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Poverty, Parasitosis and HIV/AIDS - Major Health Concerns in Tanzania

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1. Introduction

Poverty, parasitosis and HIV/AIDS are closely interlinked and co-circulate in many populations. HIV/AIDS, parasitic infections like malaria and other opportunistic infections, and in a few are by far the commonest causes of ill-health and death in the poorest countries of the world, that happen to be in the tropics and temperate countries in Africa, Asia, and South America. Parasitic infections remain an important cause of morbidity and mortality in developing countries especially among HIV-infected persons.

There are indications that HIV-1 (the most prevalent in Tanzania) interacts significantly with many other parasitic infections within individual hosts, but the population-level impacts of co-infection are not well-characterized. Among those parasitic opportunistic infections, C*ryptosporidum parvum, Isospora belli*, *Cyclospora cayetanensis* and *Microspordia* species frequent causes of diarrhea. Likewise, *Pneumocystis jiroveci* pneumonia and *Candida* species infections have been implicated in life threatening fungal infections among people living with HIV/AIDS.

In this chapter, poverty is defined as a state of having little or no money and few or no material possessions. Poverty can be caused by unemployment, low education, deprivation and homelessness. Lack of health facilities and low-cost healthy foods, along with public space for physical activities, may be among the factors that contribute to poor health and even higher risk of death due to curable diseases among patients who live in poverty.

HIV/AIDS deepens poverty and increases inequalities at every level, household, community, regional and sectoral. The HIV/AIDS epidemic undermines efforts at poverty reduction, income and asset distribution, productivity and economic growth resulting in reverse progress of development targets. Certainly, there is relationship between poverty and the development of epidemics of communicable diseases and at the same time epidemic diseases, like any illness, have the potential to increase poverty.

The impact of HIV/AIDS and poverty on children has different dimensions that include children being deprived of education to care for sick adults, thus compromising their basic right to education, placing the household at further long-term risk for poverty that may take decades to reverse. Illiteracy and/or lack of skills also appear to influence vulnerability to HIV infection. A correlation between educational qualifications and HIV infection exists, which indicates that people with formal educational qualifications acquire economic independence and threfore do not engage in risky behaviors as compared to those without it.

Financial constrains or budgetary deficits in government expenditure on health in Tanzania, like most African countries translate into an increase in a number of untreated disease conditions, including sexually transmitted diseases that are known to facilitate the rapid transmission of HIV. HIV/AIDS appears to interact strongly with poverty and has increased the depth of vulnerability of the affected families/households because of being in needs (caring ill relatives, death and decreased manpower to seek for daily bread). The relation between poverty and HIV/AIDS is bidirectional. However, there is conceptual confusion about the nature of the relationship, probably because of lack of rigorous scientific researches on the links between poverty, HIV/AIDS and parasitosis.

There is strong evidence that socio-economic and gender inequalities exacerbate the spread of HIV while AIDS-related diseases and death increases these inequalities, a potentially vicious cycle. Poverty per se may not be the most important factor conditioning the risk of being exposed to HIV, but undoubtedly, it is the poor in these countries, and especially poor women, who are suffering the most with the subsequent impacts of AIDS. Given that malnutrition is a function of poverty, there is thus a good reason to assume that poverty helped to hasten the spread of HIV in sub-Saharan Africa.

However, when examining HIV vulnerability at the level of households and communities, the evidence appears to be mixed. Since the richer and better educated are likely to have better access to reproductive health care, condom use is generally low in Africa and other resources-limited countries. It is also postulated that poverty is placing individuals from poor households at greater risk of exposure to HIV through the economically-driven adoption of risky behaviors. Moreover, poverty and food insecurity (malnutrition) seem to increase sexual risk taking, particularly among women who may engage in transactional sex to procure food for themselves and their children.

Consequently, combating malnutrition, including worm infestation, requires more than providing treatment. It requires multisector approaches to address the broader causes such as poor water and sanitation provision and lack of food security. The approaches should also focus on HIV/AIDS control involving but not be limited to mainstreaming of HIV implications into the policy and practice of many sectors.

Although HIV prevalence has fallen in Tanzania over the past decade, thousands of people become infected with HIV every year and 86,000 Tanzanians died from AIDS in 2009 alone. While the poor are undoubtedly hit harder by the downstream impacts of AIDS, in a variety of ways, their chances of being exposed to HIV in the first place are not necessarily greater than wealthier individuals or households. Poverty, in a nutshell includes illiteracy, parasitosis/diseases, food insecurity plus HIV/AIDS are the major problem currently facing Tanzanians. Therefore, a joint effort is urgently required to combat them in their totality, with that approach, the victory is evident.

2. Poverty, parasitosis and HIV/AIDS

2.1 HIV/AIDS

2.1.1 A brief history and Trends of HIV and AIDS in Tanzania

The first cases of AIDS were reported in the Kagera region (north western of Tanzania) in 1983 and by 1987 every region in the country had reported AIDS cases. In 1985, the government set up the National AIDS Control Programme (NACP) to coordinate the response and established AIDS coordinators in each district in the country, Tanzania Commission for AIDS (TACAIDS). In order to confront the growing epidemic, the NACP

developed a medium term plan for the period 1987-1991 which was then followed by two more medium term plans covering 1992-1996 and 1998-2002. These plans had three main aims: the decentralization of the health sector responses, reducing HIV transmission and relieving the social consequences of HIV/AIDS through care and assistance. However, according to Tanzania's first National Multisectoral Framework (2003-2007) the three medium term plans did not halt the spread of HIV. By the time the third medium term plan came into being HIV prevalence had reached 8 percent.

A national policy, which had been under development since 1991, was finalized in 2001, following the declaration of 'war' on HIV/AIDS by former His Excellency President Mkapa. TACAIDS was then established in 2002 to coordinate the multisectoral response, bringing together all stakeholders including government, business and civil societies to provide strategic guidance to HIV/AIDS programmes, projects and interventions.

A study published in 2005, using evidence drawn from the past 20 years exposed some findings which challenged some widely held assumptions about the effects of HIV and AIDS. The study found that generally the highest prevalence of HIV was found amongst the well off individuals/households, particularly affecting rich women, as opposed to poorer and rural households (Shelton et al., 2005). The findings pointed out that wealthier people tend to have the resources which lead to greater and more frequent mobility and expose them to wider sexual networks, encouraging multiple and concurrent relationships. But it was also observed that the wealthier people tend to have greater access to HIV medications that prolong their lives and are more likely to live in urban areas, which have the highest prevalence.

However, HIV prevalence gap between poor urban groups and poorer rural communities is slowly closing. A 2008 study found that knowledge of sexually transmitted infections (STI) was 'alarmingly low' in rural Tanzania and associated with low condom use and HIV infection. Reduced prevalence has mainly been noted among the most educated (those who attended secondary school) while among those with no formal education, prevalence has not decreased and the number of new infections has risen (Hargreaves et al., 2010). Because access to health care and knowledge of HIV and AIDS is typically lower in rural areas. This led to adoption of more aggressive measures towards educating the rural people on preventive efforts and thus further spread of the epidemic.

In a nut shell, some contextual factors shaping the HIV/AIDS epidemic in the country include:

- Poverty and transactional sex with increasing numbers of commercial sex workers
- Men's irresponsible sexual behaviour due to cultural patterns of virility
- Social, economic and political gender inequalities including violence against women
- Substance abuse such as alcohol consumption
- Local cultural practices e.g. widow cleansing
- Mobility in all its forms which leads to separation of spouses and increased establishment of temporary sexual relationships
- Lack of male circumcision

2.1.2 Immigration and rural-urban migration of people

HIV infection is unevenly distributed across geographic area, gender, age, groups and social economic classes in the country. The prevalence rates of HIV/AIDS range from less than 1%

to more than 15% in certain regions. Nevertheless, the epidemic has struck more the most economically active group of adults, those aged 15-45, which is also the productive age-group of individuals. Population movement is common in Tanzania, especially among the youths (18-45 years old). Growth and/or expansion of the mining sector and education have led to greater urbanization and mobility between rural and urban areas. This created an avenue for young and sexually active persons to interact socially and forming 'high risk sexual networks', which include sex workers, women at truck stops and miners, whom most of them are HIV seropositive.

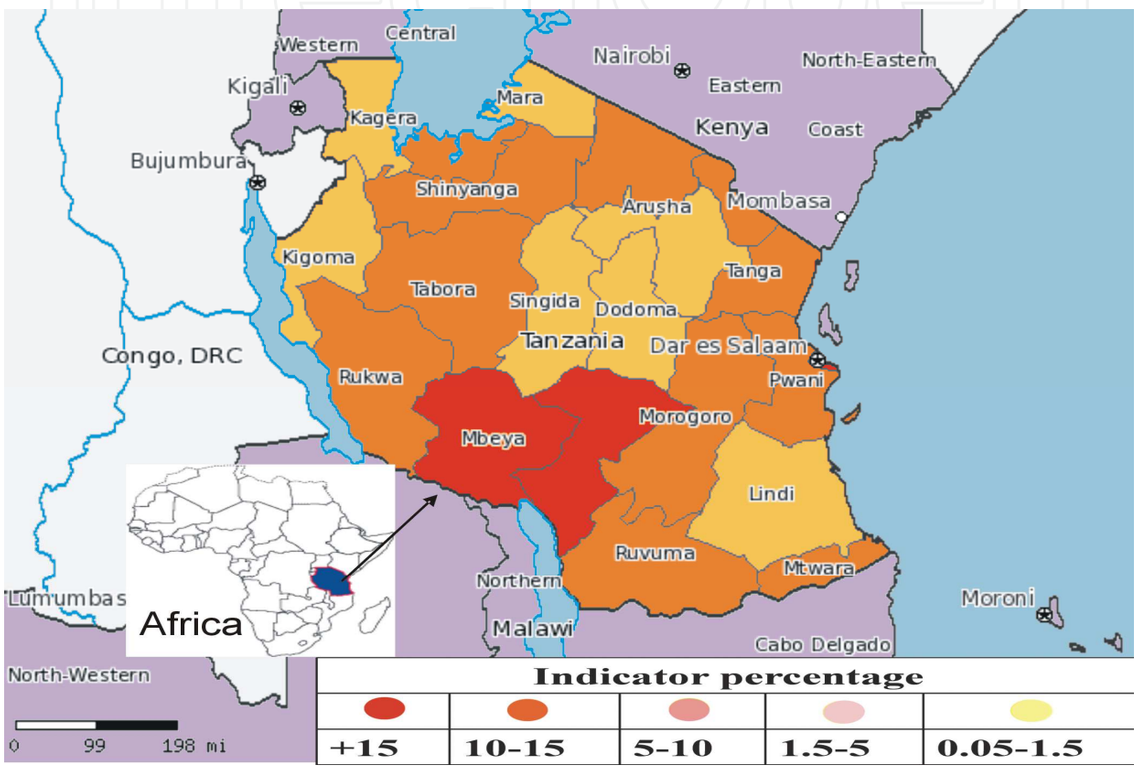

Fig. 1. Prevalence of HIV/AIDS among adults in Tanzania

Tanzania is also well known for its hospitality. Since the era of the Former President Late Mwalimu Nyerere, founder of the nation, our country has been hosting a myriad of refugees from all over the continent, who definitely had established close ties with citizens. This relationship could also have brought some ''exogenous and detrimental effects''.

2.1.3 Dependence on foreign aids on combating HIV infections and stigma

On the other hand, Tanzania's coastal trade as well as its border with eight countries exposes its vulnerable populations to HIV. In border and lake areas, 1 in 5 people are HIV positive. A study conducted by International Organization of Migration in 2010, revealed that transport workers, fishermen, border personnel, and seafarers were more likely to have multiple sexual partners and less likely to use condoms than the general population. Worse still, access to treatment and prevention initiatives in these areas was also minimal; underlining the regional variation in access to HIV services.

About only 8 years ago, the National Policy on HIV/AIDS recognized antiretroviral therapy as a right for all people living with HIV, at a time when no Tanzanians were receiving antiretroviral treatment. In 2003, the William J. Clinton Foundation and a group of Tanzanian experts created a Care and Treatment Plan (2003-2008), which was then adopted by the Tanzanian Government. The five-year plan proposed the roll out of antiretroviral therapy aiming at providing antiretroviral drugs free of charge to all people living with HIV by 2008. However, by 2004, only about 0.5% of those with advanced HIV were receiving treatment. Presumably, one of the major reasons is the Tanzanian HIV/AIDS response being heavily dependent on foreign funding sources. Because about 95% of the funding for HIV/AIDS programmes comes from foreign donors of whom more than two thirds is from the Global Fund and the U.S. President's Emergency Plan for AIDS Relief (PEPFAR). HIV/AIDS funding makes up one third of all aid coming to Tanzania.

Discrimination leads to an unwillingness to take an HIV test and to disclose results to family, friends or sexual partners. One study conducted in Dar es Salaam in 2005, found that only half of HIV positive respondents had disclosed their status to intimate partners. Time lapse from receiving results to disclosing them was 2.5 years for men and 4 years for women. Stigma, specifically fear of abandonment, job or property loss and violence were reasons for this delay. Contradictory perceptions exist among Tanzanians in respect of contracting STI, some believe that is a symbol of being sexually active and potent while other think it is ungodly and is a shame. However, when it comes to seeking for medical attentions, both groups find it uneasy. Such mixed notions ascribe to fear and hesitancy to disclose their status thus that increasing the chance of transmitting the STI, HIV inclusive to a partner, by avoidance of preventative measures and thus delay of timely of treatment (Mshana et al., 2006; Roura, 2009). In most regions/communities lack of knowledge about HIV/AIDS is a major cause of stigma and discrimination. Four out of ten women and a third of men surveyed in the 2007-2009 HIV and Malaria Indicator Survey reported that they would not buy fresh vegetables from a shopkeeper who has HIV, and half of all women and 40% of men said they would feel it necessary to keep it a secret if a family member was infected with HIV.

2.2 Poverty and HIV/AIDS

2.2.1 Economy overview

Tanzania is one of the world's poorest economies in terms of per capita income, however, Tanzania average 7% gross domestic product (GDP) growth per year between 2000 and 2010 on strong gold production and tourism. The economy depends heavily on agriculture, which accounts for more than one-fourth of GDP, provides 85% of exports, and employs about 60% of the work force. The World Bank, the IMF, and bilateral donors have provided funds to rehabilitate Tanzania's aging economic infrastructure, including rail and port infrastructure that are important trade links for inland countries.

“Hunger is the first obstacle to ending poverty. Hungry is poverty. A person who is always hungry is always poor. We can talk about the eradication of poverty all we want. We can never achieve it, if we don't first end hunger. The hungry live in rural areas and urban slums, in refugee camps and on farming homesteads. Wherever they are, hungry families live in the grey area between crisis and normality. Their poverty keeps them vulnerable to hunger. And hunger keeps them poor. Investments in infrastructure need to place more emphasis on ensuring that the assets truly are for the poor like community-based ponds, woodlots, roads and the like. Yet physical infrastructure alone cannot lead to less poverty or

better food security. A bridge may make the local market half an hour away rather than half a day. But when you have no education, poor health, and no energy, all the opportunities the market holds are beyond your reach," Statement by the Executive Director World Food Programme.

Therefore, defining poverty is a great challenge to researchers because of its complex nature. Poverty is the state of having little or no money and few or no material possessions. Poverty can be caused by unemployment, low education, deprivation and homelessness. Lack of local health clinics and low-cost healthy foods, along with public space for physical activities, may be among the factors that contribute to poor health and even higher risk of death due to curable diseases among patients who live in poverty. East Africans are some of the poorest people in the world ranking in this order: Kenya-Uganda-Tanzania. Therefore, Tanzania being one of the poorest countries, her citizens face several socio-economic problems, especially for unemployed, elders and orphans. The World Bank defines poverty as "the inability to attain a minimum standard of living" and produced a "universal poverty line", which was "consumption-based" and comprised of two elements: "the expenditure necessary to buy a minimum standard of nutrition and other basic necessities and a further amount that varies from country to country, reflecting the cost of participating in everyday life of society. The World Bank uses this definition largely for inter-country comparisons, and is not necessarily depicting what happens in households.

Poverty is also about more than income and economics. There are many types of poverty:

- Service poverty, where people are unable to access or are not provided with services such as health and education;
- Resource poverty, where though they have sufficient incomes people are unable to access resources because they may be poor in terms of their rights, representation or governance.

However, for reasons of space and scope of this theme, we focus on types of poverty outlined above centering on income and social capital, which seem to apply in Tanzania.

2.2.2 Decomposition changes in poverty

Most of the tools used in the analysis of poverty can be similarly used for the analysis of inequality. One could draw a profile of inequality, which would look at the extent of inequality among certain groups of households. This informs on the 'homogeneity' of the various groups, an important element to take into account when designing interventions. Gini-coefficient of inequality: This is the most commonly used measure of inequality. The coefficient varies between 0, which reflects complete equality and 1, which indicates complete inequality (one person has all the income or consumption, all others have none). Using 1993 as a baseline for Tanzania, **Table 1** below shows how per capita growth rates and changes in inequality would translate into changes in poverty over a 20-year period. With a zero real per capita growth rate and no change of inequality, the poverty rate would

Poverty rate with	1993	2005	2015
0 % growth , no growth in Gini	50	50	50
1.5% growth, no change in Gini	50	35	18
1.5% growth, Gini reduction by 0.5%/year	50	30	3
3.0% growth, no change in Gini	50	25	5

Table 1. Poverty, Inequity, and Growth in Tanzania.

remain unchanged. A 1.5 percent sustained per capita growth rate with no change in the distribution of income (all household get a 1.5 percent income gain per year) would yield a substantial reduction in poverty. If inequality were to improve at the same time, the poverty reduction would be greatly accelerated, even with a similar growth level.

Equally, the impact of economic growth can be analyzed by various ways viz. by comparing inequality between different groups, by decomposing inequality to assess the major contributors to inequality, by analyzing inequality, growth and poverty and their relationship and finally, by decomposing changes in inequality over time as shown in **Table 1**.

Therefore, it is occasionally feasible to elucidate how much of the observed changes in poverty over time can be attributed to changes in distribution and to changes in mean income or consumption. For example, lower poverty could result either from a general increase in the income of all households (without change in the income distribution) or from a decrease in inequality (redistribution from the rich to the poor without change in mean income or consumption). A change in poverty can always be decomposed into a growth component, a redistribution component, and a 'residual' component. An example can be taken from rural Tanzania, which experienced a decrease in poverty but an increase in inequality. Decomposing changes in poverty incidence (headcount) and depth (poverty gap) reveals that while the poor benefited from growth over the period, the rich captured a much greater share of economic improvement. In fact, if the distribution of income hadn't changed, the reduction in poverty incidence would have been much larger and the poverty gap would have also decreased. **Table 2** below presents the results of the analysis and show that, using a high poverty line, the head count would have decreased by 38% and the poverty gap by 24%. The changes in distribution (and interaction factors) resulted in a decrease in the head count of only 14% and in the poverty gap of only 2%.

Poverty line	Growth component	Redistribution component	Residual	Total change in poverty
		Head count index		
High	-38.5	11.8	12.6	-14.1
Low	-34.4	16.7	5.7	-12.0
		Poverty gap index		
High	-23.7	20.5	1.6	-1.6
Low	-19.0	22.9	-1.9	2.0

Table 2. Decomposition in changes in Poverty in Rural Tanzania (1983-1991). Source (Ferreira, 1996)

Tanzania suffers widespread and severe poverty, with nearly 60% of the population living on less than $2 per day and an estimated 20% living on less than $1 per day. As a result, Tanzania is ranked 164th out of 177 countries in the U.N. Human Development Index. As it also suffers a heavy disease burden, Tanzania exemplifies the intimate link between poverty and health. Lacking funds and infrastructure lead to difficulties in accessing primary health care for more than two-thirds of the population, with 39.9% lacking the money needed for treatment and 37.6% unable to travel the necessary distance for treatment.

Consequently, HIV/AIDS may deepen poverty and exacerbates inequalities at every level, household, community, regional and sectoral. The epidemic undermines efforts at poverty reduction, income and asset distribution, productivity and economic growth. HIV/AIDS

has reversed progress towards international development goals because of the influence it has on all development targets. There is an undoubted relationship between poverty and the development of epidemics of communicable diseases and at the same time epidemic diseases, like any illness, have the potential to increase poverty.

It has been suggested that illness and poverty affect household resources and income, as consequence of rising costs of medical care/treatment, and an increased need for nutritious foods. When adults/parents are stricken by HIV/AIDS or any other illness, with the progression of the illness, the demand for care also rises. Children are often withdrawn from schools to care for sick adults, compromising their basic right to education. The deprivation of education could place the household at further long-term risk for poverty, lack of skills and disempowerment. Consequently, this results in a cycle of household impoverishment that may take decades to reverse. Illiteracy and/or lack of skills also appear to influence vulnerability to HIV infection. One previous study in South Africa found that those with tertiary educational qualifications had lower rates of HIV infection than those with only school level qualifications. This implies that people with the necessary educational qualifications, thus acquiring economic independence for survival do not engage in risky behaviors than those with limited education.

Financial constrains or budgetary deficits in government expenditure on health in most African countries translate into an increase in a number of untreated STIs that are known to facilitate the rapid transmission of HIV. This could have serious long-term health implications resulting from the rapid spread of HIV. Apparently, HIV/AIDS appears to interact strongly with poverty and has increased the depth of vulnerability of those households already vulnerable to shocks. HIV/AIDS has acted to intensify the disadvantages imposed on the poor households and communities. HIV/AIDS leads to financial, resource and income impoverishment as households become poorer as a result of the illness and death of members, and in many cases it is the income-earning adults who are lost. Ultimately the high cost of care and burials leave heavy burden on the already overburdened households, orphans and dependants, people living with HIV/AIDS (PLHAs) and vulnerability to HIV infection (Ngalula et al., 2002). Therefore the 'poverty factor' at the household level has to be addressed simultaneously with the National efforts to combat the HIV/AIDS epidemic.

However, the impact is more than financial. Illness and death leads to an erosion of social capital and socially reproductive forces. In other words, the term social capital refers to the effort that goes into the reproduction of social and economic infrastructures. Therefore, at the social level, there are variety of relationships such as physical infrastructures, beliefs about trust, rituals of bargaining and price setting, mechanisms for regulating weights and measures, means of resolving disputes, and repeated activities which ensure that all these things continue to exist. These are not only matters of economic activity but also the maintenance and development of institutions, the reinforcement of community daily activities that also become negatively affected as consequence of the HIV/AIDS. Ultimately, the human capital loss has serious social and economic development in all sectors and at all levels. Hence, raising living standards of households and communities over the long-run through productivity-enhancing investments in agricultural technology generation and diffusion, improved crop marketing systems, basic education, infrastructure, and governance could improve their ability to withstand the social and economic stresses caused by HIV/AIDS.

It is now recognized that poverty significantly influences the spread and impact of HIV/AIDS. In many ways it creates vulnerability to HIV infection, causes rapid progression of the infection in the individual due to malnutrition and limits access to social and health care services. Poverty causes impoverishment as it leads to death of the economically active segments of the society and bread winners leading to reduction in income or production. Impacts of HIV/AIDS in most developing countries, Tanzania inclusive, have been visualized with poor resolution microscopes and thus studies of the effects of AIDS on households and most focus on economic impacts of death rather than illness. These show a distorted image. In Northern Tanzania (Kagera), a study conducted by the World Bank with Tanzanian co-investigators found that households stricken by deaths of their beloved ones especially adults, spent more on medical care and number of working time was significantly reduced. Similarly, it was revealed that even in well off families/households (for African standards) 29% of non-orphaned children were stunted while 50% of orphaned children were wasted. In poorer households 39% of non-orphaned children were stunted while 51% of orphaned children were wasted. These figures point to the effects on all children of growing up in a poor society.

Most African countries were performing poorly even before HIV/AIDS began to spread, largely due to their government's start-stop approach to economic reform. With the HIV/AIDS epidemic now at full force, these countries can only begin counteracting the effects of the epidemic if they undertake far more dramatic restructuring than they were prepared to previously. However, HIV/AIDS has worsened the situation as school-going age children born from HIV victimized families suffer both long-and short term consequences. Some of the effects are poor physical conditions, compromised immune systems and mental functioning is negatively affected by stunted growth, which is their long term effect. Hence, it may affect the ability of children to benefit from education and to function socially and economically later in their lives. Usually, orphans are unlikely to have proper schooling as the death of a parent or both parents in a family reduces a child's school attendance or just being unable to pay for schooling. This may automatically force the child to engage into ''adults' duties''. Sick adults may have reduced expectations of the returns to investing in children's education, as they do not expect to live long enough to recoup the investment. When a child goes to another household after its parents' deaths, the obstacles become greater as the child is not their own, particularly when even their own children are not guaranteed with all basic needs: food, clothing and school fees. These are some of the results of poverty (under-resourcing of public education) as consequence of the HIV/AIDS epidemic. AIDS increases teacher deaths and they may be difficult to replace, particularly in deprived, rural or otherwise remote communities. On the other hand, teachers' illness can lead to pupils being untaught for extended periods and replacement is difficult while staff members are on sick leave.

Therefore, the relationship between HIV/AIDS and poverty is synergistic and symmetrical. HIV/AIDS impacts households on two main levels, namely the social and economic levels. On a social level, households have to deal with issues around stigmatization, social exclusion and disintegration of family structure and social support networks. On the economic level, households and the surviving members have to pay for medical costs and funeral expenses and, if the deceased was a breadwinner, there will be further financial impacts in a form of a loss of income. Thus HIV/AIDS can directly contribute to poverty.

As much as HIV/AIDS exacerbates poverty through morbidity and mortality of productive adults, poverty facilitates the transmission of HIV. In developing countries, especially those in Sub-Saharan region, HIV/AIDS is reaching a stage at which AIDS morbidity and mortality

are increasing rapidly (Dorrington et al., 2001). Once adults are sick and some are bedridden, then the young and elderly are forced to care for them. The situation can exert unbearable pressure on households in their struggle for survival. Poor households are usually the worst hit and more vulnerable to the long-term effects of HIV/AIDS and poverty. In some regions of Tanzania, more than half of hospital beds are occupied by patients with HIV/AIDS related conditions. The treatment costs related to these admissions may lead to further impoverishment especially at household level. Poverty is associated with vulnerability to severe diseases like HIV, through its effects on delaying access to health care and inhibiting treatment adherence. The costs incurred while seeking diagnosis and treatment for HIV/AIDS are common causes of delays in accessing health care especially for the poor. To a certain extent, this forces some families to seek medical attentions from traditional healers, due to their easy accessibility and affordability. Poor households may not necessarily have the financial resources to seek help from health facilities, nor food security to enable members to adhere to their treatment. The lack of these resources is significant cause of the delays in accessing health services by poor households. Food security here is concerned with physical and economic access to food of sufficient quality and quantity. Food security is necessary, but by itself insufficient, for ensuring nutrition security. Nutrition security is achieved for a household when secure access to food is coupled with a sanitary environment, adequate health services, and adequate care to ensure a healthy life for all household members.

Poverty limits the options for treating infectious diseases like HIV. Infection with other STI is an important co-factor of HIV, and it provides a point of entry for HIV. Poor households become even more vulnerable when unable to raise the necessary funds to pay for treatment as they largely depend on the state to provide these services. Women in Tanzania also have severely limited access to education, employment, credit, and transportation. As a result, northern coastal women—married and unmarried, young and old—are increasingly turning to sex work, exposing them to a high risk of HIV infection. "We accept that it is now the female burden to provide for our children," says a woman from Mkwaja village. "We risk dying from AIDS for the sake of our children''. The true scope of HIV prevalence in the coastal region is not known because of low diagnostic and testing rates. (Fifty-eight percent of all Tanzanian adults living with HIV/AIDS are women). A barmaid with a 3-years old baby in Mbeya says '' I do sell myself in order to safeguard my child/daughter and guarantee that she has something to eat. If I don't do that right now, for sure she will die in my arms. Then what is the point seeing my kid dying or me dying in the next ten or so years?''. Mbeya is one the two badly HIV/AIDS stricken regions in the country with prevalence rate of 15-20%; another region is Iringa. Mbeya has boarders with Malawi and Zambia. This kind of despair and total lack of hope of improving wellbeing at household, drive this woman and several other into ''problem''.

Contradictory evidences exist with regard to HIV vulnerability at the level of households and communities (Wojciki 2005; Giraldo, 1997). Early studies had demonstrated positive correlations between household economic resources, education, and HIV infection, as the epidemic progressed; however currently, this relationship is somehow slightly changing. Generally, in Tanzania and most African countries, relatively rich and better educated men and women have higher rates of partner change because they have greater personal autonomy and spatial mobility. The richer and better educated are likely to have better access to reproductive health care, condom use is generally low in Africa and other parts of the developing world. At a later stage, however, it has been argued that individuals with higher socioeconomic status tend to adopt safer sexual practices, once the effects of AIDS-

related morbidity and mortality become more apparent. It is also postulated that poverty (possibly itself fuelled by AIDS) is placing individuals from poor households at greater risk of exposure to HIV via the economically-driven adoption of risky behaviors. Poverty and food insecurity are thought to increase sexual risk taking, particularly among women who may engage in transactional sex to procure food for themselves and their children.

For that reason, HIV/AIDS mortality can change the demographic structure of the household, reverse the roles of the members, exacerbate poverty, rob children of their parents thereby creating more orphans, infringe on the basic rights of the child in areas such as education, food, nutrition, health and others. Unless households are strengthened and empowered through focused interventions, poor households are likely to fall deeper into poverty for the generations to come. For PLHA and poor, it will be harder for them to sustainably access antiretroviral therapy, since some may not afford transport costs, regardless that they may be willing to go for the ART. But also some may be unable to afford to buy nutritious food, which is actually part and parcel of ART. At the household level, poverty will worsen the impacts of other livelihood stresses and shocks, and close down options for effectively dealing with hardships and challenges of life.

Therefore, food security is an important element for the survival of any household across the spectrum of wealth. Households affected by HIV and poor may find it difficult to maintain their food security. Both HIV and poverty exert tremendous pressure on the household's ability to provide for the basic needs like food. Let alone that poor nutrition is often linked with adverse outcomes in HIV/AIDS. Poor nutritional status is linked to vulnerability to progression from HIV infection to mortality. Poor nutrition weakens the body's defense against infection, and infection in turn weakens the efficiency of absorption of nutrients. Micronutrient deficiencies undermine the body's natural defenses against infections, thus contributing further to the vulnerability to HIV infection. Households experiencing food shortages as a result of poverty and effects of HIV/AIDS increase the chances of fast progression of HIV infection to AIDS and inevitable death of the ill person. Given that malnutrition is a function of poverty, therefore there is a substantial reason to assume that poverty helped hasten the spread of HIV in sub-Saharan Africa.

2.3 Poverty, malnutrition and HIV/AIDS

The term malnutrition was previously regarded as synonymous with undernutrition. This was before the emergence of the nutrition transition. Nowadays, a broader and more comprehensive definition of malnutrition is necessary since in actual sense, the term refers to the entire spectrum of deviant nutritional status, from short stature and below normal weight. There is considerable information describing the nutrition situation in different parts of Tanzania, based on spot surveys, child growth monitoring systems or research work. Most of this information is not nationally representative and is focused more on children under-fives than other population groups. Only scanty serial national information is available making it possible to discern trends over time in only a few indicators and in specific areas of the country. Tanzania's main problems of nutrition are similar to those of other countries in Sub-Saharan Africa. They are related to undernourishment, and these are protein-energy-deficiency (PED), iron deficiency anaemia (IDA), iodine deficiency disorders (IDD) and vitamin A deficiency (VAD).

Apart from these deficiency disorders, there are two nutrient excess disorders represented by fluorosis in the northern and north-western and central parts of mainland; and the

problem of overweight, obesity and diet-related non-communicable diseases which seem to be increasing especially in the urban elite and business sections of the community emulating unhealthy food habits and lifestyles. Compared to Sub-Saharan Africa; Tanzania is just slightly better than the average of 30% for underweight. For developing countries, excluding China, Tanzania is worse off for stunting but better off in wasting and underweight. Since the young population depends on a small adult population to sustain the economy and to provide the resources needed for adequate food, care and health; malnutrition in adults is of serious consequence. In such a situation, malnutrition becomes both a result and a cause of poverty. Malnourished adults cannot respond well to the challenges of economic and even political reforms which need both physical and mental energy. Maternal malnutrition is of even more severe consequence, as malnourished women produce malnourished children who will grow into malnourished adults creating a vicious cycle.

The nutritional status of adults is anthropometrically measured by using the Body Mass Index (BMI) which is a number derived from dividing the subject's weight in kilograms by the square of the height in meters (W/H^2). The distribution of Body Mass Index (BMI) in a few regions surprisingly show that more than a third of adults have low BMIs with rural areas having higher prevalence rates of undesirable BMIs than urban areas. Nonetheless, economic and historical analysis, epidemiological evidence, and common sense indicate that widespread childhood malnutrition will undermine investments in health, education, and ultimately economic development. Investments in cost effective treatments combined with broader nutrition based interventions must get higher priority.

Demographics, food supply and epidemiological transitions determine the specific relevance of the malnutrition-infection interaction in individual circumstances. Until the advent of the AIDS pandemic, average life-expectancies were rising in Tanzania and everywhere in the world; this was largely as a consequence of a reduction in early deaths from infectious childhood diseases. Today, HIV/AIDS is a world-wide calamity, though its impact is heavily felt in Africa. All of the poverty-related poor health and poor health care factors that go into increased susceptibility to HIV also affect the speed with which it progresses to full-blown AIDS and to death by opportunistic infections (Barnett et al. 2001). In particular, poverty-related lack of access to medical treatment, either to reduce viral load or to prevent and treat opportunistic infections, results in a lower quality of life, more rapid development of AIDS, and more rapid demise for poor PLHA. For example, people infected with HIV, who also have latent tuberculosis, are 30-50 times more likely to develop active TB. Similarly, ten percent of HIV infected persons develop cryptococcal meningitis, a fungal infection which leads inexorably to an extremely painful death within 30 days unless treated with powerful fungicides.

Proper nutrition is required to ensure optimal health. Consumption of a wide variety of foods, with adequate vitamins and minerals intake, is the basis of a healthy diet. Nutritionists outline that no single nutrient is the key to good health, but that optimum nutrition is derived from eating a diverse diet, including a variety of fruits and vegetables. Because foods such as fruits and vegetables provide many more nutrients than vitamin supplements, food is the best source for acquiring vitamins and minerals.

2.3.1 Nutrition, immune system and infection/parasitosis

Nutrition (also called nourishment or aliment) is the provision, to living cells, tissues and organisms, of the materials necessary (in the form of food) to support life. Many common health problems can be prevented or alleviated with a healthy diet. One of the most innovative

researches in the past 40 years is the increased knowledge on how the immune system operates; from a point of view of cellular biology and humoral responses, which formed the so-called immunobiology that encompasses other field of studies like the nutritional community (Maclean and Lucas, 2008). Recognizing the magnitude of nutritional problem in the country, the government established the Tanzania food and nutrition centre (TFNC) that deal with nutrition-related issues. Some of the main responsibilities of TFNC are:

- Reduction of all forms of malnutrition to acceptable levels
- Effective coordination of nutrition activities in the country
- Promote nutrition of the socio-economical deprived and nutritionally vulnerable groups

Tanzania, like other developing countries, her citizens faces a huge problem of malnutrition (undernutrition and/or over-nutrition), and this could greatly contribute to progression of HIV to AIDS. Until recently, obesity (overweight) was regarded as symbol of wealth and being healthy by majority of Tanzanians. Obese individuals demanded respect among the societies. On the other hand, skinny or slim persons were regarded as underfed/nourished, synonymous with being poor (unable to feed oneself adequately. With advent of HIV/AIDS, skinny 'slim'' individuals were thought to be infected by HIV, from which the word ''slim'' was derived. However, when the government launched ART, and both slim and overweight individuals enrolled in the therapy, this perception has drastically changed. Though, the extent of knowledge between the urban and rural living people could still significantly differ. This to some extent is a product of illiteracy among the population. To date, overweight/over nutrition is not a major health concern to majority of Tanzanians.

Notwithstanding, over nutrition may result into accumulation/deposit of unused/excess of nutrients: excess of carbohydrates into fatty acids and proteins being converted to fatty acids and then stored as lipids as well. This kind of nutrients accumulation has detrimental effects to the body. The quantity and nature of lipids are important factors in the process of immune system modulation. Several mechanisms have been involved in the modulation of the immune system by fatty acids:

- Membrane fluidity,
- Production of lipid peroxides,
- Eicosanoid synthesis and
- Effect on gene regulation.

Speculations exist that fatty acid immuno-modulation occurs not only singly, but also as a collective action of these factors.

A change in phospholipids' fatty acid compositions due to dietary lipid manipulation is ascribable to alterations in the membrane fluidity. Fatty acids from dietary lipids can be incorporated into any of the different phospholipids within the plasma membrane and they are clearly altered by the availability of dietary lipids. The changes in fatty acid composition of this structure have great importance because of the alteration of plasma membrane characteristics. This fact may be attributed to changes produced in the activity of proteins associated with the membrane, which act as receptors, form ion channels or are related to enzymatic functions (Liu et al., 2003). Therefore, the changes in the expression of surface proteins may be due to a vertical displacement of the membrane proteins by lipid action. While the expression of surface molecules such as adhesion and major histocompatibility molecules from human monocytes are inhibited by eicosapentaenoic acid. Instead, a significant increase in the expression of human lymphocyte antigen (HLA) is observed in monocytes incubated with docosahexaenoic acid (Maclean and Lucas, 2008).

Fatty acids play a major role in the production of eicosanoids, because they are key determinants of membrane-bound enzyme and receptor expression. The influence of fatty acids on gene expression is not well understood. Nevertheless, fatty acids released from membrane phospholipids are important second messengers or substitute for the classical second messengers, such as inositide phospholipid or adenoside monophosphate cyclic (AMPc) signal transduction pathways. These messengers act in a reversible manner at a precise intracellular location for a very short time in order to amplify, attenuate or deviate a signal in a direct or indirect (by conversion from arachidonic acid to eicosanoids) pathway (Riemersma et al. 1998).

The human body is greatly affected by nutritional status that in turn has been associated with alteration of the immune responses. Several dietary fatty acids or free fatty acids are involved in the modulation of the immune system through mechanisms that modify the immune response (Franchin et al., 2000). The immune parameters susceptible to modification by fatty acids supplied in the diet or free fatty acids added into cellular cultures are lymphocyte proliferation, cytokine production, activity of natural killer (NK) cells, phagocytosis, and expression of markers in the surface of the cells (Calder, 1995; Kelley et al., 1999; Roitt et al., 2001). The immune system modulation induced by fatty acids depends on several biological and methodological factors, such as the type and concentration of fatty acids, cell types, species of experimental animals, serum used in the *ex vivo* or *in vitro* cultures and so on (De Pablo and Alvarez de Cienfuegos, 2000).

Therefore, it seems that because of immunomodulation role associated with fatty acids, in future they may be applied to the amelioration and prevention of diseases characterized by an over-activation of the immune system, such as inflammatory or autoimmune disorders. In addition to these applications, unsaturated fatty acids may be used to reduce the susceptibility against bacterial infections due to the possibility of several lipids playing important roles in the efficient elimination of different micro-organisms or reducing host resistance against an infectious process due to the immunosuppression promoted by dietary unsaturated fatty acids. However, it should be noted that immunosuppression promoted by different dietary lipids has to be balanced, because this process may lead to an impairment of host response and increase the risk of infections.

2.3.2 Protein-energy- malnutrition

Protein-energy-malnutrition (PEM) also known as protein-energy deficiency (PED) is generally a nutritional problem that results from varying proportions of protein and calorie deficiency in infants and young children of developing countries (Nnakwe, 1995). It is a global public health problem, affecting children from African, Asian, Latin American and Caribbean regions (de Onis et al., 2000; Bisai et al., 2010). PEM is directly or indirectly responsible for about half of the 10.8 million deaths per year in under five children in developing countries. In Tanzania, it is estimated that 60% of deaths, among children under 5 years of ages, are associated with undernutrition (Villamor et al., 2005). The major risk factors that can predispose a child to having PEM include poverty, lack of access to quality food, cultural and religious food customs, poor maternal education, inadequate breast feeding, and lack of quality healthcare. In addition to macronutrient deficiency, there is clinical and/or sub-clinical deficiency of micronutrients (Jahoor et al., 2008).

Recent researches show that HIV prevalence is highly correlated with falling calorie consumption, falling protein consumption, unequal distribution of income (inequity) and other variables conventionally associated with susceptibility to infectious disease, however

transmitted. "The causal chain runs from macro-factors that result in poverty: through the community, the household, the individual and into the resilience of the individual's immune system. Work in cell biology has shown the mechanisms which connect malnutrition and parasite infestation; depressing both specific and non-specific immune responses by weakening epithelial integrity and the effectiveness of cells in the immune system. PEM, IDA, VAD, all of these poverty related conditions decrease resistance to disease in general and to HIV in particular.

Trace elements are required by human body for proper functioning but unlike most vitamins and minerals that our body needs, trace elements are needed only in extremely low quantities. The human body needs about 72 trace elements for normal functioning. There are seven essential trace elements described in human body chromium, copper, cobalt, iodine, selenium and zinc. Iron is an important dietary mineral and involves in various body functions. In humans, iron is an essential component of proteins involved in oxygen transport. It is also essential for the regulation of cell growth and differentiation. Iron is main component of hemoglobin a protein molecule that carries oxygen and plays critical role to the whole respiration process of the total iron in the body 60-70% is stored in hemoglobin. The body contains between 3.5 and 4.5% of iron 2/3 of which is present in hemoglobin (Pradeep et al., 2010).

The ubiquitous iron is an essential nutrient for most tissue cells and deficiency brings about recognizable deleterious results affecting many organs. The lymphoid apparatus is no exception. Iron deficiency with or without anemia is associated with partial atrophy of various lymphoid organs and alteration in many molecular and cellular immune functions. Iron and its binding protein have immunoregulatory properties and shifting of immunoregulatory balances by iron excess or deficiency may produce severe deleterious physiological effects. People with low levels of iron tend to have low resistance to infections. Anemia in HIV infected patients can have serious implication which varies from functional and quality of life decrement to an association with decreased progression and decreased survival. The prevalence of anaemia in HIV disease varies considerably ranging from 1.3-95%. Anaemia is more prevalent in HIV positive women, children and injected drug users. Human with advance HIV infection present some evidence of iron accumulation, which is manifested with an increased ferritin concentration (Pradeep et al., 2010).

Moreover, trace metal overload suppress immune function and increase the morbidity and mortality. If the iron overload becomes severe (usually when the amount of iron in the body exceeds 15 g) the condition is diagnosed as hemochromatosis. This is an inherited blood disorder that causes the blood to retain excessive amount of iron, which lead to serious health consequences such as cirrhosis of the liver (Bullen *et al*., 1991). Irons stored in the body become depleted and hemoglobin synthesis is inhibited. Iron is central to physiology in general and required for particular steps of the HIV replication life-cycle in cells.

HIV infection has therefore been associated with disturbances in host iron metabolism. Advanced HIV/AIDS disease condition, anemia can coincide with increase ferritin and bone marrow iron content and the anemia is commonly unresponsive to iron supplementation (Strauss, 2004). Increased bone marrow iron is associated with shortened survival and increased opportunistic infections. Iron could be playing an important role in the interaction between host and virus. Host homeostasis adapt during deficiency, overload and infection to balance requirement against toxicity and availability to potential pathogens. Knowledge of these interactions is necessary to predict morbility response to disturbance in host iron homeostasis (Pradeep et al., 2010). Because the progression of HIV infection towards its

more advance stages is accompanied by increasing body iron stores there is urgent need for careful clinical studies to clarify the role of iron status on the course of HIV infection. Proper iron supplementation may provide sufficient iron to restore normal storage levels of iron and to replenish hemoglobin deficits thereby increase the survival of HIV sero-positives.
Nowadays it is well recognized that all infections have an adverse effect on nutritional status. However, the clinical and public health significance of the effect of a single infectious episode depends on the prior nutritional state of the individual. Recently, a clear synergistic relationship between nutrition and infection has been elucidated and thus most public health interventions for prevent infection focus on correction of malnutrition. With the ever increasing resistance of important pathogenic microorganisms to available chemotherapeutic agents, this area of study calls for further researches (Strauss, 2004; Villamor et al., 2005; Jahoor et al., 2008).

2.3.3 Impact of poverty on environment

The fact that HIV/AIDS affects people in their productive ages between ages 25 and 45 years, the poverty that the epidemic precipitates can have deleterious impacts on agricultural productivity and natural resources. These impacts can hit particularly hard in a country like Tanzania, where livelihoods are highly dependent on agriculture, mining and fishing. As men and women die or become too ill to work, family members are forced to find new ways to provide for their families. The loss of income from a male head-of-household puts additional burdens on his wife and children to find alternative sources of income, which can ultimately lead to more intense and less-sustainable resource use and extraction. Some regions in northern coastal Tanzania, such alternative practices often include unsustainable harvesting of forests and forest products such as wild foods and medicinal plants, which are then sold at local markets. Increased woodcutting to produce charcoal for sale is also common, especially when families face severe food shortages and must secure cash quickly to buy food. And the use of small-mesh nets for both marine and freshwater fishing has increased as widowed women and their children try desperately to make a living from declining shallow-water fish stocks.
Therefore, poverty and food insecurity increase sexual risk taking, particularly among women who may engage in transactional sex to procure food for themselves and their children. Women's economic dependence on their partners may also make it difficult for them to insist on safer sex like condom use. Several ethnographic studies have suggested that material poverty has increased the incidence of transactional sex. To some extent, this indicates that while the poor are undoubtedly hit harder by the downstream impacts of AIDS, in a variety of ways, their chances of being exposed to HIV in the first place are not necessarily greater than wealthier individuals or households. This brings a different school of thought that possibly poverty itself fuelled by AIDS, is placing individuals from poor households at greater risk of exposure to HIV via the economically-driven adoption of risky behaviors.
In Tanzania there are substantial urban-rural, regional and socio-economic divergences, which have driven youths to migrate to urban areas looking for jobs (better living) and some engaging themselves into risky activities like unsafe sex, prostitution and drug abuse just to mention a few. On the other hand, rural poor children are more likely than their urban counterparts to die, and when they survive, they are more likely to be malnourished as depicted in poverty and human development report (PHDR, 2005).

2.4 Parasitosis and HIV/AIDS

Since the first HIV/AIDS cases were described, a high prevalence of gastrointestinal alterations has been reported, especially diarrhea associated with parasitosis. This became more evident when the appearance of a syndrome named "Slim Disease", characterized by an intense weight loss accompanied by chronic diarrhea, prolonged fever and diffuse muscle weakness, was observed in Africa, especially in Uganda. However, the site and timing of the first reported cases suggest that the disease arose in Tanzania. Studies conducted in Tanzania, Uganda and other African countries have shown prevalence of 60 to 80% of parasitic organisms attributing to "Slim Disease", such as *Isospora spp., Cryptosporidium spp., Salmonella spp., Shigella spp.* and *Campylobacter* species, amounting to a prevalence of 60 to 80% (Tarimo et al., 1996; Brink et al., 2002). "Slim Disease" has been observed in advanced stages of HIV infection (Mhiri et al., 192). The expression "Wasting Syndrome" was adopted in substitution by WHO in 1987 on the basis of criteria laid down by the (Center for Disease Control [CDC, 1987]).

Opportunistic infections caused by intestinal parasites vary according to the geographical area and the endemic levels in each location. The progressive decline of immunological and mucous defense mechanisms predisposes patients to early, intermediary and late gastrointestinal manifestations of HIV infection. At later stages of the disease, the alterations in non-specific defense mechanisms in the production of immunoglobulin A and the reduction in local immune cell response also progress, thus increasing the susceptibility to a number of intestinal opportunistic pathogens, among which *Cryptosporidium parvum*, *Isospora belli* and Microsporidia species are the most prominent (Akinbo et al., 2010).

After the emergence of AIDS, these parasites, until then known solely in veterinary medicine, were no longer considered as commensal organisms and are nowadays recognized as opportunistic pathogens common to these patients. Infections by these agents constitute a major secondary aggravating factor of the disease, often responsible for worsening the general health conditions, due to manifestations of diarrhea which are difficult to control, sometimes resulting in the death of the patient. Thus, where as infections in the gastrointestinal tract play a critical role in AIDS pathogenesis and diarrheic diseases assume a prominent role, reaching a rate of up to 50% in developed countries, in developing countries there have been reports of incidence of up to 95%, as in Haiti and the African continent. Amongst the causes of diarrhea in developing countries, those of a parasitic origin are prominent in patients with HIV/AIDS (Akinbo et al., 2010).

The immune system can simplistically be divided into two components, namely, nonspecific immunity (skin, other mucosal barriers and soluble factors) and the adaptive immune system, which involve recognition of pathogens and subsequent production of antibodies as well as cell-mediated defense. The immune system is being challenged by the worldwide increment in immunological stressor agents of chemical, physical, biological, mental, and nutritional origin. However, the diversity and intensity of these risks or etiologic factors for HIV/AIDS vary from person to person, from group at risk to group at risk, from country to country, and from continent to continent. This is the principal reason why the frequency of AIDS is not homogeneous in all places and countries (Giraldo, 1998; Roitt et al., 2001).

The capabilities and possibilities of the immune system are neither infallible nor infinite. HIV/AIDS is the maximum state of deterioration that the human immune system can reach as result of HIV-infection. If the pathogenic process of AIDS is not stopped, eventually it will kill the person (Roitt et al., 2001). While the most important risk factor for HIV/AIDS in developed countries is the new epidemic of drugs abuse. The most important risk factor for

HIV/AIDS in underdeveloped countries is poverty, with all its consequences: malnutrition, unsanitary conditions, infections, parasites, and lack of hope for a better life, all of which have reached unprecedented high levels in the last few decades (Giraldo, 1998). Any component of the immune system can be functionally or genetically abnormal as a result of acquired, for instance through HIV infection, lymphomas, frequent use of high-dose steroids or other immune-suppressive medications) or congenital illnesses, with more than 120 congenital immunodeficiencies described to date that either affect humoral immunity or compromise T-cell function. Immunosuppression may also occur in malnourished persons, patients undergoing chemotherapy for malignancy, and those receiving immunosuppressive therapy. However, for parasitic infections, cell-mediated (T-cell) abnormalities predominate. Such persons tend to be susceptible to common pathogens with delayed clearance. With profound cell-mediated defects, reactivation of previously controlled pathogens may occur. Moreover, such individuals are at risk of infection by opportunistic "nonpathogenic" parasites (Cunningham and Fujinami, 2000). Nevertheless, with reconstitution of the cell-mediated immunity, the risk of parasitic infections reverts to that for a normal host.

The mechanisms, by which malnutrition, tuberculosis, STI, malaria, trypanosomiasis, schystosomiasis, leishmaniasis, systemic mycosis, as well as other infections and parasites weaken, destroy, and collapse the immune system will be briefly elucidated. These mechanisms should well understood for any attempt to design more effective interventions on the current occurrence of the HIV/AIDS epidemic in the poorest areas of the underdeveloped countries, where the poor have never before been so poor and so sick as they are now. Moreover, these poor countries, the levels of malnutrition, microbial and parasitic infections have reached very alarming levels (Cates, 1988).

Therefore, poverty, parasitosis and HIV/AIDS seem to be closely interlinked and co-circulate in many populations, particularly in underdeveloped countries. HIV/AIDS, parasitic infections like malaria and other opportunistic infections, and in a few are by far the commonest causes of ill-health and death in Tanzania and other poor countries of the world. Most of these countries happen to be in the tropics and temperate countries in Africa, Asia, and South America. Currently, parasitic infections are major health concerns attributed to high morbidity and mortality in developing countries especially among HIV-infected persons. Children aged 2 to 5 years are most at risk of severe morbidity (Chopra et al., 2006).

Programs to prevent HIV transmission are unlikely to succeed unless they address the underlying causes of its spread. HIV prevention must be based on scientific evidence regarding cofactor conditions, not, as they currently are, on unproven assumptions about the primacy of behavioral factors. In addition to food security, deworming, schistosomiasis prevention and treatment, and malaria control programs should thus be integrated as critical components of a broad-based approach to HIV prevention.

2.4.1 Protozoan infections and HIV/AIDS

Malaria is caused by an intracellular protozoan transmitted via the bite of an infected female *Anopheles* mosquito. Malaria is not an opportunistic infection for HIV-infected people, but the effect of HIV infection on the natural history of malaria has not been completely defined. HIV infection and malaria coexist in regions where the health surveillance systems are poorly performing so that the magnitude of any interaction is difficult to determine. Nevertheless, the evidence of such interaction has recently grown and is still increasing (Nkuo-Akenji et al., 2008). The incidence of symptomatic malaria episodes, severe or

uncomplicated, and the corresponding parasite density is higher in HIV infected individuals with low CD4 count (Atzori et al., 1993; Sherman, 1998).

In Tanzania, malaria is the major killer of children under 5 years of age. Prevalence rates of malaria among children between 6-59 months old vary according to geographic positions and socio-economic activities in a particular area. The highest rate has been observed around Lake Zone regions, Kagera region taking a lead with prevalence of 41.1% followed by Lindi (35.5%) while Zanzibar and Arusha have lowest rates of 0.8% and 0.4% respectively. Malaria is the cause of more mortality and morbidity in Tanzania than any other disease, in large part due to growing resistance to antimalarial drugs. It is estimated that over 1% of GDP is devoted to the disease, representing US$2.2 per capita, and 39% of total health expenditure nationally. Government facilities devote almost one-third of their resources to the disease.

Due to poor living conditions, the majority of Tanzanians suffer from malaria -a preventable disease that can have a serious negative impact on pregnant women and young children. Mothers who contract malaria during pregnancy run the risk of having low birth weight babies, maternal anemia, impaired fetal growth, spontaneous abortions, stillbirths, and premature babies. Recently, the country introduced an anti malaria campaign '' Malaria *Haikubaliki*" a Swahili slogan that means ''Malaria is unacceptable'' - which involves all sectors of the society including entertainment, business, sport and religion sectors in the battle against malaria across the country. "Africans think that malaria is inevitable; that there is nothing they can do about it. We are going to prove this wrong. We can eliminate malaria deaths," said the Minister for Health and Social Welfare, Prof. David Mwakyusa. This initiative is of paramount importance because of the presumed potential interaction between malaria and HIV/AIDS.

Researchers have demonstrated a potential role for the dual infection in fueling the spread of HIV/AIDS and malaria in endemic regions (Whitworth et al., 2000; Abu-Raddad et al., 2006; Nkuo-Akenji et al., 2008). Besides a direct effect, HIV infection may indirectly influence the malaria burden by increasing the malaria parasite biomass and consequently the probability of drug resistant parasites emerging. Antimalarial drug resistance, particularly for *Plasmodium falciparum*, is considered a major contributor to the global resurgence of malaria observed over the last three decades and one of the greatest obstacles for an effective malaria control (Marsh, 1998). The basis of resistance lies in one or several genetic mutations in the parasite genome. Malaria parasites with such mutations when in contact with a given drug survive the treatment and eventually spread (Van Geertruyden et al., 2008). Evidences collected from four longitudinal population-based studies in rural Uganda and Malawi, where malaria is highly endemic showed odds ratios of symptomatic malaria in HIV infected compared to uninfected adults of 6.0, 3.4 and 1.2 for CD4 counts <200/µl, 200–499/µl and ≥ 500/µl, respectively, in Uganda (Whitworth et al., 2000). In Malawi, incidence rates of symptomatic malaria in HIV-1 infected adults varied with CD 4 count; compared to individuals with CD4 count of ≥ 500, the malaria incidence was 3 fold higher with a CD4 count of 200–499/µL and a 4.4-fold higher with a CD4 count<200/µL (Borkow et al., 2001; Graham et al., 2006).

Additionally, it is purported that HIV infection contributes to the emergence and spread of antimalarial drugs resistance by increasing drug exposure and drug pressure. HIV infection increases the probability of a malaria infection progressing to symptomatic illness and to a higher parasite density, increasing the probabilities of treatment and contact between the parasites and the drug (Nkuo-Akenji et al., 2008). This can be explained by the fact that,

HIV-infected patients with already low immunity suffer frequently of non-malaria-attributable acute fevers that may be misdiagnosed as malaria and treated with antimalarials. This phenomenon can justify the observed increase in the parasite biomass in HIV symptomatic patients and asymptomatic carriers alike. Besides contributing to the emergence and spread of antimalarials drug resistance, HIV infection may influence and modify its expected geographical pattern as consequence of and influence of various malaria control intervention by exposing large number of Plasmodium parasites to antimalarials. A way out for this is an easy access, prompt diagnosis and appropriate combination therapy that can alleviate or halt the emergence and spread of antimalarial drug resistance (Van Geertruyden et al., 2008).

A study conducted in a rural area of Malenga Makali (Tanzania) involving 300 sexually-active adults selected at random among patients, who were attending a dispensary because of diarrhoea of at least 2 weeks' duration, revealed potential associations between the patient's health (in terms of the WHO's clinical definition of AIDS), HIV-1 seroprevalence and malaria and other parasitic infections. Although malaria infection was more common in HIV-1 seropositives than in the seronegatives, the intensity of the *Plasmodium falciparum* infections, intestinal amoebiasis and giardiasis did not appear to be correlated with HIV infection. In contrast, intestinal infections with *Cryptosporidium parvum* and *Isospora belli* were virtually restricted to HIV seropositive individuals who had had diarrhoea for a relatively long time (Atzori et al., 1993). Similarly, it was noticed that maternal weight and low CD8+ cell counts were inversely associated with low body weight (LBW). While advanced-stage HIV disease, previous history of preterm birth, *Plasmodium falciparum* malaria, and any helmintic infection are associated with higher risk of LBW (Dreyfusset al., 2001). The intestinal parasites *Entamoeba histolytica* and *Strongyloides stercoralis* seemed to be predictors of LBW despite their low prevalence in the cohort. Moreover, the newborns' body mass index, midupper arm circumference, CD4 cell count <200 x 10^6 cells/L (200 cells/mm^3), primiparity, maternal literacy, and infant HIV infection at birth are significantly associated with birth weight in addition to risk factors associated with the LBW.

Cryptosporidiasis in AIDS patients usually causes chronic, bulky and intermittent diarrhea, with liquid non-bloody stools, accompanied by pain and abdominal colic, and a noticeable loss of weight can be observed. Asymptomatic cases are rarely described, occurring mostly in developing countries, Tanzania inclusive with patients showing milder. Cryptosporidium spp are common opportunistic parasites that cause chronic diarrhoea and wasting in HIV/AIDS patients with CD4+ T-cell counts <100 cells/μL and antimicrobial agents have limited efficacy in preventing or eradicating infections with it. Although studies assessing reduction in the incidence of cryptosporidiosis are lacking, diarrhoea due to cryptosporidia are known to resolve spontaneously with immune restoration among HIV/AIDS patients on antiretroviral therapy. Extra-intestinal manifestations have been clearly described in the literature, especially in the gall bladder, biliary ducts and pancreas, leading to conditions such as papillary stenosis, sclerosing cholangitis and calculous cholecystitis as well as chronic bronchitis (McGowan et al., 1997).

Isospora belli is a coccidian parasite that has a global distribution limited to mainly tropical regions in developing countries where it is endemic (especially Africa, the Middle East, and South America). The parasite invades the intestinal epithelium, where it completes its life cycle in the cytoplasm of the enterocyst. Unsporulated oocysts are excreted in feces and mature outside the host, where they develop into infective sporulated oocysts. Infection is

then acquired through ingestion of these infective oocysts. Immunodeficiency was shown to increase the susceptibility to infection with *Isospora belli*, which accounted for up to 20% of cases of diarrhea in HV/AIDS patients. The lower prevalence of isosporiasis may be ascribed to the secondary prophylaxis for pneumocystosis through administration of sulfamethoxazole-trimethoprim during the course of HV/AIDS, since *Isospora belli* is sensitive to this chemotherapeutic agent.

The diarrheic condition is also noteworthy and is accompanied by fever, intestinal colic, anorexia, abdominal pain, loss of weight and peripheral eosinophilia. Isosporiasis can also show extraintestinal dissemination features, affecting the mesenteric, periaortic, mediastinal and tracheobronchial lymph nodes. It may also be related to biliary disease, causing manifestations of acalculous cholecystitis. Despite this high prevalence, classical protozoa such as *Giardia lamblia* and *Entamoeba histolytica* are less frequent as causes of severe illnesses in HIV-infected patients, when compared with *Isospora belli*and *Cryptosporidium parvum* and they are not considered as opportunistic infections in HV/AIDS. Amebiasis may present with invasive characteristics, but this has rarely been reported in the literature (Angarano et al., 1997).

Parasite infections especially malaria, and intestinal parasites undermine the nutritional status and compromise the immune system yet further, effectively exhausting it. These parasite infections are endemic in Tanzania, but the situation is made worse by inadequate health care and infrastructure, which actually by itself a function of poverty and low levels of development that leaves most parasite infections untreated.

2.4.2 Intestinal helminthic infections and HIV/AIDS

Neglected tropical diseases (NTDs) are a devastating burden for the people of Tanzania and are prevalent throughout the country. The country is endemic with all seven of the most common NTDs: schistosomiasis, lymphatic filariasis, onchocerciasis, trachoma, and the soil-transmitted helminths (hookworm, ascariasis, and trichuriasis). Such high prevalence rates of multiple NTDs increase the risk for co-infection with two or more diseases, a phenomenon that leads to more severe health consequences (Bundy et al., 2000; Luong et al., 2003).

Intestinal helminths are ubiquitous in low-income countries with prevalence of, for example 50–80% for ascariasis, trichuriasis and hookworm infections in many populations. Intestinal helminths induce immunological alterations that favor the progression from HIV seroconversion to AIDS. After HIV has spread to the systemic circulation its replication is limited by the fact that usually few activated lymphocytes and differentiated macrophages are present in the blood stream and that resting T cells and undifferentiated monocytes are not susceptible to HIV infection. However, in patients infected with intestinal helminths the number of activated T cells expressing human leucocyte antigen (HLA)-DR and HIV coreceptors is elevated. This is followed by HIV replication, preferentially in T-helper cells (Th cells) of Th2 and Th0 type clones. While Th2 cells are usually abundant in individuals infected with helminthes. Similarly, peripheral blood mononuclear cells of patients with helminthic infection are significantly more susceptible to infection with HIV than those of uninfected controls. Finally, elevated IL-4 levels, characteristic of the Th2 type of immune response in helminthic infections, down-regulate Th1 differentiation and function.

Therefore, parasitic infection could be a primary contributing factor to HIV/AIDS in Africa where men and women alike are at risk. Parasites are also endemic in people who are also at higher risk for HIV/AIDS, and most of these countries are underdevelopment/third world.

Activation of T cells, macrophages and other antigen presenting cells is necessary for HIV replication. This fact alone, may explain why lymphotropic and monocyte-tropic viruses originated in parasite endemic regions where suitable hosts reside. Indeed, Human T-lymphotropic virus-1 (TLV-1), HIV and Epstein-Barr virus (EBV). These are lymphotropic viruses that are found in close geographical relationship with parasite endemic regions. Women, especially in these developing countries of the world, are also prone to harbor chronic vaginal infections such as trichomoniasis, or have other predisposing vaginal factors for HIV/AIDS.

Among the helminthes in association with AIDS, there is no doubt that the most important pathogen is *Strongyloides stercoralis*, which is geohelminth presents its major effects in immunodepressed patients, leading to the dissemination of the infection. This occurs in transplanted patients, individuals presenting malnutrition and patients submitted to prolonged use of corticosteroids, suffering from leukemia, lymphomas or AIDS (Cimerman et al., 1998). In immunosuppressed patients, self-infestation is speeded up and a large number of larvae are released, causing the dissemination of the infection. Nevertheless, data from a single source published in 2010; show that in Tanzania the prevalence of helminthic infections in HIV-positive patients is around 8.2%, in comparison with 15.8% in non-HIV infected population (Mwambete et al., 2010). Hookworm (*Ancylostoma duodenale* and *Necator americanus*) infections being the most prevalent among HIV patients (17.1%) followed by *S. stercoralis* (3.3%). Although, there was no significant difference in respect of prevalence rates of helminthic infections between HIV-infected and non-infected patients; the study revealed direct correlation between CD4+ counts (HIV status) with helminthic infections among the HIV-infected patients. Nevertheless, helminthic infections are a grave health problem in Tanzania particularly among children from poor households as indicated in the Table 3 below (Mwambete and Kalison, 2006).

CD+4 counts	**Helminthiasis (%)**				
	Trichuris	**Hookworm**	**Ascaris**	**None**	**Total**
< 100	5(1.4)	2(0.6)	2(0.5)	14(3.8)	**23(6.4)**
101 - 200	50(13.7)	31(8.5)	35(9.6)	125(34.3)	**241(66.2)**
201 - 400	21(5.8)	6(1.6)	8(2.2)	31(8.5)	**66(18.2)**
401 - 500	7(1.9)	0(0.0)	1(0.3)	13(3.6)	**21(5.8)**
501 - 600	2(0.5)	0(0.0)	0(0.0)	4(1.1)	**6(1.6)**
601- 700	1(0.3)	0(0.0)	0(0.0)	6(1.6)	**7(1.9)**
Total	**86(23.6)**	**39(10.7)**	**46(12.6)**	**193(53.0)**	**364**

Table 3. Prevalence of helminthiasis in relation to HIV/AIDS status (CD+4 counts)

The other most common helminthes in Tanzania include *Ascaris lumbricoides, Trichuris trichura, Enterobius vermicularis* just naming a few. These are ubiquitous parasites in tropical and subtropical areas associated with causing diarrhea and hyperinfection syndrome in

individuals with immunosuppressive disorders, including HIV/AIDS. Unfortunately, there are scant data or unavailable at all that relate and indicate the magnitude of the problem of parasitic infections among HIV-infected patients. A study conducted in rural Tanzania in 1995, revealed the prevalence rate of 81.8% (n= 287) of intestinal parasites in HIV-negative than in HIV-positive patients. The prevalence of *Ascaris lumbricoides* was higher in HIV-negative than in HIV-positive patients ($p < 0.01$; $p < 0.04$) (10.5% and 3.7% for *A. lumbricoides*). On the other hand, *Strongyloides stercoralis* prevalence was higher in HIV-positive than in HIV-negative patients ($P < 0.01$).

Schistosomiasis is another systemic helminthic infection, which is the second most prevalent tropical disease after malaria and affects approximately 200 million people in Africa, Asia, South America and other temperate regions. Severity of the infection largely depends on the intensity of infection, the *Schistosome* species involved, the topographic site affected by sequestered eggs and the immune responsiveness of the host. Urinary schistosomiasis is more prevalent in school-aged children than in adults. A prevalence of 57.9% was revealed in a study conducted in Kilosa (a semi-arid central district in Tanzania) among school–age children, which somehow is an indication of probably having even higher prevalence in lake areas (Mkopi et al., 2005; Clement et al., 2006).

In women, genital schistosomiasis occurs in about 60% of individuals infected with *Schistosoma haematobium*. Genital manifestations involve the vulva, vagina and the cervix as well as upper genital organs and result in pathology similar to that observed in some STIs. The cervix is the site predominantly affected, followed by the vagina and vulva. Thinning, erosion and ulceration of the epithelium is a typical clinical finding of genital schistosomiasis (Kjetland et al., 1996). This is important, as breaks in the integrity of the mucosal barrier, due to either trauma or sexually transmitted genital ulcer diseases are associated with an increased risk of HIV transmission (Mabey 2000).

Likewise, Schistosome eggs stimulate a complex array of cellular and humoral immune responses. Inflammations/granulomata form around eggs with a radius 5–10 times larger than the size of the egg. These granulomata are composed among others of activated lymphocytes, macrophages and epitheloid cells as well as some Lagerhan's giant cells, cell types known to express the CD4+ T cell receptor. In the wall of the bladder a considerable proportion of these granulomata ulcerate into the lumen. Similarly, in genital schistosomiasis egg-induced ulcerative lesions could be the port of entry for HIV. In the cervix, peri-oval granulomata are frequently formed near the basal layer of the epithelium. Within the egg granulomata, and also in adjacent areas, T cells, macrophages and Lagerhans cells abound (Helling-Giese et al., 1996). In mice, CD4+ T cells amount to 8–10% of all granuloma cells. The abundance of CD4 receptor-bearing cells within the confines of the granulomata and in adjacent areas make a rapid binding of virus after penetration through the friable and eroded epithelium of the cervix very likely. Therefore, genital schistosomiasis in males also points at an increased risk of HIV transmission. *Schistosoma haematobium* infection in males induces a chronic inflammation in the pelvic genitals, which looks like bacterial urethritis, accompanied with an increased viral shedding in the semen in HIV co-infected individuals.

Dar es Salaam, which is the largest commercial city, has an extensive drain network, mostly with inadequate water flow, making *Anopheles* and *Culex* larvae common. However, the importance of drains as larval habitats was previously unknown. The researchers analyzed detailed surveys of both mosquito habitats and drain conditions in the city; their findings suggest that simple but well-organized environmental management interventions, aimed to

restore and maintain the functionality of drains, may help reduce mosquito-borne disease transmission. Lymphatic filariasis (LF) is a leading cause of disability due to parasitic infections in Tanzania. As of 2004, all districts were endemic with the disease, and prevalence rates reached up to 45%. Onchocerciasis (river blindness) has been documented in five regions and 15 districts throughout Tanzania, with an estimated 4 million people at risk for infection overall. In certain focal endemic areas, prevalence rates reach up to 64%.

Lymphatic filariasis is widely distributed in the tropics. It particularly occurs in areas with a high HIV prevalence such as sub-Saharan Africa and south-east Asia. There is no data available to show an impact of HIV infection on the prevalence or on the natural history of the disease. However, lymphatic filariasis is an example for a harmful interaction in the other direction. Replicative capacity of HIV is significantly enhanced in peripheral blood mononuclear cells from patients with untreated lymphatic filariasis. Consequently, untreated patients with lymphatic filariasis and co-infected with HIV could be at risk for rapid progression to AIDS once infected with HIV. Furthermore, the chronic manifestations of filariasis can have significant, and often very negative, social impacts. The chronic disabling manifestations of this disease, including lymphoedema of the limbs, breasts and external genitalia, have a profoundly detrimental effect on the quality of life of affected individuals.

The degree of social disability varies between cultural settings, but the degree of stigmatization appears to be directly correlated with the severity of visible disease (Evans et al., 1993). In conservative contexts, affected individuals avoid seeking treatment for fear of drawing attention to their condition. Failure to treat the disease results in recurrent acute febrile attacks and progressive damage to the lymphatic system. Without access to simple hygiene advice, sufferers are unable to prevent further progression of the outwardly visible complications of LF.

Women bear a double burden in societies where much of their role and identity is dependent upon marriage and the ability to give birth to children. Young unmarried women with LF may be forced to lead a reclusive existence in an attempt to hide their illness or because their limited marriage prospects make them a burden to their families. In Thailand and in West Africa there is a general perception that children born to a woman affected by LF will be similarly affected. Shame and anxiety related to difficulties in conceiving children are common for LF patients around the world (Evans et al., 1993). Young females with LF are considered poor marriage prospects because the disease's recurrent debilitating acute episodes limit their ability to perform paid and unpaid work. The costs associated with long-term health care as the disease progresses result in perceptions of these women as financial burdens.

2.4.3 Ectoparasite infections and HIV/AIDS

Epidermal parasitic skin diseases (EPSD) occur worldwide and have been known since ancient times. Despite the considerable burden caused by EPSD, this category of parasitic diseases has been widely neglected by the scientific community and health-care providers. This is illustrated by the fact that in the 2006 edition of The Communicable disease control handbook, a reference manual for public health interventions, only one EPSD (scabies) is mentioned (Fieldmeier and Heukelbach, 2009). Epidermal parasitic skin diseases fulfill the criteria defined by Ehrenberg and Ault (2005) for neglected diseases of neglected populations, but are not listed on national or international agendas concerning disease

control priorities (Ehrenberg and Ault, 2005). This probably explains why efforts to control EPSD at the community level have very rarely been undertaken (Heukelbach et al., 2002).
Six EPSD are of particular importance: scabies, pediculosis (head lice, body lice and pubic lice infestation), tungiasis (sand flea disease) and hookworm-related cutaneous larva migrans. They are either prevalent in resource-poor settings or are associated with important morbidity. This chapter focuses on these diseases, summarize the existing knowledge on the epidemiology and the morbidity in resource-poor settings and focus on the interactions between EPSD and poverty. The distribution of EPSD is irregular, and incidence and prevalence vary in relation to area and population studied. A study in a resource-poor community in urban Bangladesh, for example, showed that virtually all children aged less than 6 years developed scabies within a period of 12 months (Stanton et al., 1987). In a rural village in Tanzania, the overall prevalence is 6% and in rural India 13%, while in Australian Aboriginal communities the prevalence in this age group approached 50% (Sharma et al., 1984; Currie and Carapetis, 2000). Situation is even worse in displaced communities, where of 5-9-year-olds children in Sierra Leone, 86% are infested with *Sarcoptes scabiei*.
Poverty influences the epidemiology of EPSD in several ways because it creates animal reservoirs, ensures ongoing transmission, facilitates atypical ways of spreading the infectious agent and increases the chances of exposure. This results in an extraordinarily high prevalence and intensity of infestation and significant morbidity of EPSD. Again, stigma, lack of access to health care and hesitancy in seeking health care are the reasons why EPSD frequently progress untreated.
Inequality and neglect seem to be the major driving forces that keep the disease burden at an intolerably high level. Health-care stakeholders and political decision-makers must acknowledge that EPSD are debilitating and merit much more attention from health professionals than hitherto given. The ongoing uncontrolled urbanization in Tanzania and other developing countries makes it likely that EPSD will remain the overriding parasitic diseases for people living in extreme poverty and remain indicators of neglect by societies and particularly public health policies.

3. Conclusion

There are plenty of ways to help prevent HIV/AIDS by changing the biological and economic context in which the epidemic is spreading. Behavioral interventions are necessary, but their effectiveness is often a matter of faith more than documented results. So far, neither preaching abstinence nor handing out condoms has had an appreciable impact on the epidemic because sexual behavior is not the most important difference between high-prevalence and low-prevalence populations. Governments can change customs regulations or deliver safe water supplies and multivitamins more easily than they can chase down every person having unprotected sex. It is not a coincidence that the countries with the highest rates of HIV have serious environmental, economic, and bureaucratic problems. Most of the worst affected countries like Tanzania have problems of border-breach, ineffective parasitic infections control programmes, risky environment for HIV infection that all together is HIV/AIDS related.
Researchers have shown that there is a common immunopathogenetic basis for the detrimental interaction between HIV and pathogens biologically as different as for example, plasmodia and helminthes. Chronic immune activation by parasitic infection could be one of the several causes of T cells depletion in HIV infection and could considerably contribute to

the progression of HIV disease. Infection with intestinal helminthes, parasitic organisms living in a compartment aside of the systemic immune system, induces a status of chronic immune activation. The prolonged and enhanced immune activation is even more prominent in systemic parasitic infections, whether protozoa or helminthes. Even before HIV infection supervenes, chronic immune activation induced by parasites is associated with several of the immunological features of HIV disease.

In principle, the fact that parasites preferentially activate a Th2 type of help, among other functions Th2 cells down-regulate the development of Th1 cells, inhibit macrophage activity and impair the cytotoxic T-lymphocyte response. They encourage entry of HIV through the CD4 receptors located on the Th2 cells surfaces. The early presence of IL-4 is the most potent stimulus for Th2 differentiation. The inducing effect of IL-4 dominates over other cytokines so that, if IL-4 levels reach a certain threshold, differentiation of the Th cell into the Th2 phenotype ensues, thus favoring proliferation of the HIV, and particularly because of the low levels of Th1 cells that are necessary for the containment of HIV infection via cell-mediated immunity, whereby intracellular pathogens like HIV could be removed.

In contrast to HIV infection, which in the tropics is mainly a sexually transmitted disease, parasitic infections usually abound in childhood and/or adolescence. Moreover, in endemic areas sensitization towards the respective antigens already occurs early during prenatal period (*in utero*) as mothers are likely to be infected with the parasites as well. Notwithstanding, exposure to antigens *in utero* results in generation of cytokine responses similar to those found in adults and the ability of primed T cells to react accordingly can persist into childhood. Such early exposure generate memory cells of Th2 type of help would be a considerable disadvantage when later in life the immune system of the affected individual encounters HIV. Consequently, not only is HIV infection acquired more easily but more rapid progression from asymptomatic HIV infection to AIDS disease.

Summarily, it seems evident that people living in the tropics not only face a health threat in view of still expanding HIV epidemic, they also have to fear that once infected with HIV this will alter the natural history of parasitic infections they are suffering from in an unfavorable way. Besides the parasites they harbor impair the immune response towards HIV. In this way makes rapid progression from HIV infection to AIDS rather likely. As the great majority of parasitic diseases can be treated and /or to some extent be prevented, it is now logical that the control of parasitic infections should be included as a tool in the combat of HIV infections.

Although HIV prevalence has fallen in Tanzania over the past decade, thousands of people become infected with HIV every year and 86,000 Tanzanians died from AIDS in 2009 alone. While the poor are undoubtedly hit harder by the downstream impacts of AIDS, in a variety of ways, their chances of being exposed to HIV in the first place are not necessarily greater than wealthier individuals or households. However, the poor are more prone to parasitic infections. Poverty (illiteracy, food insecurity, poor public services and inequality), parasitosis and HIV/AIDS are the major problem currently facing Tanzanians. Therefore, a joint effort is urgently required to combat them in their totality, with that approach, the victory is evident.

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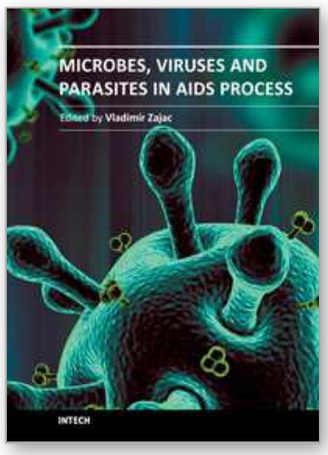

Microbes, Viruses and Parasites in AIDS Process

Edited by Prof. VladimÃr Zajac

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The main goal in compiling this book was to highlight the situation in Africa in terms of AIDS and opportunistic diseases.Â Several chapters reveal great poverty, an apocalyptic situation in many parts of Africa.Â Global migration of people resulted in their exposure to pathogens from all over the world. This fact has to be acknowledged and accepted as African reality. New, unconventional hypotheses, not determined by established dogmas, have been incorporated into the book, although they have not yet been sufficiently validated experimentally.Â It still applies that any dogma in any area of science, and medicine in particular, has and always will hinder progress. According to some biologists, in the future, AIDS is very likely to occur in a number of variations, as a direct result of the ongoing processes in the global human society. Thus, we urgently need a comprehensive solution for AIDS, in order to be ready to fight other, much more dangerous intruders.

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