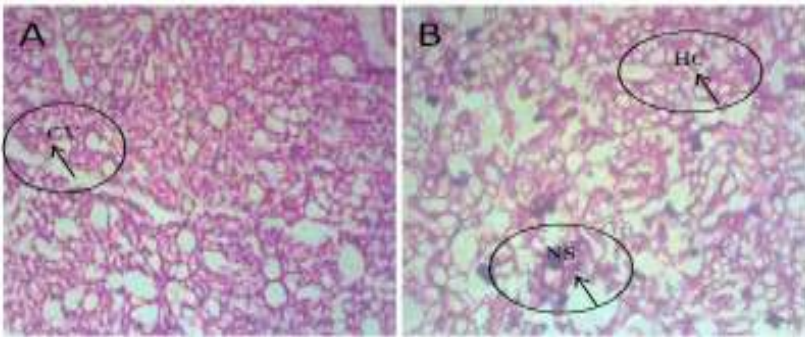
Accurate diagnosis is a pivotal requirement in the management of malaria infection in a population, thus Awosolu et al. (2022) and Olusi et al. (2024) evaluated the sensitivity and specificity of rapid diagnostic test (RDT) and microscopy on different populations of febrile malaria patients, concluding that both tests remain reliable in the diagnosis of malaria infections.

The prevention of malaria through the elimination of the vectors requires a sound understanding of the biology of the mosquitoes. Thus Oniya, Adeyekun and Olusi (2019) investigated the ecological factors favouring mosquito breeding, and found that nearness to residence, temperature, dissolved oxygen, total dissolved solids and pH of breeding site all have varying degrees of effects on the survival of the larvae of different mosquito species. While Olusi et al. (2021) highlighted the major species of malaria vectors in Akure and the selected ecological factors influencing their patterns of their breeding during the two major seasons, Akeju et al. (2022) identified the important physicochemical parameters influencing the abundance of anopheles larvae, that could be manipulated in other to achieve a reduction in the population of the larval stages of this vector.

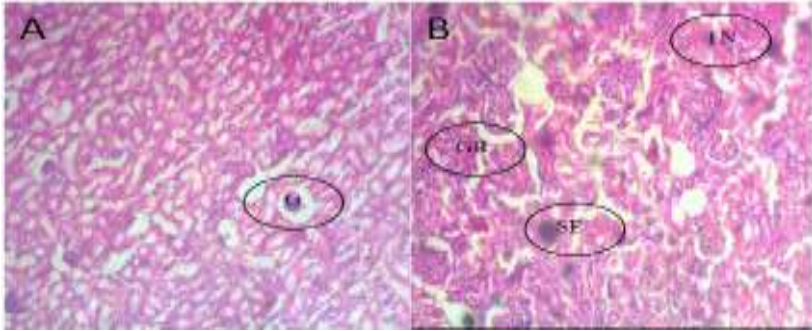
The control of malaria vectors using synthetic chemical compounds has been shown to have toxic residual effects, because of its accumulation into the food chain over time, this has necessitated the search for environmentally friendly alternatives such as botanicals. Ethanolic extracts of *Occimum gratissimum* (scent leaves) and *Azadiracha indica* (neem leaves) oils have been reported to exhibit repellent effect on mosquitoes (Awosolu et al., 2018). Antimalarial activities were demonstrated in the ethanolic extracts of *Cajanus cajan* (L), *Crescentia cujete* L and *Myrianthus preussil* Engl. against

*Plasmodium berghei* (Olusi et al., 2015; Ibukunoluwa et al., 2015). In the absence of any previous report of the antimalarial activity of these plants then, our report was considered ground breaking recipe and premier publication on the antimalarial potentials of the plants.

The liver and kidneys are among the primary organs that are most impacted in malaria infection. Olalekan et al. (2024) and Olusi et al. (2025) reported on the various hepathological and renal changes in the liver and kidneys of humans and mice, respectively, infected by *P. falciparum* and *P. berghei* (Plates 4 & 5). Liver diseases characterized by permanent inflammatory process that predisposes to liver cancer are responsible for over one and half million deaths annually (Olaniran et al., 2024a). Liver function biomarkers are important indices that help in the assessment of disease severity. Olaniran et al. (2024a) reported extensively on changes in amino transferase, aspartate aminotransferase and Albumin profiles in asymptomatic malaria outpatient in Osun State.



**PLATE 4:** Photomicrograph of a section of the liver from mice showing, **B** - liver architectural structure with abnormal central vein, hyaline cast (**HC**) coupled with necrosis (**NC**) compared to the control (**A**) with no necrosis, H and E, Mag. x400

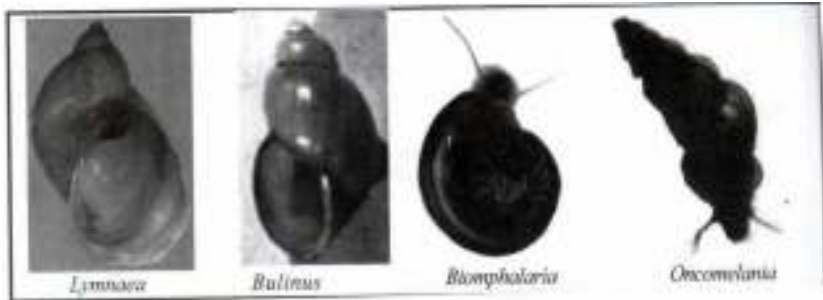


**PLATE 5:** Photomicrograph of a section of the kidney showing, **B-**nephron structure distorted glomeruli (**GR**), congestive acute tubular necrosis (**TN**) coupled with severe edema compared to the control with a normal glomeruli with no necrosis or edema (**A**), H and E, Mag. x400

### 5.3.3 Schistosomiasis

Acknowledged as one of the oldest known parasitic diseases of man, haven been found in mummified remains from ancient Egypt and China (Otubanjo, 2013), schistosomiasis is caused by any of the following species of the genus *Schistosoma*: *Schistosoma haematobium*, *S. mansoni*, *S. japonicum*, *S. intercalatum* and *S. meki*. Man is the only important host of *S. haematobium*, *S. haematobium* and *S. mansoni* occur in most of Africa, especially among rural dwellers who are in frequent contact with natural bodies of water such as rivers, streams and ponds for agricultural, domestic and recreational purposes.

Schistosomiasis, also known as bilharziasis, is a water and snail borne disease, certain species of aquatic snails (Plate 5) serve as intermediate host in the transmission of the disease.



**Plate 5:** Aquatic snails intermediate hosts of schistosomiasis (Source: Otubanjo, 2013)

The different species of schistosomes require their own specific snail hosts, for instance *Schistosoma haematobium* (*Bulinus* sp.), *S. mansoni* (*Biomphalaria* sp.), *S. japonicum* (*Oncomelania* sp.), *S. intercalatum* (*Bulinus* sp.) and *S. mekongi* (*Lymnaea* sp.).

Urinary schistosomiasis is next to malaria as the most common parasitic disease of man in its prevalence and public health implications (Olusi et al., 2016; Ajakaye, et al., 2016). It is endemic in 44 African countries, Nigeria inclusive, with an estimated 101.28 million people at risk, 25.83 million people infected and over 20,000 deaths per year (Vinod, 2008; Otubanjo 2013). Human migration, agricultural and hydro-electricity developments have been blamed for the continuous expansion of the disease to new areas as these projects increase the natural habitats of the snail intermediate host. Human water contact is the principal determinant in the prevalence of schistosomiasis. The disease have been identified as a constant occupational hazard among farmers in agricultural communities. Economic hardship in rural communities resulting in the migration from rural to urban areas has also contributed in no small measure to the distribution and increase in the geographic scope and pattern of the disease.

Several data exist on the distribution of schistosomiasis in Africa. Infection occurs in all age brackets, however, the prevalence of schistosomiasis have been shown to be age related (Tables 2 & 3) The intensity of infection reaches a peak in the school adolescent age of 12-16 years range in many communities and thereafter declines, these observations were also made in the surveys conducted by Awosolu et al. (2020), Olusi et al. (2024a & b), and Aniaguya et al. (2024).

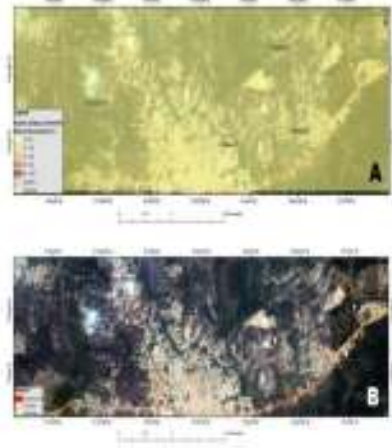
**Table 2:** Prevalence of urinary schistosomiasis in relation to gender, age and intensity of infection (Olusi et al., 2024b).

Variables	Number Examined	Number Infected (%)	Intensity	
			Light Infection (%)	Heavy Infection (%)
<b>Gender</b>				
Male	97	42 (43.3)	32 (76.2)	10 (23.8)
Female	141	34 (24.1)	29 (85.3)	5 (14.7)
Total	238	76 (31.9)	61 (80.3)	15 (19.7)
<b>Age Group</b>				
0-10	66	42 (63.6)	34 (81.0)	8 (19.0)
11-20	46	24 (52.2)	20 (83.3)	4 (16.7)
21-30	10	2 (20.0)	1 (50.0)	1 (50.0)
31-40	17	2 (11.8)	1 (50.0)	1 (50.0)
41-50	32	4 (12.5)	3 (75.0)	1 (25.0)
51-60	15	0 (0.0)	0 (0.0)	0 (0.0)
61-70	52	2 (3.8)	2 (100.0)	0 (0.0)
Total	238	76 (31.9)	61 (80.3)	15 (19.7)

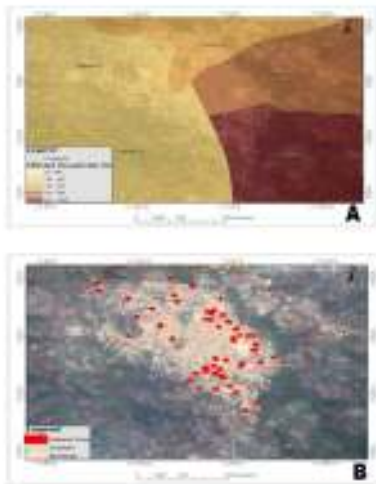
**Table 3:** Prevalence of *Schistosoma haematobium* infection among the sampled population of Aye-Oba and Aye-Amodo in Osun State, Nigeria based on gender and age (n = 427) (Aniaguya et al., 2024).

Variables	No. Examined	No. Positive	Prevalence (%)	P Value
<b>Gender</b>				
Male	224	87	38.8	0.004
Female	203	52	25.6	
Total	427	139	32.6	
<b>Age Group</b>				
1-10	125	66	52.8	0.001
11-20	113	60	53.1	
21-30	29	2	6.9	
31-40	25	2	8.0	
41-50	42	4	9.5	
51-60	26	0	0.0	
61-70	67	5	7.5	
Total	427	139	32.6	
<b>Location</b>				
Aye-Oba	238	76	31.9	0.759
Aye-Amodo	189	63	33.3	
Total	427	139	32.6	

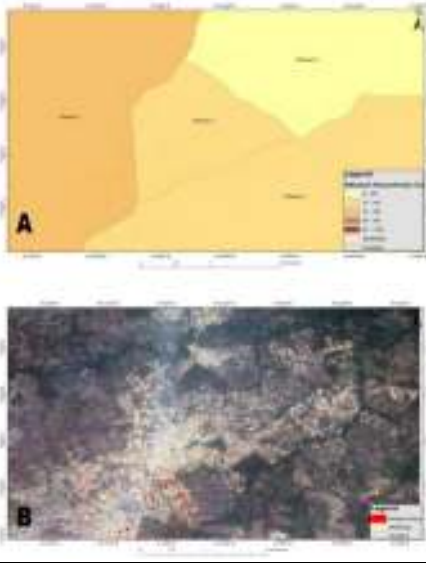
To further understand the epidemiology of urinary schistosomiasis, Olusi et al. (2016) integrated spatial disease and demographic data using ARCGIS 10.0 to determine infection patterns of urinary schistosomiasis in Akure North LGA. Four hundred and sixty three randomly selected households were screened for urinary schistosomiasis, 31.7% of them harboured at least one type of the disease. Spatial statistics showed the infection pattern to be clustered (Figures 3a, 4a & 5a) with contributing areas revealed by hot spot (Figures 3b, 4b, & 5b) in Oba Ile, Igoba and Itaogbolu, respectively. On the influence of environment on the distribution and prevalence of schistosomiasis, Ajakaye et al. (2016) found a negative correlation between infection and elevation, but positive correlation between vegetation, temperature and proximity to water body.



**Figure 3:** (a) Map showing percentage of affected buildings. (b) Map showing hot spots in Oba Ile clusters in Oba Ile clusters.



**Figure 4:** (a) Map showing percentage of affected buildings in Ita Ogbolu clusters. (b) Map showing hot spots in Ita Ogbolu clusters.



**Figure 5:** (a) Map showing percentage of affected buildings in Igoba clusters. (b) Map showing hot spots in Igoba clusters.

### 5.3.4 Fascioliasis

Fascioliasis, an important zoonotic disease principally caused by two species of the genus *Fasciola* (*F. gigantica* and *F. hepatica*), is a major domestic livestock disease characterized by host liver damage. Commonly referred to as liver rot or liver fluke disease, fascioliasis affects farm livestock such as cattle, sheep, goats and buffalo, and is a major impediment to global food production because of its widespread distribution. *F. gigantica* occurs primarily in Tropical regions of Africa and South East Asia, while *F. hepatica* is predominant in the temperate. It is the commonest of the gastrointestinal helminthes of serious economic importance adversely affecting livestock productivity particularly in cattle. The long term result of fascioliasis is gross shortage of dietary animal protein (Olusi, 1996).

Studies on fascioliasis in many parts of Nigeria (Olusi, 1996; Olusi and Amuta 2001; Akeju et al., 2024) points to the fact that the disease is endemic in cattle slaughtered in large abattoirs. On the field, infections are usually chronic and asymptomatic notwithstanding they lead to retarded growth, delayed and reduced productivity, and increased susceptibility to secondary infections. The occurrence of human fascioliasis is a serious condition that is increasingly being associated with changing dietary habit, particularly the consumption of organically grown vegetables in some parts of the world (Europe), where it is impacting on the disease. Estimated number of human infections stand at 360,000 in Bolivia, 20,000 in Equador, 830, 000 in Egypt, and 720,000 in Peru. Over 17 million people are estimated to be infected with liver fluke worldwide with sheep and goats remaining main reservoir of human infections (Otunbanjo, 2013).

### **5.3.5 Trypanosomiasis**

Transmitted principally by flies of the *Glossina morsitans* species, the disease is called animal trypanosomiasis or N'agana of livestock. The parasites reside in intracellular fluids as well as the blood stream of many African wild animals that serve as reservoirs, but it is highly virulent for domestic animals. The disease affects livestock resulting in serious economic loss which has been estimated globally at approximately US\$4.5 billion, direct losses from bovine trypanosomiasis in East Africa are estimated to range from US\$600 million to US\$1.2 billion.

Thrombocytopaenia (platelet aggregation) is a complication of trypanosomiasis. Though proteases are known generally as inducers of blood coagulation and platelet aggregation, whether or not active proteases are released by *T. brucei* was undertaken by Olusi (1987) and Nwagwu et al. (1988) as a first step towards defining the biochemical basis of platelet aggregation in trypanosomiasis. These authors were the first to report on the extracellular release of proteases by *T. brucei* in 1988, through a WHO grant to Prof. N. Nwagwu of the Department of

Zoology, University of I badan. The enzymatic properties of these proteases were later publish in 2004 (Olusi, 2004).

### 5.3.6 Onchocerciasis

Onchocerciasis (commonly known as river blindness) is a debilitating disease caused by *Onchocerca volvulus*. It is one of the neglected tropical diseases and a major public health problem especially in West Africa. Transmitted through the bite of infected black flies of the genus *Simulium*, the disease manifests as a reaction to the presence of microfilariae in the skin and in the eyes. The disease is a major obstacle to development within its region of endemicity as it results in both blindness and dermatological problems (Plate 6a & b).



**Plate 6:** Blindness and depigmentation of skin caused by onchocerciasis

Endemic onchocerciasis areas include 28 countries in the savannah and forest zones of Africa, 6 countries in Latin America and in Yemen. In Africa, an estimated 85 million people are at risk of this infection while approximately 20 million are infected. According to Thylefors and Alleman (2006), the disease is considered the fourth leading cause of blindness globally and second to polio as a cause of long term disability and disfiguring of skin in endemic areas.

It is estimated that there are more than 25.7 million people infected with *O. volvulus* with 746,000 visually impaired while 265,000 are blinded. The Disability Adjusted Life Years (DALYs) for the disease is estimated at 389,000 worldwide (Otubanjo, 2013). Amuta and Olusi (2000, 2001), Amuta et al. (2000, 2002) and Adeshina et al. (2017) screened various human populations in Benue and Ondo States for onchocerciasis via skin snips and serological methods and recorded prevalence rates that ranged between 37% and 75%. These high prevalence rates of infection calls for concern considering the devastating manifestations of the disease.

## **6.0 ANTHROPOGENIC ABIOTIC AND BIOTIC STRESSORS AFFECTING PARASITE TRANSMISSION**

The majority of modern day ecosystem stressors are driven by industrialization combined with human population growth, consequently the stressors affecting the transmission of parasites can be divided into the following broad groups:

### **(a) Climate change**

The multiple components of climate change, including temperature, precipitation and atmospheric carbon dioxide, have been extensively studied individually (Thomas, 2004), but the interactions between these environmental stressors and their consequent effects on parasite transmission are complex. Thus, there is considerable uncertainty about how future climate variations and changes will affect disease dynamics (Barber et al., 2016). Multiple stressors might affect multiple life-history traits, potentially influencing both parasite and host fitness (Paljmans, 2009). In combination, these stressors may counteract each other, such that the overall rate of parasite transmission remains unchanged. Higher temperatures, for example, often increase parasite growth, reproduction and infectivity (Macnab and Barber, 2012), yet can also increase parasite mortality, which account for why there is no net change in the number of transmitted parasites (Scott and

Nokes, 2009). Poikilothermic hosts are particularly vulnerable to temperature shifts, but also show remarkable adaptations and such responses by the host can be damaging to the parasite. Some fish, for example, exhibit adaptive behavioural traits to reduce transmission risk, by actively selecting thermal conditions that are detrimental to parasites behaviour (Mohammed et al., 2016).

Different environmental parameters may have additive, multiplicative or antagonistic and nonlinear effects on transmission. For example, flooding events can be a key driver of some water-borne disease epidemics (Cann et al., 2013), while drought conditions cause hosts to aggregate at sites where water is available, amplifying transmission and triggering outbreaks of vector-borne diseases such as African horse sickness and Rift Valley fever (Baylis et al., 1999). Other environmental transmission relationships are likely to be a result of host-parasite range shift due to climate warming. This can change the distribution of vector-borne diseases, including malaria (Park et al., 2016) and Rift Valley fever (Leedale et al., 2016).

### **(b) Pollution**

Pollutants can cause sub-lethal physiological stress to hosts and hence reduce their capacity to withstand parasite invasion and/or proliferation, potentially increasing infection levels indirectly (France and Graham, 1985). However, pollutants also impact parasites themselves, and in aquatic ecosystems, both the infective stages of parasites and their intermediate hosts can be highly sensitive to their effects (Lafferty, 1997). Heavy metals can inhibit the release of trematode cercariae from molluscan hosts, as well as impair their swimming behaviour and longevity (Cabello et al., 2013). Pharmaceutical pollutants are widespread stressors likely to affect host susceptibility to diseases. The scale of this threat is increasingly more apparent in aquaculture, for example, in Chilean salmon farms alone, hundreds of tonnes of antibiotics are used annually (Cabello et al., 2013). Eutrophication, arising from excessive nutrient input, is another

important stressor of aquatic ecosystems associated with elevated intermediate host densities, parasite fecundity and increased prevalence of certain pathogenic infections (Johnson and Carpenter, 2008). Other forms of pollution are less well studied with regard to disease transmission.

**(c) Habitat loss and fragmentation**

Habitat alteration due to climate change or anthropogenic activities poses a major threat to the ecosystems, often leading to substantial loss of biodiversity, ecosystem functioning and services, and reduced resilience to external stressors (Edwards et al., 2014). This in turn may alter host-parasite interactions, by either increasing (Hussain et al., 2013) or decreasing (Evans et al., 2009; Chasar et al., 2009) infection levels, depending on nuances of host and parasite life history. The effects of habitat change can even have contrasting effects on closely related parasite species infecting the same host. For example, sunbirds in disturbed habitats exhibited increased prevalence of *Plasmodium lucens* but decreased prevalence of *P. megaglobularis* (Chasar et al., 2009).

**(d) Host density and farming intensification**

Over the past 50 years, there have been unprecedented changes in farming practices and associated land use (FAO, 2015). Although natural and managed forestry currently occupies about 30% land area, the impact of deforestation and land use intensification, especially on soil degradation is significant. Growth in crop production and livestock has been driven by the demand for higher yields. Livestock production is the largest user of agricultural land, accounting for more than 30% of the earth's ice-free terrestrial area (FAO, 2015), but aquaculture is the fastest growing food sector (FAO, 2015). Modern and large-scale farming practices typically rely on concentrating and containing inbred hosts, which can increase host exposure to and facilitate parasite transmission (Iguchi et al., 2003). High host density is particularly

important for tick-borne pathogens (Rosa and Puglese, 2007), as these vectors are relatively immobile and host-parasite contact frequencies tend to be driven by changes in host abundance and/or behaviour. Chronic stress induced by high stocking densities in aquaculture can have important implications for fish immuno-competence (Sadhu et al., 2014), but relationships with infection levels are variable. While high host densities can promote greater parasite population densities, the number of specific parasites per host may be reduced (Laguerre and Poulin, 2015). This 'dilution effect' is illustrated by a reduction in directly transmitted sea lice at the high host densities in salmonid cage aquaculture (Samsing et al., 2014).

#### **(e) Urbanization**

While density-dependent transmission of human parasites may be expected to increase with high population densities and ownership of companion animals, decreased human-wildlife contact and better sanitation in cities of developed countries generally point to lower levels of disease transmission among such populations (Robert et al., 2003), although there are exceptions. Dengue, for example, is more prevalent in urban areas due to the provision of suitable human-created microhabitats for the *Aedes* mosquito (Kearney et al., 2009). Urban environments with high human densities are potentially more vulnerable to water-borne or faeco-orally transmitted parasites if investment in sanitation infrastructure is neglected or disrupted due to socio-economic unrest.

In developing countries, human or wildlife conflicts can be a major issue. Most emerging and re-emerging human infectious diseases (EIDs) are zoonotic, typically with origins in mammalian wildlife (Jones et al., 2008) or interactions between wildlife and domestic animals (Plowright, 2015).

By 2050, it is estimated that almost half of the world's population will live in the tropics, of which approximately 66% are likely to be living in urban contexts (United Nations Department of

Economic and Social Affairs, Population Division, 2015). Millions of individuals are also expected to migrate during their lifetimes due to factors associated with the urban-rural cycle, extreme weather events, economic necessities, water and food security, and conflicts (United Nations Department of Economic and Social Affairs, Population Division, 2011).

**(f) Biodiversity loss**

Current extinction rates are estimated to be 100 to 1000 times greater than background levels (De vos et al., 2015), with biodiversity loss being one of the hallmarks of the Anthropocene (Dirzo et al., 2014). Loss of host diversity can reduce disease risk directly or indirectly through the associated loss of parasite diversity (Lafferty, 2012). For example, reduced risk of African sleeping sickness in humans (Bourn et al., 2001) has been related to the loss of wildlife host biodiversity (Reid et al., 2000). Wildlife biodiversity is often correlated with human infectious disease risks. Examples include correlations between mammalian biodiversity and global biogeographic patterns of human infectious diseases (Muray et al., 2015), elevated likelihood of observing emerging infectious diseases (Jones et al., 2008) and increases in human pathogen richness and prevalence of some diseases (Dunn, 2010).

**(g) Altered interspecific interactions**

Changes in host interactions, often linked to the stressors listed above, can drive the emergence of diseases in new hosts. I have already highlighted this problem in association with increased human wildlife contact, but this in turn might be altered by a range of non-human interactions.

Parasites have a fundamental role in food webs (Lafferty et al., 2006); thus, anthropogenic changes that reduce the density of higher trophic level species that feed on larval parasite stages (Johnson, et al., 2006) could directly increase disease transmission to competent hosts.

Parasite may also indirectly disrupt predator-prey interactions (Hatcher et al., 2006) and abiotic factors may affect trophic transmission by altering host foraging activities (Thieltges et al., 2008). In addition to these altered parasite-predator-prey interactions, parasites can affect native invasive host interactions (Tomkins et al., 2003); newly invading hosts either bring with them novel pathogens to which native hosts do not have resistance, or having escaped their own native parasites they can dilute the pool of susceptible native hosts (Dunn, 2009).

Abiotic or biotic stressors may even drive symbionts to adopt parasitism, for example, where there is high competition on the host (Skelton et al., 2016). Artificial manipulation of species interactions can be used in biocontrol, as in the case of *Wolbachia* infection of mosquitoes, which reduces their vectoring capacity (Waltz, 2016).

#### **(h) Interacting abiotic and biotic factors**

The above list is not comprehensive, but rather highlights some of the key abiotic and biotic factors which may act together as 'cocktails' of stress, with implications for increasing or decreasing disease risks. Identifying the direct and/or indirect factors responsible for changes in disease risk is challenging because multiple stressors act simultaneously on both parasites and their hosts. Depending on habitat and season, the peak impact of different abiotic stressors can occur in or out of phase with one another; thus, while some organisms may be exposed to multiple stressors simultaneously, others will experience them sequentially. Yet, the consequences of multiple, interacting environmental threats for parasite transmission remain largely unclear: when they co-occur temporally and spatially, their combined effects may be additive, antagonistic or synergistic (Noges et al., 2016). For example, while elevated sea water temperatures increase mortality rates of oyster larvae, this can be offset by simultaneous water acidification, which reduces the growth of pathogenic bacterial infections (Prado et al., 2016). On coral reefs, the interaction between ocean acidification and warming contributes to coral bleaching and reduced disease

resistance, leading to increased pathogenicity of existing pathogens and the emergence of new diseases (Athony et al, 2007). These two examples are rare, because, compared with terrestrial and freshwater systems, marine systems are often neglected with regards to assessing the impact of environmental stressors (Burge, 2014).

## **7.0 SOCIOECONOMIC IMPACT OF PARASITIC INFECTIONS**

The warm climates of the tropics particularly sub-Saharan Africa is disproportionately being inhabited by parasites where they constitute the greatest threat to the health and socio-economic status of the people. Though temperate zone do have problem of parasites, but, over time, they have either been eradicated or controlled and where they still exist, these industrialized countries of the temperate regions have acquired the manpower, materials and technological know-how cum resources to minimize their impact.

Life in the tropical Africa like most third world countries, is bedeviled by the terrible combination of poverty, ignorance and diseases, and according to Suleiman et al. (2022), parasitic infections remained major obstacles to economic development and a better life in developing countries. Parasitism contributes directly to malnutrition which in turn reduces resistance to infectious diseases. Thus, the eradication or control of parasitic infections is expected to have a considerable impact on the health condition of the people. The World Health Organization defined health as “a state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity”

There is a complex relationship between parasitic diseases and economic development. According to Oliver (1974), it is not easy to assess or measure the socio-economic impact of parasitic diseases, it is also not easy to estimate the impact of socio-economic factors on parasitic infections and their control. These observations notwithstanding, the adverse effects of parasitic diseases on economic

development is widely accepted as an article of faith. Indeed Watson (1978), went as far as saying that “parasitic diseases probably contribute the most to retardation of socio-economic development, and he declared them to be “public enemy number one”. Little wonder that the relevance of parasitology to human welfare is a topical issue today.

While birthing its special programme for Research and Training in Tropical Diseases (TDR), the World Health Organization selected the following five parasitic diseases, together with leprosy as the six most important diseases of man that deserved special attention in the tropics: malaria, schistosomiasis, filariasis, trypanosomiasis and leishmaniasis. The diseases have been selected for the following reasons: (a) They are wide spread and in some instances even affect every member of the community, thereby constituting a major public health problem. (b) There were no satisfactory methods of controlling the diseases in the prevailing circumstances of the tropical countries. and (c) They greatly retard socio-economic development. The two other diseases considered for addition were hookworm and Guinea worm.

These parasites are also responsible for staggering financial losses. Malaria, for example, is usually chronic, debilitating, and periodically disabling disease and it affects the productivity of a community because the wet season which is the peak period of agricultural activities coincidentally is the peak period of transmission of the disease. Similarly the seasonality of Guinea worm attacks coincides with the planting and harvesting seasons. Thus the crippling effects of these disease has been known to account for great economic losses.

In situations where malaria is prevalent, the number of hours of productive labour lost multiplied by the number of malaria sufferers yields a figure that can be charged as loss in manufacturing of goods, in the production of crops, or in the earning of a gross national product. Nations that import goods from countries infected with malaria, schistosomiasis, hookworms and many other parasitic diseases, pay

more for these products than they would, had the products been produced without the burden of diseases.

National and international efforts to increase productivity and standard of living in less developed countries sometimes inadvertently increase parasitic diseases. For example, schistosomiasis in Egypt increased after the construction of the Aswan High Dam on River Nile. Smaller dams for drainage and agriculture have promoted transmission of schistosomiasis, onchocerciasis, dracunculiasis and malaria. The World Bank loaned Brazil funds to pave highways into the Amazon region to settle poor urban workers for farming, ignoring advice from their own agricultural experts. This action of the World Bank and government of Brazil produced an increase in malaria and spread of the disease to new foci when the migrants returned to the cities after their farms failed.

Finally, the ravages of trypanosomiasis on man and cattle have been reputed to render an estimated area of 7million square kilometers unsuitable for crop farming and livestock raising in tropical Africa.

## **8.0 CONTROL OF PARASITIC DISEASES**

### **8.1 Concepts of Control, Elimination and Eradication**

A distinction must be made between the terms 'control', 'elimination' and 'eradication'; the latter term is often used inappropriately and should be employed with caution. The International Task Force for Disease Eradication (ITFDE) was established in 1988 to evaluate systematically the potential for eradication of candidate diseases and to identify specific barriers to eradication. The Task Force was reconstituted in 2001 to evaluate the situation at that time. The ITFDE defined eradication as 'reduction of the world-wide incidence of a disease to zero as a result of deliberate efforts obviating the necessity for further control measures'. The original ITFDE reviewed more than 90 diseases, 30 of them in depth, and concluded that dracunculiasis, rubella, poliomyelitis, mumps, lymphatic filariasis and

cysticercosis could probably be eradicated using existing technology. The term 'elimination' is increasingly being used to replace the term 'eradication', which should be used only in Global term.

The Dahlem conference held in Berlin in 1997 (Dowdle and Hopkins, 1998) also considered these issues in some details and introduced the term extinction to classify an organism that did not exist on the planet contrasting with smallpox, which had been eradicated as a cause of disease but stocks had been retained in secure laboratories. The use of the term elimination is now regarded as referring to the removal of the organism from a defined geographical region "local eradication", which creates problems for quantification of achievements towards the goal. The accepted position being that the disease is not eradicated but no longer requires ongoing investment in control and it is maintained at a level at which the problem is no longer a significant health burden (Dowdle and Hopkins, 1998).

## **8.2 Examples of Parasite Elimination and Vector "Eradication"**

The classic eradication programme was that of smallpox which achieved its target in 1977. To date, no parasitic disease has been eradicated, although attempts to eradicate Guinea worm were underway (Hopkins et al., 2002; Ruiz-Tiben and Hopkins, 2006), some degree of successes were later achieved via Bill Gate's sponsored programme. Additionally, successful "local eradication" has been achieved in some restricted geographical or epidemiological situations. For example, onchocerciasis has been eliminated from several parts of Kenya and from the Nile Jinja in Uganda, by using DDT to remove the local vectors (*Simulium neavei* and *S. damnosum*) (Davies, 1994).

The Onchocerciasis Control Programme (OCP) in West Africa had achieved the same goal, eliminating particular cytoforms of the *S. damnosum* complex using aerial application of insecticides. Local elimination has also been achieved in the case of the malaria vector *Anopheles gambiae* from Brazil in the late 1930s using larviciding

measures and house spraying with pyrethrum, a success repeated in early 1940s after the same species had been introduced in Egypt; *Glossina palpalis*, the tsetse fly vector of human trypanosomiasis was eliminated from the Island of Principe in 1905 by trapping out flies using sticky back packs on plantation workers; animal trypanosomiasis from parts of North-East Nigeria by ground spraying of tsetse flies resting sites with persistent doses of DDT; *Aedes aegypti*, the vector of yellow fever, in parts of Central and South America. Local anti-mosquito spraying has eliminated lymphatic filariasis from the Solomon Islands with no evidence that over a 20-year period there has been any resurgence; through selective treatment, filariasis due to *Brugia malayi* was eliminated from Sri Lanka with DEC, anti-larval measures (host plants killed by herbiciding), house spraying with DDT as part of the malaria eradication programme and environmental improvements.

Chemotherapeutic approaches have eliminated filariasis (due to *Wuchereria bancrofti*) from Japan, South Korea and Taiwan in Asia and Suriname and Trinidad and Tobago in the Americas (WHO, 1992; 1994). Filariasis has also been eliminated as a public health problem in large areas of China where it seems transmission has been stopped for a period of over 10 years (WHO, 2003). Long-term “elimination programmes have been successful against hydatid disease in Iceland, New Zealand and Cyprus; and malaria was eliminated from Sardinia by DDT spraying as well as in other marginal areas of distribution such as North Africa, Greece and parts of Turkey and the Middle East. One noticeable feature of these successes is that many examples cited above refer to islands or isolated populations or areas where the parasite is at the edge of its geographical range. Clearly, the advantages of isolation and a greater ability to control animal or human population movements are important.

Elimination or global eradication of any disease is difficult to achieve and costs increase per case detected, controlled or averted as the end point is reached. Whereas the high cost of eradication or local

elimination programmes may be justified as they are time limited, disease control implies a long-term commitment. Any control programme must be cost effective and should reduce the target disease to a level at which costs are sustainable by the local community or by public or private healthcare systems. Control seeks to bring the problems to a level at which, the disease is no longer of public health importance with morbidity reduced to an acceptable level within the community, an absence of mortality and, if appropriate, greatly reduced levels of disability. To translate the level of control achieved to eradication or elimination status requires a vastly increased cost per case treated or prevented which, for financial and ecological reasons, may never be feasible.

### **8.3 Components of Control**

Many parasitic diseases are zoonoses, defined as 'those diseases and infections whose agents are naturally transmitted between other vertebrate animals and man' (WHO, 1979). A list of recognized parasitic zoonoses is provided by the WHO (1979). Ostfeld and Keesing (2000) provided an up-dated list of vector-borne infections of potential public health importance, while an analysis of all emergent and re-emergent infections (Taylor et al., 2001) has identified that 75% of emerging pathogens are zoonotic and that such organisms are more than twice as likely to emerge as non-zoonotic ones. However, viruses and protozoans are more likely to emerge than the macroparasites such as helminths. The important zoonoses for which reservoir host control can have a cost-effective impact are leishmaniasis, echinococcosis and cysticercosis. Treatment of cattle with trypanocides in Uganda was the strategy used to reduce the role of cattle as a reservoir of *Trypanosoma rhodesiense* sleeping sickness (Fe'vere et al., 2005). However, the presence of animal reservoir host may be a major impediment to controlling a disease particularly if the habits and habitats of the animal host prevent the intervention either on the grounds of practicality or for reasons such as protected status of host species e.g. primates or

endangered species status. The ITFDE recognizes that the existence of an animal reservoir precludes the likelihood of the eradication of the infection.

#### **8.4 Community participation in parasitic disease control**

The drive towards primary healthcare following the Alma-Ata declaration of 1978 provoked a greater degree of involvement of Interventions for the control of parasitic diseases in healthcare through (1) the use of community leaders to support various programmes; (2) the identification of personnel to undertake health activities on a voluntary basis; and (3) emphasizing the importance of such activities in community well-being. The topic of community participation has been reviewed by Curtis (1991) who provided a series of examples in vector-borne disease control. MacCormack (1991) provides an insight into the underlying principles of sustainable vector control in a community context emphasizing that success in small pilot projects depends on particular characteristics such as leadership, a responsive, well-motivated and well-educated community support, incentives from agencies and insecticide manufacturers, and ease of communication.

#### **8.5 Why it is necessary to control parasitic diseases**

Considering their overwhelming impact, there is an urgent and compelling need to eradicate or control parasitic diseases in tropical Africa. This will not only lead to the improvement of human health but also raise the level of the quality of life and productivity of the people, which will in turn make possible the increased production of the much needed high quality protein. The justification for the application of control measures might not be readily appreciated for the following reasons: There appear to be the danger of rapid deterioration of the environment, and it is feared, for instance, that controlling parasitic diseases will lead to increased population and greater human activity (Watson, 1978). This will consequently increase the demand for food, which cannot be met because the fragile and unstable tropical African

soils that cannot withstand the more intensive cultivation required (Ford, 1979).

Added to this, is the fact that the application of fertilizers and pesticides to achieve greater productivity often results in increased cost of food which the poor rural population cannot afford. In the same vein, the control of livestock diseases like bovine trypanosomiasis, will remove the constraint to the size of cattle herd a farmer can keep. Consequently, there is the real danger of surpassing the carrying capacity of the land with the resultant consequence of overgrazing and its attendant irreversible vegetational and climate changes in ecologically unstable areas like the Sahel zone of West Africa (Jordan, 1979), Ormed in Taylor and Miller (1978). There is therefore the possibility that the freeing of man and his livestock from diseases may unleash new sets of problems such as threat of starvation resulting from population explosion, the negative consequences of which might even be greater than that of parasitism. According to Ukoli, 1984 “There is little point in removing the restraint of population and replacing it with that of starvation”

These arguments may appear plausible, but they are not sufficient grounds for not introducing control measures, because, it will be inhuman, to say the least, to leave the people at the mercy of disease, which will amount to condemning them to perpetual suffering in their attempt to eke out a subsistence living from an apparently hostile environment. The attainment of wellbeing and a reasonable quality of life for the people which is the desired goal does not depend only on the control of diseases. It should therefore be integrated with other aspects of development, for example, land use, improvement in agricultural practices, the need for birth control and education, all of which will generate the capacity in the people to seek solutions to their problems.

Just as it may not be possible to identify the nature and magnitude of the likely social benefits derivable from the control of certain parasitic diseases such as schistosomiasis (Prescott, 1979), the direct benefits of controlling other diseases like onchocerciasis may not

become immediately apparent because of the complicating effects of the incidence of other diseases like malaria, trypanosomiasis (of man and animals), malnutrition, and other facts that the victims are already compelled to contend with in the cultivation of the impoverished soils (having been forced to vacate the fertile lands as a result of the scourge), which is further degraded by their activities (Melville, 1979)

In the case of trypanosomiasis, the preoccupation with the problems of tsetse flies control, may actually be diverting attention from seeking solutions from equally pressing problems which constitute a limitation to the development of the land (Jordan, 1979). Indeed if the tsetse flies problem were eliminated or were not there ab initio, man would still have had to grapple with these problems in order to boost the productivity of the land. Therefore, the successful control of trypanosomiasis or any of the other equally important parasitic diseases, is really of little benefit if it is not integrated into a general resource development programme (Ford, 1979).

## **9.0 A PARASITE-FREE WORLD: ACHIEVABLE OR WISHFUL THINKING**

### **9.1 What happens when there are no parasites?**

What would happen if all parasites disappeared? This intriguing thought experiment, once posed in BBC Earth's "Strange and Beautiful" series (Jones, 2015), is a useful exercise for considering the ecological roles of parasites in the ecosystems. So far, humanity has managed to drive only one of its parasites to extinction: Variola, the viral genus that causes smallpox. Until it was eradicated in 1980 through global-scale public health efforts, naturally occurring smallpox was one of the most dominant drivers of mortality in recorded history, killing 500 million people in the 20th century alone (Koplow, 2003). By many metrics, the elimination of viruses, bacteria, protozoa, and parasitic worms and arthropods (here, collectively referred to as "parasites") would contribute to reduced rates of human mortality, less

disability, improvements in quality of life (Murray et al., 2012), and even reduced poverty (Bonds et al., 2010). The disappearance of parasites would also substantially benefit livestock production (Perry and Randolph, 1999) and wildlife conservation (Daszak et al., 2000), particularly in developing countries.

But, while the eradication of disease agents is critically important for ensuring human well-being, parasites often play important yet underappreciated roles in nature. Every ecosystem on earth contains parasites; indeed, virtually every metazoan is host to at least one parasite species (Poulin and Morand, 2000). Parasites represent 40% of described species (Dobson et al., 2008) and are at least twice as rich in species as their vertebrate hosts (Poulin and Morand, 2004). Considering only viruses in the ocean, a projected ~41,030 species exist, with the standing stock of carbon in viral biomass estimated at ~200 megatons (Suttle, 2005). Despite this ubiquity and abundance, the diversity of parasites is poorly known (Poulin and Morand, 2000) and our understanding of parasites' ecological influence remains rudimentary (Gomez et al., 2012; Hatcher et al., 2012).

Here, I attempt exploring a "world without parasites" as a vehicle for identifying the ecological changes that would accompany the elimination or loss of infectious organisms. **The elimination of all parasites is improbable and perhaps impossible**, but as Holt (2010) noted, "it can be illuminating to ponder all kinds of implausible and radical scenarios, in effect bracketing the real world with visions of possible worlds". This discussion is limited to parasites of animals, focusing on empirical and theoretical research on parasites' influence at several levels of ecological organization (individual, population, community, and ecosystem), posing hypotheses for general mechanisms by which parasites may be ecologically influential, and identifying attributes of parasites, hosts, and ecosystems that may predict a strong ecological influence of parasites. Focus is on ecological effects of parasites, but evolutionary effects are also likely to be important (Holt, 2010; Stringer and Linklater, 2014) Emphasis is on

those cases where parasites' effects are likely to be consistent across contexts, excluding impacts of parasites that are likely to be highly context-specific.

### **9.1.1 Individuals and populations**

The fitness effects of parasites on individual hosts, although negative by definition (Combes, 2001), vary strongly among species. A parasite may reduce its host's growth, prevent it from reproducing, or change its behaviour. Parasites may even have positive collateral effects on a host (e.g. by competing with other more virulent parasites within the same host). When individual-level effects accrue, parasites may also influence host populations in a variety of ways.

### **9.1.2 Parasites influence host immunity**

A growing body of research illustrates the ecological importance of within-host interactions among parasites, as well as interactions between parasites and the host's immune system. Although co-infections would be impossible in a world without parasites, the present discussion is focused on addressing interactions among co-infecting parasites. Even without co-infecting species, the absence of parasites can drive unexpected outcomes in host health, through effects on host immune function. Some chronic illnesses of humans including allergies and autoimmune diseases, have been linked to a lack of exposure to parasites, particularly worms (Okada et al., 2010). Paradoxically, parasites may have net positive fitness benefits for hosts if the immunologic consequence of parasite absence takes a sufficiently high toll on host fitness (Holt, 2010; Stringer and Linklater, 2014). In the absence of parasites, hosts should shed costly and useless immune defenses. But nature abhors a vacuum. Hosts that initially lost their immunity would later be susceptible to re-infection by newly evolved parasites (Stringer and Linklater, 2014; Jones, 2015).

Parasites affect the dynamics of host populations, many parasites affect the rate of host population growth and total population size. Indeed, there are numerous examples demonstrating regulation of wild host populations by parasites, including both "micro-parasites" and "macro-parasites", whose fitness effects on hosts are independent and dependent, respectively, on the number of initial infecting transmissible stages (Lafferty and Kuris, 2002). For instance, crustacean parasites such as isopods and copepods can reduce growth, reproduction, and survivorship of coral reef fishes, resulting in population-level regulation of hosts (Forrester and Finley, 2006). In British heathland ecosystems, experimental application of anti-helminthic drugs (which clear red grouse of infections with the parasitic nematode, *Trichostrongylus tenuis*) dampened the boom and bust cycles that characterize the population dynamics of infected grouse (Hudson et al., 1998). But parasites need not kill their hosts to exert regulatory effects on host populations, many parasites **castrate their hosts** (e.g. the bacterium *Pasteuria ramosa* in *Daphnia* spp: Ebert et al., 2004), thereby regulating host populations (Decaestecker et al., 2005). Removal of such influential parasites may lead to loss of regulation of host populations and an increase in host abundance.

### **9.1.3 Parasites alter the composition of ecological communities**

The effects of parasites vary among host species, and this can lead to community level effects. Many examples, mostly accumulated over the past several years, demonstrate that parasites can alter the composition of communities through demographic (density-mediated) or morphological/physiological/behavioural (trait mediated) indirect effects. Because these effects have been reviewed elsewhere (Gomez et al., 2012; Hatcher et al., 2012), I will give only a few illustrative examples here. In a classic case of a density-mediated indirect effect of parasites and of parasite-mediated apparent competition (an interaction that looks like competition between two species but is actually caused by a third factor; Stringer and Linklater, 2014), the invasive gray

squirrel (*Sciurus carolinensis*) was able to replace the native red squirrel (*Sciurus vulgaris*) throughout the UK because the invader brought with it a parapoxvirus. Only the native red squirrel experienced substantial parasite induced mortality, allowing gray squirrels to expand into the niche vacated by the natives (Tompkins et al., 2003).

Parasites may also have trait-mediated indirect effects. In the rocky intertidal zone of New England, periwinkle snails (*Littorina littorea*) infected with a trematode parasite eat less algae than do uninfected snails, probably due to infection-related changes in the digestive system, as a result, edible macroalgal species are more abundant in the presence of infected snails than in the presence of uninfected snails, with implications for the other intertidal species that use this macroalgae as habitat and food (Wood et al., 2007).

Finally, parasites may affect interactions among free-living species (Holt, 2010; Mordecai, 2011, Stringer and Linklater, 2014), for example, the presence of larval trematodes increases intertidal diversity on New Zealand mud flats by changing interactions between host bivalves and the organisms that depend on bivalve shells for habitat (Mouritsen and Poulin, 2005). Whether by effects on host density or traits, or on species interactions among hosts, the composition of free-living communities can be radically reshaped by parasites. In addition to affecting the composition of communities, parasites may also affect variability in composition (i.e. food web stability), but whether the presence of parasites generally increases or decreases such variability is controversial and may be context-dependent (Lafferty et al., 2008; McQuaid and Britton, 2015). Parasites could increase stability in community composition by regulating host populations (Anderson and May, 1978), contributing "weak links in long loops" (Neutel et al., 2002), or by producing apparent competition (Dobson, 2004).

Alternatively, parasites could decrease stability by increasing the length of food chains (Williams and Martinez, 2004), overwhelming stable predator-prey links with unstable parasite-host links (Otto et al., 2007), or merely by contributing additional species to total community

richness (Chen et al., 2011). The presence of parasites is generally thought to decrease the robustness of food webs (i.e. the likelihood of secondary extinctions occurring after a primary species loss), this is primarily because parasites themselves are prone to secondary extinctions (Chen et al., 2011; McQuaid and Britton, 2015). Whether there is a general role for parasites as a stabilizing force in free-living food webs remains an open question.

As suggested in the example of gray squirrels, parasites may mediate the ability of non-native species to invade a community (Tompkins et al., 2003). According to the "**enemy release hypothesis**", when a species is introduced into a region to which it is not native, it experiences weaker population regulation by natural enemies (e.g. parasites, predators) than it would in its native range (Prenter et al., 2004). Indeed, host species of various taxa are infected by twice as many parasites in their native ranges than in their invaded ranges (Torchin et al., 2003). If parasites disappeared, native and invasive species might be placed on equal footing, that is, release from parasitic enemies would benefit both native and invasive species. Alternatively, if the parasites of invasive hosts facilitate invasion by infecting native hosts (the "biological weapons hypothesis", as in the case of the gray squirrel; Tompkins et al., 2003), parasite loss might result in a disadvantage to invasive species and reduced rates of invasion. Native parasites also have the potential to slow the progress of invaders. For instance, European settlers were repelled from large swaths of land in southern and central Africa by trypanosomiasis, so that patterns of early European settlement mostly matched areas that were trypanosomiasis-free (Ford, 1971; Beinart and Coates, 1995). Thus, whether the loss of parasites will increase or decrease invasibility of an ecosystem ultimately depends on the relative fitness effects of invasive parasites on native and invasive hosts, the propensity of native parasites to infect invasive hosts, and other factors.

#### **9.1.4 Parasites alter trophic interactions and predation rates**

In a world without parasites, energy should become available to free-living consumers that would otherwise have been siphoned away by parasitic consumers (Holt, 2010; Jones, 2015), this follows from the expectation that the loss of parasites should ameliorate individual-level fitness effects associated with parasitism (e.g. make prey larger) and release some free-living species from regulation (e.g. make prey more numerous). But parasites can also influence host individuals through sub-lethal effects, which affect their quality and availability as prey (Holt, 2010). Whether elimination of a parasite species will increase or decrease energy flow to consumers/predators will therefore depend on the balance between the regulatory and individual-level effects of the parasite.

Thus, it is suggested that, the ability of parasites to manipulate host behaviour facilitates a substantial amount of energy flow from lower to upper trophic levels (Haderler and Freedman, 1989; Kuris et al., 2008). Host manipulation is a common strategy by which parasites alter their host's phenotype to increase their own fitness, usually by inducing or exaggerating host traits that favour parasite transmission or dispersal (Dobson, 1988; Poulin, 2010).

Adaptations for host manipulation have been documented in hundreds of parasite species across the tree of life including platyhelminths, acanthocephalans, nematodes, nematomorphs, arthropods, protozoa, fungi, bacteria, and viruses (Hughes et al., 2012), and have evolved at least 20 separate times (Paulin, 2010).

Some manipulations increase the likelihood of parasite transmission from prey to predator (trophic transmission) by inducing changes in the prey host's phenotype that make it more susceptible to predation. Other parasites induce behaviours that facilitate transmission among conspecifics, for example, in infected vertebrates, rabies can increase aggression, promoting transmission of the virus via bite wounds (Klein, 2003). Parasites may also cause their hosts to move from habitats preferred by the host to habitat suitable for the parasite

as, for example, in nematomorph parasites that induce a "water drive" in their cricket hosts, causing the crickets to drown themselves in streams, where the nematomorph emerges to complete its aquatic life stage (Hanelt et al., 2005)

Our understanding of the ecological effects of manipulation is still limited (Weinersmith and Faulkes, 2014), possibly because manipulations are diverse and can have varying, context-dependent ecological effects. The net influence of parasite loss on consumer populations will depend on the balance between loss of regulation on prey populations versus loss of manipulated prey individuals, but because many taxa in many ecosystems engage in host manipulation for trophic transmission, it is thus predicted that a world without parasite could be a world with fewer predators.

### **9.1.5 Ecosystems**

#### **9.1.5 (a) Parasites alter the cycling of energy and nutrients**

The ways in which parasites affect the cycling of energy and nutrients are only beginning to receive research attention (Preston et al., 2013), but because parasites can represent a large proportion of total biomass in some ecosystems (Kuris et al., 2008; Preston et al., 2013) and can directly alter rates of host nutrient excretion (Bernot, 2013), their influence on such cycles could be substantial. Behaviour-manipulating parasites, in particular, may have stronger effects on these cycles; it has been discussed above the influence of manipulation on the abundance of predatory species (which can be thought of as the "nodes", architecture, or topology of a food web), but parasites can also affect the movement of energy and nutrients through food webs (Kuris et al., 2008). For instance, by inducing behaviours in intermediate hosts that increase their susceptibility to predation, parasites may intensify trophic interactions and strengthen predator-prey linkages (Lefevre et al., 2009). Parasites may also alter the rates of other important ecosystem processes, such as grazing (e.g. rinderpest: Sinclair et al., 2008), decomposition (e.g. nematomorphs: Sato et al., 2011) and

bioturbation (e.g. trematodes; Mouritsen and Haun, 2008), as well as carbon sequestration and cycling of other nutrients (e.g. marine viruses; Danovaro et al., 2011). Whether energy flow to upper trophic levels is strengthened or weakened by parasite removal will depend on the relative influence of manipulative versus host-population regulating parasites.

**(b) Parasites alter across-ecosystem subsidies**

In many cases, parasites' manipulation of their hosts to move from habitat preferred by the host to habitat suitable for the parasite can result in a transfer of energy and nutrients from one ecosystem to another. To demonstrate this effect, Sato et al. (2011) showed that parasite-driven energy subsidies from terrestrial ecosystems in Japan (where crickets were experimentally added to stream reaches at rates equivalent to the rate at which nematomorph-infected crickets enter stream habitats) are sufficient to set off a trophic cascade. In this cascade, fish predators switch to feeding on crickets, releasing their usual prey, benthic invertebrates, from predation pressure, and thereby decreasing biomass of benthic algae and increasing the leaf breakdown rate.

Thus, in the absence of parasites, we may observe weakening of across ecosystem subsidies (e.g. nematomorph-infected crickets will no longer cross the boundary between terrestrial and aquatic ecosystems), but the extent of the contribution of manipulation or other parasite-mediated processes to across ecosystem subsidies remains largely unknown.

**10.0 SUMMARY AND CONCLUSION**

A parasite is any organism that makes its living off another organism to the detriment of the host and parasites have been rather successful so much so that up to halve of earth's 7.7 million known species are parasites (Panko, 2017). But researchers warned that climate change could drive up to one-third of earth's parasite species to

extinction by the year 2070. That kind of mass die-offs could spell ecological disaster.

According to Collins Carlson, a graduate student studying global change biology at the University of California at Berkeley, “one thing we’ve learned about parasites in the past decade is that they are a huge and important part of ecosystems that we have really neglected for years”.

A team of researchers led by Anna Philips of the Smithsonian National Museum of Natural history digitized tens of thousands of species and their locations in an online data base, creating what have been regarded as the biggest parasite record of its kind. Computer models were then used to predict what will happen to more than 450 parasite species when climate change altered their habitats, based on how their ranges have changed over the past two centuries. They concluded that even under the most optimistic scenarios, roughly 10% of parasite species will go into extinction by 2070 and in the direst version of events, fully one-third of all parasites could vanish. This kind of die-offs would have myriads of unfortunate consequences. Considering the facts that, parasites play an important role in regulating the populations of their hosts and the balance of the overall ecosystem. First, they kill off some organisms and make others vulnerable to predators, for example, when infected with the nematode *Trichostrongylus tenuis*, the red grouse bird emits the scent that helps predators find and eats it more easily, thus serving to control the birds population.

Parasites can also have more indirect effects. Periwinkle snails infected with the trematode species *Cryptocotyle lingua* for instance, eats significantly less algae along their Atlantic coast homes, because their parasite weakens their digestive tracts. Their small appetite make more algae available for other species to consume and there are millions of undiscovered parasite species whose ecological niches we can only guess at.

Parasites and their hosts have often evolved together over many years maintaining a delicate balance. After all, parasites usually have little interest in killing their hosts, since that will mean losing their homes and sources of nutrients. That is why tapeworms are rarely fatal to the people they infect, the worms have evolved to travel to their guts and feed on the food their host ingest and they rarely siphon off enough calories to actually kill their host. But when parasites go extinct, it creates new open niches in an ecosystem for other invasive species to exploit. That can create opportunities for new encounters between parasites and hosts that are not familiar with each other, and have not developed non-lethal relationships. In 2024 for instance, a tape worm species that was hitherto foreign to humans was found in a man's brain leading to seizures and inflammation of the brain.

A world without parasites is impossible to achieve, and can only be approximated in specific circumstances (such as zoo enclosures, aquaria, and intensive agricultural practice). In such enclosures, which, despite strenuous effort, are often still hotbeds of infection (e.g. hospitals), even if parasites did somehow all disappear, other species would evolve to occupy the newly vacated niches (Lloyd-Smith, 2013).

Its improbability notwithstanding, imagining such a world can help expose the otherwise hidden ecological roles of parasites. These roles are hidden because the ecosystem of a parasite (i.e. inside the host) is often nested within the ecosystems that ecologists are accustomed to considering, for example, forests, grasslands, and coral reefs. A better understanding of how parasites contribute to the communities and ecosystems in which they are embedded is a critical need as we consider how to make the world "less wormy" (Loker, 2013).

There are some cases in which elimination of a parasite species is both possible and highly desirable. In these instances, potential benefits to human health and wellbeing trump any other considerations. However, many of the contemporary disease challenges faced by society and imperiled wildlife, involve more complex chains of

transmission, frequently including multiple host species, multiple parasite species, reservoirs, or resilient environmental resting stages. As a result, total eradication will often be impossible, and "ecological surprises" associated with control efforts will probably appear with greater frequency. For example, without an appreciation for the antagonistic relationship between worms and protozoa living in the human intestine (Martin et al., 2013), a well-intentioned de-worming campaign could make people very sick.

I do not argue that human parasites should be conserved, but rather I emphasize the utmost importance of understanding the ecology of a parasite before attempting to control or eradicate it. As Jones (2015) wrote, "**Surprisingly, a world without parasites might not be a nicer one**". Thoughtful planning will prevent the loss of ecologically important parasites and the processes they facilitate, as we progress slowly towards a parasite-free world.

## **11.0 ACKNOWLEDGEMENTS**

I give all thanks, majesty and adoration to God Almighty and to Jesus Christ, my Lord and Saviour, for being my perfect guide right from infancy and later in life when I was drawing the picture of my career, despite being born to farming parents who were neither lettered nor well-travelled. He has always been my source of direction and inspiration in all my life's endeavours.

I am profoundly grateful to God for the life of my late father, Prince Dauda Ojo Olusi, whose vision and foresight laid the foundation for my educational journey. Though he did not acquire Western education himself, he deeply appreciated its immense value. At a time when the criterion for admission into school required that a child's right-hand fingers touch the left ear (a test I could not pass due to my tender age), he nevertheless ensured that I was enrolled in Infant One. As a Muslim and a polygamist blessed with many children, he remained resolute in his conviction that none of his children should be denied the opportunity of education. Sadly, he bade us all farewell in 1996 while I

was away in Germany. How I wish he is here today to witness the fulfilling fruits of his sacrifices, vision, and foresight that date back to the early sixties. Nevertheless, I take solace in the comforting belief that he rests peacefully in the bosom of his Creator, while his cherished memory continues to live on in our hearts.

I will forever remain very grateful to my dear mother, Mrs. Musiatu Aina Olusi, for her unquantifiable love and care. A disciplinarian par excellence, her disciplinary actions over very minor childhood misbehaviours combined with relentless counselling and fervent prayers have been responsible for shaping my journey through life thus far. Mama, I celebrate you today, ma. Currently in her nineties and the only surviving wife of my late father, she has lived a life of total submission to God Almighty and to her husband, in addition to praying for and raising God-fearing children.

I am greatly indebted to my elder brother, Elder Samuel Olanrewaju Olusi, currently the head (Olori Ebi) of the Royal family of Olusi (The Olusi Dynasty of Ikakumo) for making my education possible by taking over my responsibility from our father since my early teen years. I lived with him from when I was 11 years old and he saw me through primary and secondary schools. He was also my sole source of funding during the course of my undergraduate studies at the University of Maiduguri. Your invaluable financial support and encouragement have been of immense help to me, thank you very much sir.

I wish to express my profound and heartfelt gratitude to one of my two cherished and extraordinary mentors, an administrator par excellence, an academic colossus, and a distinguished prince of the Ife Royal Family, Emeritus Professor Adebisi M. Balogun. I remain deeply indebted to him for the genuine love he has consistently shown to me and my family, as well as for the special interest he has taken in my personal and professional development. It was through his goodwill and gracious intervention that the transfer of my service to the Federal University of Technology, Akure, was successfully facilitated. Since

then, he has remained a steadfast pillar of support to me and my family. Sir, I will forever cherish your rare kindness, love, and fatherly affection. I pray that Almighty God, in His infinite mercies, richly reward you with long life, sound health, and even greater prosperity in Jesus' mighty name.

I remain eternally grateful to my other extraordinary mentor and Ph.D. supervisor, Professor Jerry A. Ajayi, of blessed memory. He was the very embodiment of humility, an exceptional scholar whose character bore no trace of self-aggrandizement. A towering academic figure, he devoted his life to scholarship and the nurturing of future generations of scientists. At the time of his passing, he had successfully supervised seventeen Ph.D. graduates, including my humble self. Remarkably, five of these scholars later attained the distinguished rank of Professors of Parasitology, while one subsequently rose to the esteemed office of Vice-Chancellor of a University in Delta State. His remarkable legacy of mentorship, intellectual excellence, and quiet dignity will forever remain a guiding light to all who were privileged to be his students.

I acknowledge the everlasting impact of the following mentors from different Universities that I have been privileged to work with or study under, Professor Ikenna Onyido, Prof. Mark Nwagwu, Dr. Christen Betterton-Jones, Dr. Ngozi Okafor, Prof. (Mrs.) Bunmi Otubanjo, Prof. Titi Hassan, Late Prof. E. O. Ogunwolu, Prof. (Mrs.) O. Ajayi, Dr. M.A. Makinde and Prof. D.D. Duhlinska.

I also acknowledge with deep gratitude the love, kindness, and encouragement shown to me by my late father- and mother-in-law, Chief Gabriel Sunday Obagaye and Honourable (Chief) Mrs. Victoria Ebus Obagaye, both of whom I held in the highest esteem during their lifetime. They were a constant source of inspiration and encouragement to me. Daddy and Mummy, I remain deeply appreciative of your goodwill and kindness. May your gentle souls continue to rest peacefully in the bosom of your Creator.

I equally wish to place on record my profound appreciation for the strong family ties and sincere affection I have enjoyed with my in-laws. In particular, I acknowledge with gratitude Their Excellencies, Alhaji (Dr.) Sikiru Tae Lawal and Princess Yemisi Modupe Lawal, the former Deputy Governor of Ekiti State, and his beloved wife, whose goodwill and support I sincerely cherish. I also appreciate the warmth and cordial relationship I share with my other brothers- and sisters-in-law, namely: Mrs. Omolara Fatuase (late), Mr. and Mrs. Rotimi and Obioma Obagaye, Mr. Toyosi Obagaye, and Mr. Dele Akinyemi.

I wish to express my heartfelt and enduring appreciation to my late immediate younger brother, Mr. Adeyinka Olusi. It has been over twenty years since your departure, yet your memory remains vividly alive in my heart. May your soul rest in perfect peace (Amen). I equally extend my sincere gratitude to my other siblings and their spouses, Pastor and Mrs. Hammad Ayeoribe, and the late Mr. Adeyemi Adebusoye and Mrs. Moriamo 'Wunmi Adebusoye, whose love, support, and companionship have been a constant source of encouragement throughout my life.

I also wish to acknowledge with deep appreciation my step-brothers and step-sisters: the late Edwin Sanusi Olusi, Mrs. Margaret Aina Balogun, Mallam Alade Olusi (Alasco), Mr. Ibrahim Adesunloye Olusi, Mrs. Adedunke Oloba, Mr. Adetokunbo Olusi, Dr. Felix Ilori Olusi, Mrs. Stella Rekiyatu Olusi, Mrs. Risikatu Olope, Mallam Abubakar Jimoh Olusi, Mrs. Alice Alimotu Obalowose, Mr. Jimoh Olusi, Mr. Enimola Olusi, Mallam Abdulrazaq Olusi, as well as my cousins Mrs. Rhoda Obaude and Barr. Adegbenro Obanewa. Your guidance, support, and warm fellowship have enriched my life immeasurably.

I remain profoundly grateful to my esteemed uncles, Mallam Jomoh Adeleye, Dr. Joseph Olugbade, Mr. Segun Olugbade, the late Chief Adebayo Olugbade, Engr. Felix Adekanye, and Prince Henry Fajimi Olusi whose wisdom, mentorship, and unwavering encouragement have played a pivotal role in shaping both my personal

and professional journey. I also appreciate my nephew, Mr. Oyeniya Oloba, and my niece, Dr. (Mrs.) Abosede Akinyemi, for their steadfast support and inspiration.

My appreciation goes to my current Head of Department, Professor J.O. Akinneye, and to all my esteemed colleagues in the Department of Biology—affectionately known as “The Biology Family”, for the warm and collegial relationships we have enjoyed since I joined the department nearly two decades ago. I am particularly grateful to Professors C.O. Adedire, A.T. Aborisade, M.O. Ashamo, M.O. Oniya, B.O. Odiyi, O.E. Oladipo, B.W. Adu, J.A. Adeyemi, I.A. Simon-Oke, and O.J. Afolabi, as well as Drs. K.D. Ileke, O.A. Alabi, K.L. Akinwande, B.M. Macaulay, O.O. Oladele, O.M. Akomolafe, E.O. Onagbola, A.A. Sorungbe, G.A. Ogunwole, O.B. Awosolu, E.A. Oyeniya, S.E. Abiya, and Messer O.S. Babatunde, along with Mrs. A.I. Ogundayomi and Mrs. O.H. Owokoniran, for their camaraderie, support, and collegial spirit over the years.

I want to specially express my gratitude to Professor Chris O. Adedire, the former Dean of the defunct School of Sciences (SOS), for the warm reception, mentorship, and unreserved cooperation extended to me during my tenure as Head of the Biology Department (2009–2012). My sincere thanks also go to other colleagues who offered their unflinching support and collaboration during this period, sharing in the successes and challenges of departmental leadership. I equally extend my heartfelt appreciation to the dedicated technical staff of the Department, ably led by Mrs. E.T. Ojo (née Alade), and including Mrs. Afolami (the Departmental Secretary during my tenure as HOD), Mrs. B.B. Ola-Salawu, Mrs. E. Gbolahan-Ayoade, Mrs. S. Paul-Ogo, Ms. O. Adejuyigbe-Imolehin, Mr. S.B. Jimoh, Dr. C.T. Adebo, Mrs. T.M. Ayodeji-Ojo, Mrs. F.J. Adefisayo, and Mrs. A.K. Akindolani. Your tireless service, professionalism, and unwavering commitment have been central to the smooth functioning and success of the department, and I remain profoundly grateful to each of you.

I am greatly indebted to Dr. Uwe Groß of the Institute of Microbiology and Hygiene, Wurzburg, Germany for granting my visit to his Toxoplasmosis Laboratory, for formal training on the immunology and serological diagnosis of toxoplasmosis and for his loving care during my nearly one year stay in Germany. I also thank Prof. Jurgen Hessemman, Head, Institute of Hygiene and Microbiology, Wurzburg and my other colleagues that assisted in the conduction of the serological tests, Dr. Weissbrich, Ms. Irmi and Ms. Vera.

I remain eternally grateful to my spiritual father, Reverend E.A. Adebayo of First Baptist Church, who introduced me to Christ and the teachings of Christianity early in life, having been born of Muslim parents. The biblical principles and the model of a Bible-believing, true Christian life that he instilled in me, coupled with my early participation in the church choir, have been instrumental in shaping the disciplined lifestyle and moral foundation I have embraced throughout my adult life.

I am also profoundly grateful to the Spirit-filled, indefatigable, and anointed man of God, the Founder and General Overseer of Jesus Our Spring of Life Mission International, Reverend (Dr.) Oluyinka Fadiran, and his wife, Pastor (Mrs.) Sola Fadiran. I must place on record that “My Pastor,” as I fondly called him, was the divine instrument who connected me with my extraordinary mentor, Emeritus Professor A.M. Balogun, a connection that has had a lasting impact on my academic and personal journey. Sir, I pray for ever-increasing anointing in Jesus’ mighty name. My sincere appreciation also extends to all members of Spring of Life Bible Church, Ijapo and Oshinle Parishes, particularly Pastor (Mrs.) Alaba Akomolafe, Mr. and Mrs. Babalola, and Mr. and Mrs. Olatuja, for their constant support, prayers, and fellowship.

I am equally thankful to my Lord Bishop of the Akoko-Edo Diocese, The Rt. Rev. E.O. Saiki, the Vicar of my home church, St. Peter’s Anglican Church, Ikakumo, Rev. Elijah A. Alabi, and the Archdeacon of my home Archdeaconry, Ven. Imodu Afemikhe, for

their spiritual guidance and mentorship. I also appreciate the past vicars of St. Peter's Anglican Church, including Canon Emmanuel Ajulo, Rev. Kingsley Oluwashanu, and Rev. Joseph Odewale, whose faithful service and pastoral care have been a blessing to my family and me. My gratitude extends further to the following men of God: Rev. Titus Ademolu and Rev. Joshua Adeleke of St. John's Anglican Church, Oba-Ile, and Pastor Adaolowo of Christ Apostolic Church, Otukpo, Benue State. Your prayers, counsel, and unwavering support have been instrumental in shaping my spiritual life and grounding me in faith.

I extend my sincere and heartfelt appreciation to the Executive Committee (EXCO) and all members of the University of Ibadan Alumni Association, Ondo State Branch, under the current leadership of Mr. Akinola, with whom I am privileged to serve as First Vice-Chairman. I am deeply grateful for the cordial, supportive, and enduring relationships I have enjoyed with the Association since joining a few years ago.

I am equally thankful to all Honorary Members of the Officers' Mess of 32 Artillery Brigade, Nigerian Army, Owena Cantonment, under the able chairmanship of Dr. Patrick Oludare, with whom I also serve as Treasurer. Honourable members, your companionship, encouragement, and fellowship have been a source of great joy.

I wish to acknowledge with special gratitude the members of the group of ten elders, "The Like Minds," including Elder Adedipe, Hon. 'Demola Ijabiyi, Dr. Aroge, Dr. Olawale, Pastor Ojomo, Engr. Ekpemogun, Prince Olusa, Pastor Adekoje, and Engr. Orhukpe, all residents of the NTA Zone of the Oba-Ile Housing Estate, Akure. Your camaraderie has been truly wonderful from our early morning Saturday workouts, to evening gatherings at members' residences, the shared entertainment, and the light-hearted banter and senior-jokes we exchange. These moments have made your companionship both memorable and extraordinary.

To the members of the Housing Estate Residents Association, NTA Zone, where I currently serve as Chairman, I offer my profound

gratitude for your cooperation, trust, and collective effort throughout my first tenure of three years. I am particularly thankful to the dedicated service of Mr. 'Bola Adesuyi, our Financial Secretary, and Mr. 'Tunde Omotoso, our PRO, whose commitment and diligence greatly facilitated my work as Chairman.

I remain deeply appreciative of my faraway but close friends and associates in the University system, including Prof. Oladele B. Akogun, Prof. Okwoli Amali, Prof. Fabian Kaankuka, Prof. Regina Ega, Dr. Adesola Satimehin, Engr. Luis Alimonu Oparaku, and Mr. Sugh Loho, for their enduring friendship and professional support.

I must also acknowledge friends from my formative years: Mr. Bankole Samson (of blessed memory), Rev. Raphael Ojo Balogun, Mr. Martins Itakpe, Mr. Ambrose Ogboru, Mr. Bayo Kuku, and Mr. Samuel Adebayo, whose companionship and encouragement have remained meaningful throughout my life.

I am deeply honoured to have begun my teaching and professional career under highly respected Principal, late Chief Asaniyan, and Vice Principal, Mr. Olotu, I thank you both for giving me a sound beginning and the confidence you reposed in me at the start of my career. I also extend my gratitude to Mr. Esan, then Principal of Eyemojo Comprehensive High School, under whom I served as Vice-Principal, for the trust and confidence extended to me despite my youthful age at the time.

I am profoundly appreciative of the brilliant, obedient, and dedicated scholars I have had the privilege of mentoring. To my Ph.D. students, Dr. N.O. Awodi, Dr. M.R. Ibukunoluwa, Dr. O.G. Ajakaye, Dr. S.A. Salawu, Dr. M.O. Adediran, Dr. A.V. Akeju, Dr. O. Olaniran, Dr. R.N. Aniaguya, Dr. O.A. Olalekan, and Miss O. Fehintola (my current Ph.D. student), as well as my MSc students, D. Ochokwunu, R.H. Houmsou, K.H. Mohammed, T. Akande, E.G. Gundu, T. Ichor, C.O. Agbulu, A. Toluhi, and M.Tech students, Y.F. Jaiyeoba, A.F. Abe, O.E. Adetore, A. Ogunyemi, F. Adenrele, O.J. Ajayi, S.F. Adefisoye, S.A. Adegbehingbe, O.F. Kumuyi, O.S. Babatunde, A.A. Olonisakin,

M. David, O.V. Iyaniyi, E.E. Gbolahan-Ayoade, C.D. Udeh, Y.A. Agunbiade, and O.I. Ojo, I am proud of your achievements and the strides you are making in academia and beyond.

I am also sincerely thankful to the Vice-Chancellors under whom I have served during my nearly four-decade career in the University system: Prof. Shekarau Aku, Prof. F.S. Idachaba, Prof. J.I. Ayatse (current Tor Tiv), Prof. Akere, Prof. I. Ajibefun, Prof. Adebisi M. Balogun, Prof. A.G. Daramola, Prof. A. Fuwape, Prof. Ige, and our current visionary, humble, God-sent and transformative FUTA Vice-Chancellor, Prof. Adenike Adediji.

A young, vibrant, intelligent, and exceptionally beautiful damsel in her late teens, the cynosure of all who could appreciate natural beauty, Mrs. Olufunke Iyabode Olusi (née Obagaye), was the lady I met in 1980 during my undergraduate years. Our courtship spanned nearly seven years, culminating in our union as lawfully wedded spouses in 1987, almost forty years ago. She has been a pillar of responsibility, commitment, reliability, virtue, and prayerfulness. Indeed, her devotion is such that I fondly refer to her as the Deaconess of our nuclear family, leading with devotion in almost all of our daily family prayer meetings with her long and heartfelt prayers. I count myself exceedingly blessed and eternally grateful to God for giving me my heart's desire and the biblical "wife of my youth" (Proverbs 5:18).

I extend my heartfelt appreciation to my beloved children: Adebukola, Adedolapo, Adedamola, Adedamilola, Olajide and Echezona, and precious grandchildren: Temiloluwa, Oluwatobiloba, Iremide and Oluwatofiyani. You are all wonderful and I am very happy and proud to have you as God's priceless gifts to me. Your love, support, and accomplishments continue to bring immense joy and pride to my heart.

As I bring this 196<sup>th</sup> Inaugural Lecture to a close, I return all glory, honour, and adoration to the Almighty God, the Owner of my soul and the Author of all my achievements. It is by His grace alone

that today has been made possible. He alone is worthy of all praises, now and forevermore.

Thank you all for your attention, and may God bless you abundantly.

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