THE ASSOCIATION BETWEEN NUTRIENT INTAKE, BODY ANTHROPOMETRY AND QUALITY OF LIFE OF HIV/AIDS PATIENTS IN GHANA.

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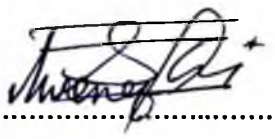
THIS THESIS IS SUBMITTED TO THE UNIVERSITY OF GHANA, LEGON IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF M.PHIL NUTRITION DEGREE.

DEPARTMENT OF NUTRITION AND FOOD SCIENCE
UNIVERSITY OF GHANA, LEGON.

AUGUST, 2003.

DECLARATION

I, Douglas Twenefour, hereby declare that with the exception of references to other people's works, which have duly been cited, this work is the result of my own research under the supervision of Dr. W. B. Owusu.



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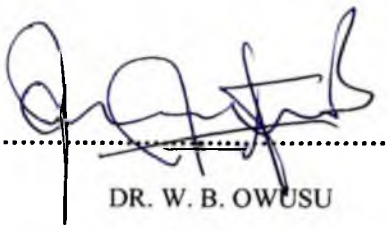

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DEDICATION

This work is dedicated jointly to Dr. Anna Lartey of the Department of Nutrition and Food Science, and the late Prof. Richard Orraca – Tetteh, formerly of the same department for inspiring my interest in HIV/AIDS issues.

ACKNOWLEDGEMENT

As expected in projects such as this, the putting together of the final product would not have been possible without the inputs of many individuals, who helped in diverse ways to enable me achieve this goal. I am therefore indebted to these individuals and will want to seize this opportunity to express my sincere gratitude to all who made it happen. First of all, I want to be grateful to God almighty for being kind to me and seeing me through to the successful completion of this project work.

For his constructive criticisms and intuitive suggestions during the course of his supervision of this work, I am very grateful to Dr. W. B. Owusu. He provided me with immense academic guidance and his intellectual comments on earlier drafts have made significant contributions to the quality of this product. I am also indebted to Prof. Asibey – Berko, Dr. Anna Lartey, Dr. Esther Sakyi – Dawson, Dr. K. Tano – Debrah, and colleague students of my department who were present during my results presentation for their insightful submissions and criticisms that have gone a long way in reviewing various aspects of this work.

My sincere gratitude goes to Prof. Neequaye and Dr. Lartey of the Department of Medicine, Korle Bu Teaching Hospital for allowing me to recruit patients at the Hospital for the study. I want to thank Barbara of the Wisdom Association for helping with the data collection at Korle Bu, Mama of Kumasi and Albert Dompreh of the Serological department of Komfo Anokye Teaching Hospital for their selfless assistance during data collection at KATH. The following matrons also facilitated data collection at KATH; Matilda, Boakye, Lartey and Ansah.

I wish to thank my parents; Mr. Thomas Twenefour and Miss Florence Akoto for their financial support and parental guidance that has been a source of inspiration for my hard work. I

thank my stepmother; Miss Maud Okrah and my siblings; Joyce, Rita, William, Eric and Bernard for providing me with a cosy environment for working.

My appreciation also goes to Miss Vivian Kofie for her spiritual guidance, Miss Valerie Ablordeppey for her warmth, morale support and encouragements, and Dzidzi for her continuously infectious beams.

To my mates; Fidelis, Esther, Godfred, Eric, Grace, Emma, Mawuli, Gloria, Michael, Prudence and Mary, whose hard work have continuously kept some of us on our toes, I say keep up the good work. And to my cronies; Dr. Max, Owura, Armani, Kwame, Lawrenz, Martin, Toli, Ralph, Afila, Glow, Wayo, Napo, Borketey, Yelu and the rest; I say 'comrades, keep on ……'.

I am forever indebted to you all, may God bless you all.

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ABBREVIATIONS AND ACRONYMS

AIDS	Acquired Immune Deficiency Syndrome
ARV	AIDS associated retrovirus
BCM	Body Cell Mass
BMI	Body Mass Index
CDC	Centres for Disease Control and Prevention
ELISA	Enzyme – Linked Immunosorbent Assay
GRID	Gay – Related Immune Deficiency
HIV	Human Immunodeficiency Virus
IFA	Indirect Immunofluorescence Assay
KSOI	Kaposi's Sarcoma and Opportunistic Infections
LAV	Lymphadenopathy – Associated Virus
MTCT	Mother – To – Child – Transmission
NTCA	National Technical Committee on AIDS
PCP	*Pneumocystis Carnia* Pneumonia
PEM	Protein Energy Malnutrition
PGL	Persistent Generalised Lymphadenopathy
PLWHA	People Living With HIV/AIDS
RDA	Recommended Daily Allowance
RIPA	Immunoprecipitation Assay
TLV	T – cell Lymphotropic Virus
UNAIDS	The Joint United Nations Programme on HIV/AIDS
WB	Western Blot (WB)

ABSTRACT

HIV/AIDS is a public health problem that is increasingly having its toll on human resources, particularly given the fact that the most vulnerable age group is the youth. Even though tremendous achievements have been chalked through the use of antiretroviral drugs, these drugs are expensive and unaffordable by PLWHA in Ghana. Good nutrition has therefore been deemed as a complement to anti retroviral drugs for maximum benefit to this group of people. Ironically, most studies into the nutritional aspects of HIV/AIDS have been done in industrialised countries involving subjects with completely different lifestyles, better nutrition and relatively better health care. Extrapolating these findings to the African context could lead to erroneous conclusions. There is, therefore, the need for more research in our part of the world for better policy actions. The objective of this study was to study the association between nutrient intake, body anthropometry and quality of life of HIV/AIDS patients in Ghana. The study involved 281 patients who had been tested and confirmed seropositive for the HIV virus and were attending the Korle Bu and Komfo Anokye Teaching Hospitals. Subjects were grouped into two, based on the stage of their infection; those with HIV but not AIDS as the first group (i.e. non-AIDS group) and those with AIDS as the second group (i.e. AIDS group). Clinical (symptoms), anthropometric (weight and height) and dietary (using 24hr recall method) data were collected simultaneously by the use of semi-structured questionnaires. Quality of life scores were calculated on the basis of the number of clinical symptoms (eleven in all) that a particular subject was free from. On a scale of 0 – 11, the higher the score the better the quality of life. Non-AIDS male subjects had a mean BMI of 23.8 ± 3.2 Kg/m^2, which was significantly higher ($P < 0.0001$) than that of the AIDS male subjects, who had 18.3 ± 2.3 Kg/m^2. Also, non-AIDS female subjects had an average BMI of 23.9 ± 3.4 Kg/m^2, which was significantly higher ($P <$

0.0001) than that of the AIDS female subjects, who had 18.0 ± 2.3 Kg/m^2. There were no significant differences between the BMIs of the two sexes in the same group. The levels of nutrient intakes were better in the non-AIDS subjects than the AIDS subjects. Non-AIDS subjects met the RDA for vitamins A, B_{12} (males alone), C; phosphorus, iron, zinc (males alone), protein and dietary fibre; but not vitamins B_1, B_2, Niacin, B_6, Folate, D and E; minerals Ca, Mg, K, Na, I_2, Se, Cu; as well as energy. AIDS subjects did not meet RDA for any of the nutrients, except the males who met 91.2% of RDA for iron. Energy intake correlated significantly and positively with BMI ($r = 0.687$, $p<0.0001$) and weight ($r = 0.572$, $p<0.0001$), and negatively with the stage of the infection ($r=-0.725$, $p<0.0001$) and avoidance of certain food items ($r=-0.411$, $p<0.0001$). Energy intake also correlated significantly and negatively with occurrence of anorexia ($r=-0.407$, $p<0.0001$), anaemia ($r=-0.395$, $p<0.0001$), diarrhoea ($r=-0.358$, $p<0.0001$), oral lesion ($r=-0.336$, $p<0.0001$), sore throat ($r=-0.286$, $p<0.0001$), tuberculosis ($r=-0.219$, $p<0.0001$), vomiting ($r=-0.206$, $p<0.0001$) and skin rashes ($r=-0.160$, $p=0.007$). Energy intake did not correlate significantly with the intake of multivitamin and mineral supplements, alcohol consumption or smoking. Quality of life correlated significantly and positively with iron ($r=0.547$, $p<0.0001$), protein ($r=0.545$, $p<0.0001$), energy ($r=0.542$, $p<0.0001$), zinc ($r=0.526$, $p<0.0001$), Niacin ($r=0.510$, $p<0.0001$) and all the other nutrients studied except vitamin A. BMI also correlated significantly and positively with protein ($r=0.718$, $p<0.0001$), iron ($r=0.691$, $p<0.0001$), energy ($r=0.687$, $p<0.0001$), zinc ($r=0.682$, $p<0.0001$) and Niacin ($r=0.681$, $p<0.0001$) and all the other nutrients studied. The quality of life of subjects correlated significantly and positively with their BMI and weight. It is recommended that future studies involving PLWHA should include the use of CD4+ lymphocyte counts in HIV/AIDS disease staging.

CHAPTER ONE

INTRODUCTION

1.1 Background Information

The acquired immune deficiency syndrome (AIDS) was first described by the Centres for Disease Control and Prevention (CDC) of the US in June 1981. Since then it has claimed millions of lives and remains a major public health challenge, and has stimulated an unprecedented amount of biomedical research, which has led to a major expansion of knowledge in many aspects of this infection. Many unsolved problems however remain regarding the pathogenesis, management and control of the infection.

The first AIDS case in Ghana was reported in March 1986. This rose to 43,578 as of December 2000 (Yeboah, 2001). By the end of 2001, the number of reported cases in the country had risen to 52,961, which is reported to represent only 30% of the actual number of cases in the country. The Joint United Nations Programme on HIV/AIDS (UNAIDS) estimated that about 28,000 of Ghanaians had died of AIDS by 2001 with about 360,000 more living with the virus as of December 2001. An estimated 24.8 million people worldwide had died of AIDS, including 19.3 million in sub-Saharan Africa and an estimated 40 million more are reported to be infected with HIV worldwide out of which sub-Saharan Africa accounts for 28.1 million (UNAIDS, 2001).

The syndrome has been shown to be due to infection with a retrovirus, the Human Immunodeficiency Virus (HIV) (Gallo *et al.*, 1984). Even though the virus has been isolated in body fluids like tears, sweat, urine and saliva, only blood, semen, vaginal fluid and breast milk have enough concentrations to be infective. Infection by the virus is followed after some months by seroconversion, at which time the infected individual makes specific antibody responses to

viral glycoprotein antigens and this may be accompanied by a glandular fever-like illness and is referred to as stage I disease (CDC, 1986a; CDC, 1987a). Individuals may then remain clinically well for several years following seroconversion; this is termed stage II disease. Stage III disease is characterised by Persistent Generalised Lymphadenopathy (PGL), which may be associated with constitutional symptoms such as fever, night sweats or weight loss. Stage IV disease is defined by the occurrence of an opportunistic infection, an HIV-associated malignancy, or substantial weight loss (CDC, 1986a; CDC, 1987a).

The course of AIDS is often complicated by profound weight loss, cachexia, multiple nutrient deficiencies and particularly, protein energy malnutrition (PEM) (Jasan, 1988). The two major causes of malnutrition and wasting in patients with HIV/AIDS infection are impaired digestion and absorption caused by gastrointestinal pathogens or impaired mucosal function and altered metabolism related to cytokine activity, which is caused by the HIV infection itself and/ or secondary opportunistic infections. Throughout the progression of the disease, the nutritional status of people with AIDS is challenged by malabsorption, diarrhoea, oral/oesophageal problems, nausea, vomiting and infections.

PEM and other nutrient deficiencies are known to be associated with depression in cellular immunity with abnormal T cell and macrophage function (Chandra, 1983). Such changes could compound the immunodeficiency in people with HIV/AIDS, rendering them more susceptible to infections or exacerbating the severity of existing infections. The synergism between PEM and HIV infection on immunocompetence is thought to lead to a more rapid advancement to AIDS (Jain and Chandra, 1984).

Wasting and weight loss are very important features of the disease process to the extent that weight loss is now included in the widely-adopted clinical classification of HIV infection

produced by the CDC (CDC, 1987a). In this classification, weight loss of more than 10% of body weight has been adopted as one major sign for determining whether an HIV positive individual has AIDS or not. The wasting and weight loss that occur during HIV infection are closely associated with disease progression (Hoover *et al.*, 1992) and may be very substantial (Kotler *et al.*, 1985; Kotler *et al.*, 1989). Indeed, timing of death from AIDS correlates well with the decline of nutritional status, particularly the progressive depletion of body cell mass (BCM) (Chlebowski *et al.*, 1989).

Nutritional therapy is therefore an important adjunct in the clinical care of patients infected with HIV. In addition to helping delay the progression of HIV disease to AIDS (Graham *et al.*, 1991; Abrams *et al.*, 1993; Timbo and Tollefson, 1994; Baum *et al.*, 1995; Tang *et al.*, 1997a), achieving and maintaining optimal nutritional health in these patients will optimise existing immune system function (Coodley *et al.*, 1994; Harbige, 1996), reduce the incidence of complications of HIV infection, particularly opportunistic infections (Coodley *et al.*, 1994; Harbige, 1996), reduce the overall cost of medical care (Kotler, 1992), help achieve maximum benefit from drug therapies (Kotler, 1992), and improve the patient's quality of life (Kotler, 1992, Coodley *et al.*, 1994).

Nutritional health affects both cell-mediated (T-cell) and humoral (B-cell) immune function. Specific nutrients are known to influence antibody production, phagocytic cell function, complement levels, and T-lymphocyte function (Dent *et al.*, 1994). Moreover, CD4+ T lymphocytes (helper cells) may be affected by nutrition status to a greater extent than are other types of T cells (Abrams *et al.*, 1993; Baum *et al.*, 1995; Harbige, 1996). By remaining nutritionally healthy, patients with HIV will avoid further compromise to immune functions, which are susceptible to nutrient deficiencies. Specific micronutrients have been associated with

immunocompetence. For instance, vitamin A is known to enhance lymphocyte stimulation response to mitogens, increases natural killer cell activity and reduces binding of bacteria to epithelial cells (Chandra, 1988).

Although there is no cure yet, significant advances have been made using antiretroviral and other therapies, which prolong the survival of many patients. However, these drugs are very expensive and are not affordable to many individuals, especially in developing countries. These, coupled with the fact that most patients need continuous treatment, presumably for life or until the drug is no longer tolerated or effective render sole dependence on drug therapy not the best alternative economically.

The association between malnutrition and negative health outcomes has been extensively studied in many countries around the world. Outcomes of such studies have associated malnutrition with slower wound healing, greater complication rates, higher morbidity and mortality rates, and longer hospital stays, all of which result in higher medical care costs. Because prevention is less expensive than treatment, prevention of malnutrition must be a primary focus of clinical care for the HIV/AIDS patients.

1.2 **Statement of Problem**

HIV/AIDS is a challenge to our livelihood, quality of life and longevity. Presently, various researchers are seriously investigating the causes, management and potential cure for the infection. One special area in this critical endeavour, is the relationship between nutrition and HIV/AIDS.

Malnutrition is a common complication of HIV-infection that is very serious and can even cause death. It was one of the earliest complications of AIDS to be recognised and is one

of the common initial AIDS-defining diagnoses to be reported to public health authorities. Fortunately, studies prove we can improve the nutritional status of the population in general, and HIV/AIDS individuals in particular.

Several studies have shown that HIV positive individuals have an increased likelihood of developing deficiencies of total kilocalories and specific nutrients, including protein, vitamins A, B_6, B_{12}, C, and E, folic acid, magnesium, selenium and zinc (Baum *et al.*, 1994; Coodley, 1995; Chlebowski *et al.*, 1995), and that those with vitamin A deficiency have four- to six-fold greater risk of dying than those who do not (Semba *et al.*, 1994a). However, most of these studies have involved subjects from industrialised countries. These subjects are mostly homosexual men and/ or intravenous users who have access to better health care and good nutrition, therefore extrapolating these research findings to sub-Saharan African populations may lead to erroneous conclusions.

Numerous factors influence human nutritional status. These include sex, race, age, environmental factors, physical activity, the availability and utilisation of food, and others. The need for more research on this deadly disease and the fact that no studies had been done in Ghana on the nutritional status and quality of life of patients with HIV/AIDS are what necessitated this study.

1.3 Objectives

The principal objective of this study was to study the association between nutrient intake, body anthropometry and quality of life of HIV/AIDS patients in Ghana. The specific objectives were to:

1. Quantify daily nutrient intakes from diets by subjects
2. Determine various factors possibly impacting on these intakes and
3. Determine the effects of intakes and anthropometric indices on quality of life

CHAPTER TWO

LITERATURE REVIEW

2.1 Historical Perspective

The Acquired Immune Deficiency Syndrome (AIDS) infection was first recognised in June 1981 as a disease entity, with the report of 26 cases of rare neoplasm Kaposi's Sarcoma and 5 cases of *Pneumocystis Carnia* Pneumonia (PCP) (an unusual opportunistic infection seen only in severely immunocompromised or immunosuppressed patients – for example, chemotherapy patients and those with malnutrition) in previously healthy young homosexual men in the USA (Diamond, 1992). However, the earliest known AIDS-related human death, recognised in retrospect by molecular studies on blood samples occurred in 1951(Diamond, 1992).

Following these reports of PCP and other rare life-threatening opportunistic infections, the CDC formed a Task Force on Kaposi's Sarcoma and Opportunistic Infections (KSOI) (The AIDS Epidemic in San Francisco, 1995). Around this time a number of theories were developed about the possible cause of these opportunistic infections and cancers. Early theories included, infection with cytomegalovirus, the use of amyl nitrite or butyl nitrate "poppers" and "immune overload" (Gottlieb *et al.*, 1981; Goedert *et al.*, 1982; Shearer and Hurtenbach, 1982).

The disease still did not have a name, with different groups referring to it in different ways. The CDC generally referred to it by reference to the diseases that were occurring, for example lymphadenopathy (swollen glands), although on some occasions they referred to it as KSOI, the name already given to the CDC task force (CDC, 1982a; CDC, 1982b). In contrast,

some still linked the disease to its initial occurrence in gay men, with the Lancet calling it the 'gay compromise syndrome', whilst at least one newspaper referred to it as GRID (gay-related immune deficiency) (Brennan and Durack, 1981; Altman, 1982).

In 1982, the name Acquired Immune Deficiency Syndrome or AIDS (an acronym coined by Dr. Bruce Voeller) was given to this clinical syndrome (Time Magazine, August 12, 1985). An anagram of AIDS, SIDA was created for use in French and Spanish (Direction Generale De La Sante, 1982). The doctors deemed 'AIDS' suitable because people acquired the condition rather than inherited it, because it resulted in a deficiency within the immune system, and because it was a syndrome, with a number of manifestations, rather than a single disease (Connor and Kingman, 1988).

By the following year, AIDS definition was established by the United States' Centre for Disease control (CDC) (CDC, 1985; CDC, 1987b). As knowledge of the natural history of HIV increased and in order to remain consistent with the clinical management of AIDS, the AIDS case definition has been expanded thrice; first in 1985 to include other tumours and a wider range of viral, bacterial, fungal and protozoal infections then in 1987 to include encephalophathy and wasting syndrome. The most recent revision of the system published in 1993 emphasised the importance of the CD4+ T-lymphocyte count in the categorisation of HIV-related clinical conditions (CDC, 1993).

Even though earlier observations had suggested that an infectious agent was responsible for the illness, it was not until May 1983, that a French team led by Dr Luc Montagnier of the Pasteur Institute in Paris first published evidence of a new virus that appeared to play a role in the disease AIDS (Time Magazine, August 12, 1985). The following spring, Dr. Robert Gallo of

the National Cancer Institute in Bethesda, USA, announced that he had conclusively identified the virus and produced it in large quantities.

Researchers in various countries gave different names to the virus identified to be responsible for the AIDS infection. Whilst French researchers named the virus lymphadenopathy-associated virus (LAV) (Barre-Sinouss *et al.*, 1983), different researchers in US named it human T-cell lymphotropic virus-III (HTLV-III) (Coffin *et al.*, 1986) and AIDS associated retrovirus (ARV) (Levy *et al.*, 1984). To credit both French and US scientists, many researchers used the term HTLV-III / LAV or LAV / HTLV-III.

In May 1986, the International Committee on the Taxonomy of Viruses recommended a new name for this virus as Human Immunodeficiency Virus (HIV). The committee chose this name because it identifies the group affected; Humans, and it describes the major effect of the virus; Immunodeficiency (Coffin *et al.*, 1986). Two types of HIV have since been isolated from people with AIDS. These are HIV-I found in most parts of the world and HIV-II; confined to West Africa (Diallo, 1992) but now being detected in India (Haslett *et al.*, 1999).

In Ghana, surveillance of AIDS began in 1985 (Asamoah-Odei *et al.*, 1990) following the formation of the National Technical Committee on AIDS (NTCA). The first AIDS case in Ghana was reported in March 1986 (Neequaye *et al.*, 1987).

2.2 Aetiology of AIDS

2.2.1 Virology of HIV

The Human Immunodeficiency Virus (HIV) belongs to the *Lentivirinae* subfamily of the retroviruses, which have an RNA genome (Haslett *et al.*, 1999). Lentiviruses are so called because they produce clinical disease after a long latent period. HIV (being a retrovirus)

contains a reverse transcriptase, an enzyme that transcribes viral RNA into DNA. HIV gains entry to the host cells by binding to the CD4+ receptor using the viral surface membrane glycoprotein gp120. Clouse *et al.* (1989) reported that different isolates of HIV have variable degrees of cytopathicity for CD4+ cells.

There is a huge diversity amongst HIVs, which occur in two main types: HIV-I and HIV-II. Disease caused by HIV-II is similar to disease caused by HIV-I but is generally milder, slower to progress and poorly transmitted vertically (Haslett *et al.*, 1999).

HIV infects CD4+ helper T lymphocytes, which are responsible for initiation of nearly all immunological responses to pathogens. HIV infects mononuclear cells using the *env* glycoprotein. Partial uncoating and reverse transcription of the viral RNA follows this, with subsequent integration of the double-stranded complementary DNA (cDNA) into the host genome. The integrated provirion then serves as a template for the synthesis of viral proteins, which ultimately assemble into progeny virions that are released into the infected host cell by lysis (Strebel and Bour, 1999).

Following infection by HIV, there is a gradual attrition of the CD4+ cell population, resulting in gradual and increasing failure of most aspects of immune function but particularly cell-mediated immunity (Haslett *et al.*, 1999). After a prolonged clinical latency period, the ability to replace destroyed cells is outpaced by ongoing destruction, disrupting communication within the cellular immune system and leading to the characteristic immunodeficiency of AIDS and its opportunistic infections and neoplasm (Klein and Gourevitch, 1998).

The predominant opportunistic infections seen in HIV disease are intracellular parasites (for example *Mycobacterium tuberculosis*) or pathogens susceptible to cell-mediated rather than antibody-mediated immune responses (Haslett *et al.*, 1999). HIV also indirectly infects cells of

the central nervous system. This is probably due to migration of HIV-infected monocytes to the brain, where they become microglial cells, resulting in damage to the central nervous system.

2.2.2 Transmission of HIV

HIV has been isolated from various body fluids. The greatest concentrations have been found in blood, semen and cerebrospinal fluid (Gallo *et al.*, 1984; Groopman *et al.*, 1984; Levy *et al.*, 1985). Lower concentrations have been detected infrequently in tears, saliva, breast milk, urine, and cervical and vaginal secretions. HIV has also been isolated in brain tissue, lymph nodes, bone marrow cells and skin (Levy *et al.*, 1985; Vogt and Awitt, 1985). In spite of all these isolations, it is only blood, semen, vaginal fluids and breast milk that have enough concentrations of HIV to be infective (WHO, 1994).

Sexual transmission has been recognised as the commonest mode of spread of the virus. It has been estimated to account for 75 – 80 % of all HIV infections in Ghana (Napuli, 2001). Even though both homo and heterosexual activities with infected partner have been documented to transmit the virus, whereas the number of cases attributable to heterosexual transmission in the US and Europe is small, it remains the major transmission channel in Africa especially among commercial sex workers and their patrons (Mc Fadden *et al.*, 1986; Redfield and Wright, 1985). Relations with multiple sexual partners increase the chances of encountering someone who is infected, especially in areas where HIV is very common (Blattner *et al.*, 1985; Goedert *et al.*, 1985).

Among sexual practices, receptive anal intercourse with an infected partner is especially likely to lead to infection. The mucosa lining the rectum is delicate and tears easily during anal intercourse. This allows infected lymphocytes and viruses in the semen to enter the tissue and

bloodstream of the receptive partner, whether male or female (Calabrese *et al.*, 1985; Lyman *et al.*, 1986; Nicholson *et al.*, 1985).

Male circumcision has been suggested to be protective against HIV transmission. According to a report by Simonsen (1988) from a Nairobi STD clinic, uncircumcised men were 2.7 times more likely to be infected with HIV than circumcised men were. In a contrasting report from a study by Kelly *et al.* (1999a) in Uganda, it was found that HIV-I prevalence was 14.1% in uncircumcised men compared with 16.9% for men circumcised at 21 years and above. It was 10% for men circumcised at age 13-20 years, and 6.9% in men circumcised at and before 12 years. The conclusion therefore was that prepubertal circumcision after age 20 years was not significantly protective against HIV infection (Kelly *et al.* 1999a).

Transmission of HIV through contaminated blood and blood products has been documented (Curran, 1984). It has been reported that about 2% of AIDS cases in US and about 5% in Europe have occurred among haemophiliacs and others receiving contaminated blood (CDC, 1986b). In some African countries, blood transfusion may be an important means of HIV transmission. Mann *et al.* (1988) reported that in Zaire, Uganda and Rwanda for example, 6% of samples of donated blood contained antibodies of HIV. In Ghana, HIV prevalence among blood donors has been reported to be 3% even though the transmission of contaminated blood and blood products is 5% (Napuli, 2001). Infections through injections can also be acquired, when needles or syringes are contaminated and reused without the necessary sterilisation. This is very common among intravenous drug users.

Mother-to-child-transmission (MTCT), also known as vertical transmission has also been documented. This occurs through three routes; through placental barriers during pregnancy (*in utero;* 21%), during labour and delivery (*intrapatum;* 65%) and through

breastfeeding (*postpartum*; 14%) (Stone-Jimenez, 1999). Blanche *et al.* (1989) reported that one-quarter to one-half of babies born to HIV infected women were infected. It has also been estimated that in Africa, vertical transmission of HIV is about 30 – 50%. In Ghana, the overall MTCT rate is estimated at 15% (Napuli, 2001).

A study done in Europe by Laponite and Michaud (1985) in which 271 children born to infected mothers in eight European centres were followed from birth for an average period of one year revealed that ten of these infants developed AIDS and AIDS related complex by the 9th month, of whom 5 died. Twenty-two others had symptoms and signs that suggested HIV infection. Of this number, 12 developed immunological abnormalities, 9 of which were confirmed infected.

Dunn *et al.* (1992) estimated that breastfeeding approximately doubles the risk of vertical transmission of HIV. Bobat *et al.* (1997) also reported in their work in Durban, South Africa that the highest overall rate of vertical transmission was in exclusively breastfed children (39%) and lowest in never-breastfed infants (24%).

In 1988, Van de Perre *et al.* (1988) reported that they had found HIV antibodies in all milk specimens from four HIV-positive mothers in Rwanda; in three out of the four cases, the babies were also HIV-positive. Ruff (1994) also reported in 1994 that they found HIV DNA in 70% of milk samples from women zero to four days postpartum, and 50% of milk samples collected from women six to eleven months postpartum. This report led to the suggestion that maturity of breast milk influences transmission of the virus.

A year later, Nduati *et al.* (1995) in Kenya, studied 212 breast milk samples, collected at varying times after deliver, from 107 HIV-positive women. They found that at nine months after delivery 58% of samples of breast milk had detectable HIV-I-positive cells. A very significant

finding of this study was that there was a significantly high prevalence of HIV in mature milk than in colostrum and that women with more advanced HIV disease and women with severe vitamin A deficiency were more likely to have HIV in their milk.

2.2.3 Diagnosis of HIV

Diagnosis of HIV infection has been made on serological basis because the majority of the course of infection is clinically undetectable. HIV immunoassays have become progressively more sensitive and specific with the use of recombinant and/ or synthetic peptide antigens as the basis of the test (Best and Dax, 1997). Early assays used antigens prepared in cell culture systems and therefore contained viral and cell-derived proteins that compromised their specificity.

The most specific type of test available (the Western Blot) provided that blots were interpreted using stringent criteria to assure that specificity. However, Western blots provided a series of disadvantages including their high cost, the lack of standardisation in their production and their subjective reading. This led to the World Health Organisation (WHO) Expert Committee on Diagnostics (1992 – 1993) of the WHO former Global Programme on AIDS to discuss and promote the idea of alternative strategies (Tamashiro *et al.*, 1993).

By early 1984, a sensitive and specific blood test had been developed. It was able to detect antibodies to HIV in the serum of infected individuals, and was approved for use in laboratories and blood transfusion centres in the US by January 1985. Presently, standard HIV serological testing involves a rapid screening assay, and a longer confirmatory test that verifies the presence of viral antibodies in reactive samples (Davey and Lane, 1990). In addition to these

conventional assays that test for the presence of antibodies to the virus, there are other assays that test for the presence of the virus or its products in clinical samples.

The main screening assay is the enzyme-linked immunosorbent assay (ELISA), which was developed for the detection of HIV-specific antibodies. For this technique, viral antigens are used to coat the bottom of microtitre wells. Serum from an individual is then added to the well and allowed to incubate for an appropriate length of time to allow binding of antibodies in the serum to the antigen. Unbound components of the incubation mixture are washed away and a second antibody raised against human immunoglobuliin G (IgG) and conjugated to an enzyme is added to the well and incubated to allow binding. Unbound products are washed away and chromogenic substrate is added. The bound enzyme utilises the substrate added, with concurrent development of a colour reaction. The intensity of the colour, measurable with a spectrophotometer, is proportional to the amount of bound enzyme, which in turn is proportional to the amount of HIV-specific antibody that is bound to the HIV antigen on the plate (Schupbach *et al.*, 1984).

Other screening assays for HIV infection that are more rapid and adequate for use in the field or in less developed countries have been described (Auwanit *et al.*, 1990). Unlike ELISA assay, their use does not require expensive laboratory equipment. Some of these rapid, instrument-free screening assays include the rapid latex agglutination assay and the dot-blot immunobinding assay.

Schupbach *et al.* (1984) have reported three main confirmatory assays for HIV. These are Western Blot (WB), radioimmunoprecipitation assay (RIPA) and indirect immunofluorescence assay (IFA). The most popular in the HIV field is the WB technique. For this technique, the patient's serum is reacted with a strip of nitrocellulose containing three major

groups of HIV proteins: the envelope glycoprotein, the gag structural proteins and the *pol* gene products. Antibodies from the patient's serum bind to the HIV proteins. They are detectable on the nitrocellulose after incubating with an anti-human antibody conjugated to an enzyme followed by the addition of the substrate (Schupbach *et al.*, 1984).

The interpretation of the pattern of the reactivity on the WB strips is the difficult part of the assay and has undergone a series of modifications by the CDC and the WHO. These control centres have based their recommendations for definition of a positive WB on the accumulated reactions seen in different geographical areas and in various stages of the disease. The general recommendation is that a positive WB must possess reactions to at least two of the three major bands: gp160 or gp120, gp41, and p24. Blots showing no reaction at all are considered negative while those with fewer reactions than recommended are considered intermediate (Schupbach *et al.*, 1984). The use of the ELISA screening assay along with the WB confirmatory test for determining infection with HIV is now standard practice, and has a positive predictive value of 99.5% in both low-and high-risk populations (Schwartz *et al.*, 1988).

2.3 **Disease Progression**

2.3.1 Immunopathogenesis of HIV

The fundamental abnormality in HIV-infected individuals is the progressive decrease in number of CD4+ lymphocytes (Fahey *et al.*, 1990). Even though the pathogenicity of HIV in infected CD4+ T-lymphocyte has not been fully elucidated, various theories have emerged. Whichever theory is involved, the most fundamental consequence is that HIV infection can lead

to cell death, and immune function decreases as CD4+ counts decline (DeHovitz, 1995). Several mechanisms have been proposed.

HIV can lead to the development of large multinucleated cells called syncytia. Koot *et al.* (1993) has postulated that viral variants, which are more likely to cause syncytia, may be more pathogenic. Another possibility is that HIV itself is directly cytoxic, perhaps through the accumulation of viral gene products. Yet another possibility is that HIV causes a programmed cell death, even in uninfected cells (DeHovitz, 1995).

2.3.2 Laboratory markers for disease progression

Decline in CD4+ lymphocyte counts is the laboratory marker that has emerged as one of the most important predictors of HIV disease progression. Reports have it that the rate of CD4+ cell count decline vary, but averages 40 to 80 cells/mm^3/year (DeHovitz, 1995). However, some patients may have relatively stable CD4+ counts for years, while others may experience a rapid decline over a period of months.

In a cohort of 288 HIV-infected homosexual men followed in San Francisco, progression to AIDS at rates 87%, 46%, and 16% were observed after 3 years of follow-up for patients with CD4+ counts of 200, 201 to 400, and greater than 400, respectively (Moss *et al.*, 1988). Similar results were observed in homosexual men participating in the Multicentre AIDS Cohort Study (MACS). MacDonnell *et al.* (1990a) reported that in the Chicago arm of that study, 87% of men with CD4+ counts of less than 200 cells/mm^3 progressed to AIDS within 4 years compared to only 21% with counts of at least 400 cells/mm^3.

Studies have also shown that a low percentage of CD4+ cells is associated with an increased risk of AIDS and Taylor *et al.* (1989) have suggested that this parameter may be associated with less variability than absolute number of cell. Most clinicians utilise both CD4+ count and percentage in assessing the stage of HIV disease. However, several studies have indicated that the slope of the CD4+ count is the best predictor of rapidly progressing disease and this has led to the suggestion that perhaps more important than the actual CD4+ count or percentage value is the trend of CD4+ cell count over time. In one such study by Burcham *et al.* (1991), an individual who had a decrease in CD4+ percentage value of 7% or more over a 1year period had a relative risk of developing AIDS that was 35 times greater than those who had stable CD4+ cell percentage over the same time period.

Other markers have also been used to monitor disease progression. One such marker is serum HIV p24 antigen level. DeHovitz (1995) reported that high levels of antigens may be seen at the time of primary HIV infection and that these levels decline with the development of immune complexes and serum anti-HIV antibodies. Another marker that has been used is β2-microglobulin level. In the San Francisco General Hospital study, β_2-microglobulin levels greater than 5.0 mg/L had a 3-year progression rate to AIDS of 69% compared with 12% among patients who had levels less than or equal to 3.0 mg/L (Moss *et al.*, 1988). Neopterin levels may be a marker of CD4+ lymphocyte and cellular immune serum activation and in a study by Kramer *et al.* (1989), increased serum and urine levels of neopterin were associated with progression of HIV disease.

Each laboratory marker has independent predictive value in estimating the probability of disease progression. However, Yarchoan *et al.* (1991) studied CD4+ count and the risk for death

in patients infected with HIV and reported that CD4+ count has the strongest correlation with disease progression and fatality.

2.3.3 Disease staging

Prior to the discovery of HIV, patients were diagnosed based on the presence of AIDS-defining opportunistic infections and malignancy. Following the discovery, several staging systems were proposed. Two of these systems; the Walter Reed staging system and the CDC system, were used in clinical settings. Whilst the Walter Reed system relied on skin-testing, the CDC system relied on the presence of HIV-associated diseases (DeHovitz, 1995).

In recognition of the demonstration that CD4+ count is the best predictive marker of the relative risk of developing HIV-related opportunistic infection, the CDC revised their AIDS case definition in 1993 and introduced a clinical staging system that uses CD4+ count values (CDC, 1992). This new definition included all patients previously fulfilling the definition of AIDS, plus all patients with CD4+ counts of less than 200 cells/mm^3. This has led to the categorisation of disease staging into the following based on clinical CD4+ count; acute primary HIV infection (seroconversion), early asymptomatic HIV disease (CD4+ >200 cells/mm^3), early symptomatic (CD4+>200 cells/mm^3), late HIV disease (CD4+ between 50 and 200 cells/mm^3), and advanced HIV disease (CD4+ <50 cells/mm^3).

2.3.3.1 Acute Primary HIV Infection

This stage represents the first two to six weeks after infection and lasts for one to two weeks. There may be no symptoms at this time, however, reports from studies by Cooper *et al.* (1985), and Wallace and Harrison (1988) indicate that between 50 and 90% of individuals develop a non-specific syndrome called the acute retroviral syndrome or acute seroconversion, which is indistinguishable from influenza or infectious mononucleosis.

Acute seroconversion syndrome comes with symptoms including fever, arthralgias, myalgias, and fatigue. A study of the clinical manifestation of acute infection with human immunodeficiency virus in cohort of gay men by Fox *et al.* (1987) reported that physical examination may reveal a diffuse erythematous rash, as well as a diffuse and symmetric adenopathy. Immune deficiency can also be present at this stage of the infection.

Another study by Podzameczer *et al.* (1988) revealed that oral candidiasis, oesophageal candidiasis, and even *Pneumocystis carnia* pneumonia may be present in some patients at this stage. Yet another study by Cooper *et al.* (1988) demonstrated that acute seroconversion is characterised by marked lymphopenia with depletion of both CD4+ and CD8 lymphocytes.

2.3.3.2 Early Asymptomatic HIV Disease

The most common clinical signs of this stage of the disease are dermatologic manifestations such as seborrheic dermatitis or pruritic folliculitis (DeHovitz, 1995), even though mild to moderate lymphadenopathy may be present in some patients. As the

asymptomatic stage of the disease progresses, evidence of immune deficiency correspondingly increases. However, a study by MacDonnell *et al.* (1990b) reported that CD4+ count at this stage is normal (between 700 – 1000 cells/mm^3).

2.3.3.3 Early Symptomatic HIV Disease

Early clinical manifestations of HIV disease are present at this stage. These include a wide range of constitutional symptoms such as headache, fever, fatigue, myalgia, malaise, night sweats, anorexia, diarrhoea, and weight loss (DeHovitz, 1995). Dermatologic manifestations are also present at this phase of the disease. Incidence of oral hairy leukoplasia and other forms of oral manifestation including aphthous ulcers have been reported in about 5% of patients with early HIV infection (DeHovitz, 1995) as well as tuberculosis. Most patients at this stage have CD4+ counts above 200 cells/mm^3

2.3.3.4 Late HIV Disease

Patients at this stage of the disease have CD4+ count between 50 and 200 cells/mm^3 These patients are classified as having an AIDS-defining condition. The major clinical manifestation may include pulmonary tuberculosis, recurrent bacterial infections, or invasive cervical cancer. Individuals at this stage are at risk of developing a wide range of opportunistic infections such as *Pneumocystis carnia* pneumonia, toxoplasmosis, lymphoma, and cryptococcal meningitis (DeHovitz, 1995).

2.3.3.5 Advanced HIV Disease

This stage represents the last stage of the disease and patients at this stage have CD4+ counts below 50 cells/mm^3. These patients are at high risk of dying within a 2-year period. Reports from studies by Drew (1991) and Ellner *et al.* (1991) indicate that these patients are at high risk of developing disseminated *Mycobacterium avium* complex infections or cytomegalovirus (CMV). However, the most pronounced manifestation of the infection at this stage is the HIV wasting syndrome accompanied by diarrhoea with corresponding fat malabsorption.

2.3.4 AIDS definition in Ghana

In October 1998, data on all reported cases of AIDS were reviewed and a new case definition for the country adopted (MOH, 1998). This definition states that; "a person is said to have AIDS when he or she has two of the major signs and one minor sign plus evidence of HIV by the ELISA method, or when he or she has three major signs with or without a minor sign plus evidence of HIV by the ELISA method" These minor and major signs were adopted from the WHO.

In a publication by the MOH in 2000 for the Ghana National Drugs Programme, the minor and major signs for the case definition of AIDS for adults and children were separated. Paediatric or adult AIDS is suspected in an individual presenting with at least two of the following major signs associated with at least one of the following minor signs, in the absence of known cases of immunosuppression such as cancer or severe malnutrition, or other

recognised cases. The individual must also test positive for the HIV antibody. For infants the major signs are; weight loss or abnormally slow growth, chronic diarrhoea for at least one month and prolonged fever for at least one month. The minor signs are; generalised lymphadenopathy, oro-pharyngeal candidaisis, repeated infections (*otitis medis pharyngitis*), persisitent cough, generalised dermatitis, confirmed maternal HIV infection (MOH, 2000).

For adults, the major signs are; weight loss of more than 10% of body weight, chronic diarrhoea for at least one month and prolonged fever for at least one month. The minor signs are; persistent cough for at least one month, generalised pruritic dermatitis, recurrent herpes zoster, oro-pharyngeal candidaisis, chronic progressive and disseminated herpes simplex infection and generalised lymphadenopathy (MOH, 2000). In addition to the above definition, the publication states that the presence of generalised Kaposi's sarcoma or *Cryptococcal meningitis* in an HIV positive patient are sufficient by themselves for the diagnosis of AIDS.

2.4 **Weight Loss**

2.4.1 Patterns of Weight Loss

Weight loss is independently predictive of event-free survival in HIV disease (Moseson *et al.*, 1989; Palenicek *et al.*, 1995; Guenter *et al.*, 1993). It is often multifactorial, even within individual patients, and may occur for completely different reasons in different individuals. Coodley *et al.* (1994) reported that more than 50 percent of patients experience a loss of greater than 10 percent of their usual body weight. HIV-associated cytokine production causes a cascade of secondary effects that perpetuate weight loss via anorexia and hypermetabolism.

Macallan *et al.* (1993) identified two distinctive patterns of weight loss in a cohort of 30 individuals with the HIV infection. Slow progressive weight loss appears to be primarily associated with gastrointestinal disturbance and inadequate caloric intake, while rapid episodic weight loss followed by periods of weight gain is associated with the development and resolution of systemic infections.

Coodley *et al.* (1994) reported that weight loss may be a less predictive indicator of malnutrition than the loss of lean body mass. This is because weight loss as a single assessment factor is not sensitive to changes in body composition related to intracellular and extracellular water volume. According to Kotler *et al.* (1985), significant loss of body cell mass may not be reflected by body weight due to a relative expansion of extracellular water volume. Although periods of episodic weight loss may be followed by weight gain, loss of body cell mass is typically not recovered.

The timing of death due to wasting or loss of body cell mass is related to the extent of depletion rather than to a specific cause (Harbige, 1996; Kotler, 1994). With greater degrees of wasting, survival time is shortened (Sttmann *et al.*, 1995). Studies indicate that a body cell mass that is 54 percent of normal is correlated with death in HIV disease. This corresponds to a body weight of 66 percent of ideal (Grunfeld and Kotler, 1991). Schwenk *et al.* (1993) identified anorexia, infections and diarrhoea as risk factors having the strongest predictive correlation for loss of body weight and body cell mass. Various studies have recommended that anthropometric measurements and bioelectrical impedance analysis should be routinely performed to accurately assess changes in body weight and body cell mass in patients with HIV disease (Kotler *et al.*, 1996; Ott *et al.*, 1995; Parisien *et al.*, 1993).

Due to altered metabolism in HIV disease, therapies that increase energy intake may not always promote the restoration of body cell mass in patients experiencing wasting (Mulligan *et al.*, 1993). The use of anabolic agents such as testosterone, steroids, and human growth hormone have successfully reversed the wasting process by promoting weight gain, nitrogen retention, and protein-sparing lipid oxidation (Mulligan *et al.*, 1993; Hengge *et al.*, 1996). Hengge *et al.* (1996) reported that the testosterone derivative oxymetholone blocks the effects of the cytokine TNF-α, and is associated with an average weight gain of 8.2 kg over 30 weeks in cachectic patients with HIV disease. Likewise, human growth hormone promotes weight gain, nitrogen retention, and the oxidation of fatty acids rather than protein for energy in patients with HIV disease (Mulligan *et al.*, 1993).

2.4.2 The HIV wasting syndrome

The HIV wasting syndrome is a common complication that can occur in all stages of HIV disease. The hallmark is weakness and progressive weight loss with depletion of body cell mass and relative sparing of body fat. As a diagnostic criterion of AIDS, wasting syndrome is defined as an unintentional weight loss of 10 percent or more of usual body weight, in the presence of diarrhoea, weakness, or fever for more than 30 days, that is not attributable to other disease processes (CDC, 1992). Symptoms of HIV wasting may also be caused by opportunistic infections, commonly due to cytomegalovirus or *Mycobacterium avium* complex. Hence, when symptoms of HIV wasting are present, it is very important to rule out an underlying infection as the cause.

The metabolic aberrations associated with HIV wasting are quite different from those seen in PEM, although the term malnutrition encompasses both processes. PEM develops due to the absence of or a reduction in nutrient availability. Common causes of PEM include caloric intakes below total caloric needs or nutrient malabsorption. Persons suffering from PEM, regardless of the cause, can call on adaptive responses to meet their energy needs. These responses include decreased metabolic rate, reduced production of metabolically active cytokines, and mobilization and oxidation of fat stores to meet energy needs. Hence, weight loss during PEM is minimized and primarily reflects loss of fat stores rather than catabolism of lean body mass. When patients suffering from PEM are provided with adequate nutrition, the adaptive mechanisms of starvation will reverse; anabolism will prevail and body stores will be repleted.

Conversely, the pathophysiologic mechanism of HIV wasting syndrome is thought to be a multifactorial process incorporating inadequate caloric intake, malabsorptive phenomena, metabolic derangements, and cytokine activity (Weinroth *et al.*, 1995). Cytokines alter the body's metabolic priorities and are associated with an increased metabolic rate, futile energy cycles, and catabolism of lean body mass to meet energy needs. This type of malnutrition is associated with a diminished capacity to use body fat for energy, disproportionate losses of lean body mass relative to those of body fat, and relative preservation of extracellular body water. In the presence of cytokines, wasting of lean body mass cannot be overcome simply by increasing nutrient intake, because repletion of lean body mass will not occur until the underlying stimulus driving the catabolic response is addressed and removed (Weinroth *et al.*, 1995).

Data from a study by Macallan *et al.* (1995) indicate that during episodes of rapid weight loss, patients with HIV disease actually have lower total energy expenditure, suggesting a reduced caloric intake (not elevated energy expenditure) as the primary weight loss determinant. However, during periods of reduced energy intake, patients with HIV disease cannot efficiently use fat stores for energy, and thereby continue to break down lean body mass (muscle protein) to supply energy needs (Keusch and Thea, 1993). This leads to a rapid loss of body cell mass, which is a strong predictor of survival. Current treatment for patients experiencing HIV wasting includes enteral or parenteral nutrition support; appetite stimulants such as dronabinol or megestrol acetate; anabolic agents such as testosterone, steroids, or recombinant growth hormone (Serostim); and cytokine modulators (pentoxifylline; thalidomide) (Weinroth *et al.*, 1995).

2.5 **Nutritional Aspects of HIV/AIDS**

2.5.1 Malnutrition and HIV/AIDS

The relationship between HIV/AIDS and malnutrition is a typical example of the "vicious cycle" of immune dysfunction, infectious disease and malnutrition. Changes in the immune function due to malnutrition are similar to those induced by HIV/AIDS. Recent research, much of it conducted in Africa, has shown that nutritional status may affect the progression of HIV disease in adults and the survival of HIV-infected individuals.

Long before the AIDS epidemic emerged in Africa in the early 1980s, the synergistic interactions between infection, nutritional status, and immune function were recognised. Scrimshaw and SanGiovanni (1997) reported that infectious diseases, no matter how mild,

influence nutritional status, and conversely, almost any nutrient deficiency, if sufficiently severe, will impair resistance to infection.

Infections affect nutritional status by reducing dietary intake and nutrient absorption, and by increasing the utilization and excretion of protein and micronutrients as the body mounts its "acute phase response" to invading pathogens. Anorexia, fever and catabolism of muscle tissue frequently accompany the acute phase response. Infections also result in the release of pro-oxidant cytokines and other reactive oxygen species. This leads to the increased utilization of "antioxidant" vitamins (e.g. vitamin E, vitamin C, beta-carotene) as well as the sequestration of several minerals (e.g. iron, zinc, selenium, manganese, copper) that are used to form antioxidant enzymes (Friis and Michaelson, 1998). Schwarz (1996) reported that "oxidative stress" occurs when there is an imbalance between the pro-oxidants and antioxidants, causing further damage to cells, proteins, and enzymes.

The relationship between HIV and nutrition may be more complicated than the relationship between nutrition and other infectious diseases because the virus directly attacks and destroys the cells of the immune system. Nutritional deficiencies affect immune function in ways that may influence viral expression and replication, further affecting HIV disease progression and mortality. Oxidative stress, for example, has been reported in a study by Semba and Tang (1999), to potently hasten HIV replication indirectly. HIV infection affects the production of hormones such as glucagon, insulin, epinephrine, and cortisol, which are involved in metabolism of carbohydrates, proteins, and fats. Elevated levels of these hormones, as reported by Young (1997), contribute to weight loss and wasting syndrome seen in most adults AIDS patients.

A study by Babamento and Kotler (1997), on malnutrition in HIV infection has revealed that weight loss is often the event that begins "a vicious cycle of increased fatigue and decreased physical activity, including the inability to prepare and consume food". In the same study, it was reported that wasting is preceded by changes in appetite, repeated infection, weight fluctuations and subtler changes in body composition, such as changes in lean body mass (LBM) and body cell mass (BCM).

Reduction in food intake has been reported in a study of wasting in HIV infection and AIDS by Macallan (1999), to be the most important cause of the slow and progressive weight loss experienced by people living with HIV/AIDS (PLWHA). Reduction in food intake may be due to painful sores in the mouth, pharynx, and/ or oesophagus. Side effects from medications, including nausea, vomiting, metallic taste, diarrhoea, abdominal cramps, and anorexia also result in lower dietary intakes. Nutrient malabsorption and metabolic alterations have also been reported as being responsible for weight loss and wasting in PLWHA. Keating *et al.* (1995) reported that some HIV-infected individuals have increase intestinal permeability and other intestinal defects. Also works from Ullrich *et al.* (1989), and Babamento and Kotler (1997), have reported that HIV infection itself, particularly of the intestinal cells may cause epithelial damage and nutrient malabsorption. Semba and Tang (1999) also reported that malabsorption of fats and carbohydrates is common at all stages of HIV infection in adults and children.

Macallan (1999) reported that infection results in increased energy and protein requirements as well as in inefficient utilisation and loss of nutrients and these could also account for weight lost and wasting in PLWHA. Findings from various studies suggest that, infections result in a loss of 0.6 to 1.2 g of protein per kg body weight per day in adults when amino acids are metabolised from skeletal muscle in response to the release of cytokines.

Losses have been reported to be highest with diarrhoea, dysentery, and other infections. For these reasons, protein requirements are substantially higher in HIV-infected individuals and persons suffering from chronic immune system stimulation. Furthermore, the energy requirement to replace lost protein is about 6 – 8 kcal/g of protein produced (Scrimshaw and SanGiovanni, 1997).

Findings from Babamento and Kotler (1997) indicate that wasting also results through a process called cachexia, which is characterised by a significant loss of lean body mass resulting from metabolic changes that occur during the acute phase response to infection. It revealed that during the acute phase response, the liver produces large amounts of specific proteins to bind and clear infectious agents. These proteins are reported to be from skeletal muscle; so if the response, which is induced by immune-system cytokines, is prolonged, muscle wasting becomes severe.

2.5.2 Nutritional Management of Weight Loss and Wasting

Management of weight loss in PLWHA is complicated by the fact that mechanisms for weight loss and wasting are not mutually exclusive. Weight loss and wasting due to malnutrition can be reduced by treating the immediate sources of the problem (e.g. oral thrush, mouth sores, other infections), providing preferred foods that are soft and well tolerated by the infected person, and increasing intake during periods of recuperation from acute infection (Cimoch, 1997). In contrast, weight loss and wasting due to metabolic changes cannot be reversed by feeding alone. Such programs tend to increase body fat but rarely improve body cell mass or increase protein stores of wasted muscle (Cimoch, 1997; Macallan, 1999).

Muscle wasting in industrialised countries has been tackled with relatively expensive appetite stimulants and hormones such as testosterone and recombinant growth hormone. Shabert *et al.* (1999) undertook a randomised trial in the USA with the use of a less expensive supplement. This supplement contains glutamine (40g/day) as the main amino acid together with antioxidants such as vitamins C and E, beta-carotene, selenium and N-acetyl cystine at recommended daily allowance (RDA) levels. In the trial, the supplement was given to 12 HIV-infected men and women, who had already experienced significant weight loss, for 12 weeks. Nine subjects of similar characteristics received a placebo supplement. All subjects in the trial received nutritional counselling. After the 12 week period, the subjects of the supplement group gained and maintained significantly more body weight than the control subjects (2.2kg), including 1.8kg in BCM. This compared with a change in BCM of only 0.4kg in the control subjects ($p<0.007$). It was therefore concluded that, nutritional supplementations including the amino acid glutamine, can restore BCM in HIV-infected persons already experiencing weight loss and muscle-wasting (Shabert *et al.*, 1999).

In another study, Hellerstein *et al.* (1996) gave a daily dietary fish oil supplement (18g/day) to 16 HIV-infected men for 10 weeks and monitored weight gain. An unsupplemented controlled group was also followed. Fish oil supplementation resulted in weight gain, but only among patients who did not develop new AIDS complications. This led to the conclusion that fish oil supplementation may benefit some people with AIDS but may not overcome the metabolic consequences of acute infections in others (Hellerstein *et al.*, 1996). Omega-3 fatty acid common in fish oil is required for the body to respond to inflammation and to reduce the impact of cytokines that promote wasting (e.g. inteleukin-1 and tumor necrosis factor) (Piwoz and Preble, 2000), and this may be responsible for the weight gain in the supplemented group.

Stack *et al.* (1996) also found that weight gain and/or weight maintenance could be achieved among asymptomatic HIV-infected individuals, and among HIV-positive people in the early stages of AIDS with no secondary infectious, who received at least one daily, high-energy, high-protein, liquid food supplementation along with counselling. Nutritional counselling involved recommendation of a high protein diet and foods that minimise gastrointestinal complications.

2.5.3 Micronutrients and HIV/AIDS

The role of micronutrients in other infectious diseases, such as measles, diarrhoea, and respiratory infections has been extensively studied and it is known that several vitamins and minerals are required by the immune system and major organs to fight infectious pathogens. Individuals with inadequate intakes, blood levels, or body stores of these micronutrients have difficulty resisting infection. As a result, the possible role of micronutrient in HIV/AIDS takes on special importance in individuals and populations with marginal or low nutrient intakes (Friis and Michaelson, 1998).

Studies from both industrialised and developing countries have confirmed that HIV-infected individuals have decreased absorption, excessive urinary losses, and low blood concentrations of vitamins A, B_1, B_2, B_6, B_{12}, C, E, as well as folate, beta-carotene, selenium, zinc, and magnesium (Friis and Michaelson, 1998; Tang and Smit, 1998). These studies have concluded that micronutrient deficiencies associated with HIV vary across populations and according to disease stage; are associated with an accelerated progression of HIV infection to AIDS; and are predictive of AIDS-related mortality.

2.5.3.1 Vitamin A

Of all the micronutrients, the possible role of vitamin A in HIV infection has received the greatest attention in Africa. This is in view of it well-known role in affecting child morbidity and mortality, as well as early observations that vitamin A status was associated with increased risks of MTCT of HIV (Semba *et al.*, 1994). Also vitamin A status has been associated with HIV viral load in breast milk and vaginal secretions (Nduati *et al.*, 1995; John *et al.*, 1997), progression to AIDS (Tang *et al.*, 1993), adult survival (Semba *et al.*, 1994) and infant morbidity (Coutsoudis *et al.*, 1995) and mortality (Dushimimana *et al.*, 1992). The potential for vitamin A supplementation to positively impact the course of HIV/AIDS was promising to pursue since vitamin A is beneficial in HIV-negative populations, is inexpensive, and relatively easy to administer with minimal side effects.

Semba *et al.* (1994a) undertook a nested case-control study of vitamin A deficiency and wasting as risk factors for mortality from AIDS and infections within a large prospective cohort of HIV infected injection drug users (IDUs). In their study, 50 adult subjects who died from AIDS and infections were matched with 235 controls who survived. They measured plasma vitamin A, weight, and body mass index and followed up the subjects for an average duration of 2.4 years. They reported that vitamin A deficiency occurred in 50% and wasting occurred in 38% of patients in the last visit before death. Also CD4+ cells count less than 200/microL, wasting, and vitamin A deficiency were associated with mortality. There was a higher risk of death in HIV-infected subjects with vitamin A deficiency (odds ratio [OR], 4.6; 95% confidence interval [CI], 1.8-11.3) and wasting (OR, 8.8; 95% CI, 2.7-28.2). These findings led the authors to conclude that vitamin A deficiency and wasting are common during HIV infection and are independent predictors of mortality in HIV-infected IDUs.

Semba (1997) reported that vitamin A deficiency may be caused by insufficient dietary intake of vitamin A-rich food, malabsorption, impaired storage, and/or increased utilisation or urinary losses of vitamin A during acute and chronic infection. Vitamin A deficiency causes anaemia, growth retardation and xerophthalmia; it increases the incidence and/or severity of many infections (including diarrhoea, pneumonia, measles, other respiratory infectious). While vitamin A deficiency is rare among HIV-negative adults in industrialised countries, up to one-third of HIV-positive adults in industrialised countries may be vitamin A deficient. A study by Nimmagadda *et al.* (1998) estimated that up to 60 percent of HIV infected pregnant women in developing countries suffer from vitamin A deficiently.

A lot of researchers have delved into the possible impact of vitamin A supplementation on various HIV-related outcomes in children and adults. A study by Fawzi *et al.* (1999) in Tanzania revealed that vitamin A supplementation reduced all-cause mortality by 63 percent among HIV-infected children aged 6months to 5years, and was associated with a 68 percent reduction in AIDS-related deaths and 92 percent in diarrhoea-related deaths. Prior to this study, Coutsoudis *et al.* (1995) had reported from South Africa that vitamin A supplementation of HIV-infected children reduced diarrhoea morbidity by about 50 percent.

Results from most studies of vitamin A supplementation on HIV-related outcomes in adults have yielded no significance. In a study of vitamin A deficiency and HIV-viral load in injection drug users in USA by Semba *et al.* (1998), it was found that supplementation with vitamin A and /or beta-carotene had no significant or prolonged impact on HIV viral load. Studies by both Coodley *et al.* (1996), and Fawzi *et al.* (1998) found no significant impact of such supplementation on the immune status of HIV-infected adults. In another study, Kelly *et al.* (1999) failed to establish a prolonged impact of such supplementation on diarrhoea

morbidity in HIV-infected adults in Zambia. Likewise, Kennedy *et al* (2000) found no significant impact of vitamin A supplementation on prenatal and postnatal morbidity in HIV-infected women.

These findings make it difficult for the pursuit into the potential impact of vitamin A supplementation on HIV-related outcomes in adults. In actual fact a study by Tang *et al.* (1996) found that very high intakes of vitamin A (i.e. >20,000 IU / day) were actually associated with increased mortality. An earlier study by Tang *et al.* (1993) reported the relation between total vitamin A intake and progression to AIDS to be U-shaped; the lowest and highest quartiles of intake did poorly, while the middle two quartiles were associated with significantly slower progression to AIDS.

2.5.3.2 Vitamin B_{12}

Vitamin B_{12} deficiency is associated with neurological abnormalities, impaired cognition, reduced CD4+ T-cell counts, increased bone marrow toxicity associated with the use of ziduvodine (ZDV), an ARV drug. This is relatively uncommon in healthy, non-vegetarian populations. However, studies by many researchers on people with HIV in USA have reported low serum B_{12} levels even among asymptomatic persons.

In people with HIV, Tang and Smit (1998) have reported that low serum vitamin B_{12} levels could be associated with increased mortality. A 9year study by Tang *et al.* (1997a) among homosexual and bisexual men with HIV/AIDS in USA found that men with low serum B_{12} at enrolment (i.e. <120 pmol/L) had significantly shorter AIDS-free survival times than men with adequate B_{12}, after taking into account HIV-related symptoms, CD4+ cell count, age, ARV use, and other potentially confounding variables.

2.5.3.3 Folate

The role of folic acid in HIV/AIDS remains unclear. This vitamin is required for the enzyme that produces DNA for replicating and growing cells, including those of the gastrointestinal tract (GI), blood and growing foetus. Deficiency therefore results in impaired cell division and protein synthesis, causing megaloblastic anaemia.

To date, no study has shown a relationship between folate deficiency and HIV-related outcomes. Low serum folate (measured once at enrolment) was associated with HIV/AIDS progression and CD4+ cell decline in a study by Tang *et al.* (1997a). However, only 8 percent of the subjects were initially folate deficient (i.e. < 3.4 nmol/L) and more than 50 percent of the subjects took B-complex vitamin supplements during the study. A study in Malawi by Van den Broek *et al* (1998) found that red blood cell folate levels were not significantly different among asymptomatic, HIV-infected, anaemic, pregnant women and anaemic women who were not HIV-infected in Malawi.

2.5.3.4 Vitamin E

This vitamin is necessary for the proper functioning of the immune system and it increases humoral and cell-mediated immune responses, including antibody production, phagocytic and lymphocytic responses, and resistance to viral and infectious diseases (Odeleye and Watson, 1991). The oxidative stress created by HIV and related opportunistic infections increases the utilisation of antioxidant vitamin E, possibly leading to deficiency. Vitamin E deficiency, in turn, further weakens the immune system because of its role in immune stimulation and functioning, leaving PLWHA more susceptible to opportunistic infections (Piwoz and Peble, 2000).

A study by Tang *et al.* (1997b) in the USA found that high baseline serum vitamin E levels were associated with decreased HIV progression, after taking into account HIV-related symptoms, CD4+ cell count, age, ARV use, and other potentially confounding variables. It was found that men with serum vitamin E levels greater than or equal to 23.5mmol/L lived 34 percent longer until first AIDS diagnosis compared with men with low serum vitamin E levels (Tang *et al.*, 1997b).

In another study by Allard *et al.* (1998) in Canada, it was found that 3months supplementation with vitamin E (800IU) and vitamin C (1000mg) significantly reduced oxidative stress and HIV viral load. However, in a study by Kelly *et al.* (1999) in Zambia, oral supplementation with vitamin E and other nutrients did not affect either mortality or diarrhoea morbidity, possibly because of severe fat malabsorption accompanying the late-stage disease.

2.5.3.5 Selenium

Selenium deficiency impairs the immune system and has been associated with faster HIV disease progression and reduced survival in adults (Baum and Shor Posner, 1998) and children (Campa *et al.*, 1999). Selenium is believed to play an important role in metabolising reactive oxygen species ("free radicals") and reducing oxidative stress because it is an essential cofactor for glutathione peroxidase, an antioxidant enzyme. In animal models, Beck (2000) has shown that selenium deficiency increases viral pathogenicity. Friis and Michaelsen (1998) reported that selenium deficiency may contribute to HIV replication and possibly also to increasing the infectiousness of the virus.

Selenium status of individuals is rarely measured outside of specialised chemical studies. One study in USA found that selenium deficiency (measured over time) was

significantly associated with HIV-related death, after taking into account CD4+ cell counts at baseline and over time, use of ARV drugs, and other nutritional deficiencies (protein, zinc, and vitamins A, B6, B12, and E). A report from that study indicates that selenium deficiency was the only nutritional deficiency that was independently predictive of survival after controlling for these other nutrients (Baum *et al.*, 1997).

Delmas – Beauvieux *et al.* (1996) studied the effect of selenium supplementation. They provided oral selenium (100micrograms/day), beta-carotene (60mg/day), or placebo for one year to 45 selenium and vitamin A deficient HIV-infected subjects in France. Selenium increased antioxidant enzyme functions significantly and the effects were greater than those observed with beta-carotene supplementation. These findings suggest that selenium (and beta-carotene) supplementation may reduce the impact of oxidative stress on HIV disease.

2.5.3.6 Zinc

Zinc is an essential component of the immune system and it is important for the development of non-specific and cell-mediated immunity (particularly CD4+ cells). Zinc is also required for the gene expression and the normal development of many tissues and cells. It is also an important component of many proteins, hormones, and enzymes. Zinc deficiency, therefore results in severe immune depression, frequent diarrhoea and other infections, as well as mental disturbances (Piwoz and Preble, 2000).

HIV requires zinc for gene expression, replication and integration (Baum *et al.*, 2000). Thus, persons with HIV may have low plasma zinc levels yet higher zinc intakes may be associated with faster HIV replication and disease progression. Higher intakes of zinc (in the absence of HIV) may interfere with copper and iron utilisation, and very high intakes (more

then 20 times the RDA) have produced significant immune system impairment in healthy adults (Chandra, 1997). Zinc also inhibits tumour necrosis factor (TNF), a cytokine that is important in triggering the process of wasting in HIV. Zinc deficiency, on the other hand, affects T-cell activity, including the secretion of numerous cytokines that affect HIV disease progression (Baum *et al.*, 2000).

Baum *et al.* (1997), have found that low serum zinc levels are associated with HIV disease progression and mortality in adult intravenous drug users in USA. On the other hand, Tang *et al.* (1996) have found that high intakes of zinc (from diet and supplements) are associated with reduced survival in HIV-infected men. Men consuming 14 – 20 mg/day had a relative mortality risk of 1.73 during follow-up, and those with intakes greater than 20mg/day had a relative mortality rate of 1.91, after adjusting for CD4+ cell counts, age, use of ARV, total energy intake, and presence of disease symptoms (Tang *et al.*, 1996).

Among HIV/AIDS patients, zinc supplementation was shown substantial benefits. In a study in Italy, the diets of AIDS patients were supplemented with 200mg of zinc daily. After 1month follow-up, it was realised that the incidence of opportunistic infections (particularly *pneumocystis carinii* and *candida*) had been reduced. Also the supplementation had stabilised weight and improved CD4+ cell counts among adults with AIDS who were receiving ARV therapy as compared with controls who received ARVs but no zinc supplement (Mocchegiani *et al.*, 1995; Mocchegiani and Muzziola, 2000).

2.5.3.7 Iron

Iron is required to produce new cells, amino acids and hormones. Iron deficiency results in anaemia, which is a common problem among PLWHA. It affects asymptomatic HIV-infected

adults and children, as well as people with AIDS. Even though the causes of anaemia associated with HIV/AIDS are not well understood, various theories have been propounded. Anaemia may result from cytokine-induced suppression of red blood cells production; chronic inflammation; opportunistic infections; and/or reductions in dietary intake (including iron), absorption, and retention. Anaemia may also be caused by certain ARV drugs (e.g. ZDV, which suppresses bone marrow function and synthesis of red blood cells), as well as by nutritional deficiencies of iron, folate, riboflavin, vitamin A, and vitamin B_{12} (Piwoz and Preble, 2000).

Anaemia has been found by various researchers to be associated with HIV disease progression and a two to four-fold increased risk of death in HIV-infected individuals (Piwoz and Preble, 2000). Moore *et al.* (1998) investigated anaemia and survival in HIV infection and found that the risk of death in HIV-infected individuals increases with the severity of the anaemia. In a longitudinal study in Europe found that risk of death increased in HIV-infected individual increases with the severity of the anaemia. In a longitudinal study in Europe, it was revealed that risk of death increases by 57 percent for each 1g/dl drop in haemoglobin in HIV-infected subjects, after controlling for CD4+ cell counts, viral load, and use of ARV drugs (Mocroft *et al.*, 1999). Sullivan *et al.* (1998) suggested that reversing anaemia could slow HIV disease progression and prolong survival.

Anaemia appears to be more common among HIV-infected pregnant women than the general antenatal population. In a study in Malawi, Van den Broek *et al.* (1998) found that asymptomatic HIV-infected women were twice as likely to be anaemic as uninfected women, after taking into consideration folate, B_{12}, and serum retinol levels and coexisting inflammation. In another study in Burkina Faso, Meda *et al.* (1999) reported that HIV-infected pregnant women were more likely to be anaemic (78 percent) than uninfected pregnant women (64

percent; $p<0.001$). One major flaw common to these two studies, however, was the fact that researchers in both studies did not measure malaria parasitemia, which is a common cause of anaemia at least among first time mothers in Africa.

Although anaemia is common among PLWHA, advanced HIV disease may also be characterised by increases in iron stores in bone marrow, muscle, liver and other cells (De Monye *et al.*, 1999). This accumulation of iron is likely to be due to the body's attempts to withhold iron from the plasma, although other factors (e.g. ZDV use, cigarette smoking and blood transfusion) may play a role (Boelaert *et al.*, 1996).

CHAPTER THREE

METHODOLOGY

3.1 **Study Design**: The design of this study was a hospital based cross-sectional one. Information on nutrient intake, body anthropometry and quality of life were collected simultaneously.

3.2 **Subjects**: The study involved 281 HIV positive patients who were attending the Korle Bu and Komfo Anokye Teaching Hospitals. Out of this number, 180 were AIDS-free whilst 101 had AIDS. The inclusion criteria were that a subject must have been tested seropositive for the HIV virus, and that the subject should be able to stand on the weighing scale and the platform of the stadiometer unsupported.

3.3 **Study Area**: The study was conducted in the two hospitals above. These hospitals were selected because they are the two major referral hospitals in Ghana. Also, they are the only two teaching hospitals in Ghana and are better placed for academic researches such as this. In Korle Bu, subjects were recruited from the Fevers unit, which is a special unit for HIV/AIDS patients and other patients who need to be isolated. Most of the subjects recruited from this unit were outpatients most of whom belonged to the Wisdom Association; a non-profit association for PLWHA. In Komfo Anokye, subjects were recruited from the following wards; C6, D1, D3 and the counselling unit.

3.4 **Data Collection**: Semi-structured questionnaires (Appendix L) were used to collect information on the following:

a. Socio-economic and lifestyle data: Information was collected on sex, age, tribe, educational background, occupation, alcohol consumption, smoking, multivitamin and mineral supplement use and other socio-economic indicators.

b. Clinical data: Each patient's hospital file was examined for information on clinical symptoms presented that day, a proof of HIV status and whether patient has AIDS or not. Quality of life of each subject was computed based on the number of clinical symptoms that were not presented by that particular subject. Each subject scores one unit for each clinical symptom absent and zero unit for each symptom present. In all, there were eleven clinical symptoms, so the quality of life scale ranged from 0 – 11; the higher the score the better one's quality of life.

c. Anthropometric data: Weight and height of each subject were measured.

Weight – A Salter weighing scale, weighing to the nearest 0.5kg was used to take each subject's weight. Subjects were weighed without their shoes. Other heavy things like mobile phones, keys etc were removed from subjects' pocket before weighing. Subject was then asked to stand upright on the scale for weighing.

Height – A Seca stadiometer, which measures to the nearest 0.1cm, was used to take the height of each subject. Subjects were measured without their shoes. The height of each subject was taken, with subject standing straight on the horizontal platform of the stadiometer with heels together, shoulders relaxed, arms at the sides and head in a horizontal plane. The sliding

headpiece of the stadiometer was then moved to rest on the crown of the subject's head. Reading was then made.

BMI for each subject was computed using the Weight and Height measurements. For each subject, BMI was computed using the formula;

$$BMI = \frac{\text{Weight (kg)}}{\text{Height (m)}^2}$$

d. Dietary data: The 24hr recall method was used to take information on daily food consumption. Subjects were asked to recall all foods and beverages consumed for a day. This was repeated for two other days for each subject, including a day on a weekend. Subjects were asked to estimate the quantity of each food item consumed using household measures. An FPRO software program was used to compute the amounts of nutrients in each food taken by each subject. In some instances, when a specific food is not listed in the program, a food composition table was used to compute the amounts of nutrients.

3.5 **Data Entry and Analysis**: Data entry was done using the EPI INFO version 6 software. After this data were exported to SPSS version 10.0 statistical software program for analysis. Data analysis included descriptive statistics, correlations and analysis of variance. Statistical significance was fixed at the 0.01 level for a wider confidence interval and a narrow margin of error, thereby increasing the reliability of the results.

3.6 **Quality Control**: In undertaking very important and delicate studies such as this, there was an urgent need for quality control measures to ensure reliability of findings. Firstly, it was

ensured that all patients had been tested and confirmed as seropositive for the Human Immunodeficiency Virus. This was done by verifying each subject's hospital file to confirm the existence of laboratory test results as proof of HIV status. Secondly, on each subject's hospital file, characterisation of the disease stage had been done by qualified Medical Doctors, so there were no disputes as to whether a particular subject belonged to the AIDS or non-AIDS group. It was also ensured that all anthropometric measurements were done in duplicate. Data entries were cross-checked to ensure consistency.

CHAPTER FOUR

RESULTS

4.1 Characteristics of Subjects.

In all, 281 HIV positive subjects comprising of 180 non-AIDS and 101 with AIDS were involved in the study. Out of the 180 non-AIDS subjects, 82 were males whilst 98 were females. For the 101 AIDS patients, 58 were males with 43 being females. In totality, 140 (representing 49.8%) of the subjects were males whilst 141 (representing 50.2%) were females. In hospital specifics, 169 subjects (i.e. 130 non-AIDS and 39 AIDS) from the Korle Bu Teaching Hospital were involved in the study as against 112 subjects (i.e. 50 non-AIDS and 62 AIDS) from the Komfo Anokye Teaching Hospital. The distribution of subjects with respect to AIDS status, gender and hospital is provided in Table 1 (below).

Table 1: Sex distribution of subjects studied

	HIV WITH NO AIDS[1]			HIV WITH AIDS[1]			TOTAL
	Males	Females	Total	Males	Females	Total	
Accra	55	75	**130**	26	13	**39**	**169**
Kumasi	27	23	**50**	32	30	**62**	**112**
TOTAL	**82**	**98**	**180**	**58**	**43**	**101**	**281**

[1] Confirmed by qualified Medical Officers

The average age of the subjects studied was 38.3 ± 10.7 years, ranging from 18 to 67 years. Of the 281 subjects studied, 81 (28.8%) were single, 96 (34.2%) were still married, 59 (21.0%) were widowed and 45 (16.0%) of them had either divorced or separated from their spouses. In terms of religion, 201 (71.5%) of the study subjects were Christians, 60 (21.4%) were Muslims, 5 (1.8%) were Traditionalists and 15 (5.3%) reportedly had no specific religion. Concerning the tribes of the subjects, 135 (48.0%) of them were Akans, 50 (17.8%) of them were Gas, 46 (16.4%) of them were Ewes, 42 (14.9%) of them were Northerners and 7 (2.5%) of them were Krobos. There was one Togolese non-AIDS subject who was classified as a foreigner. For educational level, 29 (10.3%) of the subjects had only primary education, 99 (35.2%) of them had only middle/J.S.S education, 104 (37.0%) had only secondary education and only 9 (3.2%) had tertiary education. However, 40 (14.2%) of the subjects had had no formal education at all. The distribution of subjects in terms of marital status, tribe and educational level within the two AIDS status groups are provided in Table 2 (below).

Table 2: Background information on subjects

		HIV WITH NO AIDS (N = 180)	HIV WITH AIDS (N = 101)	TOTAL (N = 281) (%)
Age (years)	Average	38.1 ± 10.74	38.7 ± 10.62	**38.3 ±10.7**
	Range	(18 – 67)	(18 – 63)	(18 – 67)
Marital status	Single	51	30	**81 (28.8)**
	Married	61	35	**96 (34.2)**
	Widowed	39	20	**59 (21.0)**
	Divorced/ separated	29	16	**45 (16.0)**
Religion	Christians	130	71	**201 (71.5)**
	Muslims	38	22	**60 (21.4)**
	Traditionalists	3	2	**5 (1.8)**
	No religion	9	6	**15 (5.3)**
Tribe	Akans	72	63	**135 (48.0)**
	Gas	37	13	**50 (17.8)**
	Ewes	35	11	**46 (16.4)**
	Northerners	30	12	**42 (14.9)**
	Krobos	5	2	**7 (2.5)**
	Foreigner	1	-	**1 (0.4)**
Educational level	None	19	21	**40 (14.2)**
	Primary	21	8	**29 (10.3)**
	Middle	68	31	**99 (35.2)**
	Secondary/Post-secondary	66	38	**104 (37.0)**
	Tertiary	6	3	**9 (3.2)**

In all, 199 (42.3%) subjects were not having any occupation as at the time of this study. This was made up of 46 non-AIDS subjects (representing 25.6% of their total) and 72 AIDS subjects (representing 72.3% of their total). For subjects who were still working, majority were traders. The numbers and percentages of subjects in each study group who were having the

various occupations are presented in Appendix A. Of the 119 subjects who had no occupation, 112 (94.1%) had lost their jobs apparently due to their infections. This means that only 17 subjects really did not have a job at all. The former occupations of the subjects who had lost their jobs are presented in Appendix B.

With the issue of care giving, 206 subjects (73.3%) were being cared for by one relation or the other. Only 75 subjects (26.7%) were taking care of themselves. This included 64 (35.6%) non-AIDS subjects and only 11 (10.9%) AIDS subjects. Most subjects (especially the AIDS subjects) were being cared for by their parents and spouses. The number of subjects who were being cared for by their relation and the type of relations who were giving care to these subjects in each study group are provided in Appendix C. The occupations of people giving care to subjects are provided in Appendix D.

4.2 Major Clinical Symptoms

The subjects in both HIV without AIDS and HIV with AIDS groups reported clinical symptoms of HIV infection. Diarrhoea was present in 152 (54.1%) of the subjects. This comprised of 70 (38.9%) non-AIDS and 82 (81.2%) AIDS subjects. Skin rashes of various forms and degrees were present in 134 (47.7%) subjects. This comprised of 72 (40%) non-AIDS and 62 (61.4%) AIDS subjects. Varied forms of tuberculosis and coughing were present in 95 (33.8%) subjects, comprising 44 (24.4%) of the non-AIDS and 51(50.5%) of the AIDS subjects. Sore throat was also present in 86 (30.6%) subjects. This included 35 (19.4%) of the non-AIDS (50.5%) and of the AIDS subjects. Anaemia was present in 84 (29.6%) subjects, comprising of 24 (13.3%) of the non-AIDS and 60 (59.4%) of the AIDS subjects. General bodily pains including headaches were present in 67 (23.8%) of the subjects, including 45 (25.0%) of the

non-AIDS and 22 (21.8%) of the AIDS subjects. Oral lesion and other oral problems were present in 82 (29.2%) subjects, comprising 34 (18.9%) of the non-AIDS and 48 (47.5%) of the AIDS subjects. Anorexia was present in 76 (27.0%) of subjects, including 21 (11.7%) of the non-AIDS and 55 (54.5%) of AIDS subjects. Malaria and fever from other causes were present in 63 (22.4%) subjects, including 38 (21.1%) non-AIDS and 55 (54.5%) AIDS subjects. Vomiting was present in 60 (21.4%) subjects, comprising 24 (13.3%) non-AIDS and 36 (35.6%) AIDS subjects. Anal discharges and vaginal discharges (for some females) were present in 39 (13.9%) subjects. This was made up of 24 (13.3%) non-AIDS and 15 (14.9%) AIDS subjects. The number of subjects of the two AIDS status groups who reported the various clinical symptoms of the infection is provided in Table 3 (below).

Table 3: Major clinical symptoms reported by subjects

SYMPTOM	HIV WITH NO AIDS (N = 180) (%)	HIV WITH AIDS (N = 101) (%)	TOTAL (N = 281) (%)
Diarrhoea	70 (38.9)	82 (81.2)	**152 (54.1)**
Skin rashes	72 (40.0)	62 (61.4)	**134 (47.7)**
TB/ cough	44 (24.4)	51 (50.5)	**95 (33.8)**
Sore throat	35 (19.4)	51 (50.5)	**86 (30.6)**
Anaemia	24 (13.3)	59 (58.4)	**83 (29.5)**
Oral lesion	34 (18.9)	48 (47.5)	**82 (29.2)**
Anorexia	21 (11.7)	54 (53.5)	**75 (26.7)**
Bodily pains	45 (25.0)	22 (21.8)	**67 (23.8)**
Fever/ malaria	38 (21.1)	25 (24.8)	**63 (22.4)**
Vomiting	24 (13.3)	36 (35.6)	**60 (21.4)**
Anal discharge	24 (13.3)	15 (14.9)	**39 (13.9)**

4.3 Alcohol Consumption, Smoking and the use of Multivitamin and/ or Mineral Supplements.

In all, 120 (42.7%) of the subjects had never consumed alcohol. This included 81 (45.0%) non-AIDS and 39 (38.6%) AIDS subjects. Of the subjects who had been involved in alcohol consumption, 22 (7.8%) had consumed alcohol within the past week. This was made up of 16 (8.9%) non-AIDS and 6 (59%) AIDS subjects. Also, 24 (8.5%) of the subjects had last consumed alcohol in the past between one to four weeks. This included 18 (10.0%) non-AIDS

and 6 (5.9%) AIDS subjects. Sixty-four subjects (representing 22.8% of the total) had last consumed alcohol over previous one to six months. This comprised 32 (17.8%) non-AIDS and 32 (31.7%) AIDS subjects. About 18 percent of the subjects (n=51) had last consumed alcohol over the previous six months. This included 33 (18.3%) non-AIDS and 18 (17.8%) AIDS subjects. The numbers of subjects in each group who last consumed alcohol in the time duration given above are provided in Appendix E (I).

Smoking was not prevalent among the study population. About 80% of the subjects (n = 199) had never smoked. This was made up of 133 (73.9%) non-AIDS and 66 (65.3%) AIDS subjects. Of the subjects who had smoked, only 8 (2.8%) in total; comprising 6 (3.3%) non-AIDS and 2 (2%) AIDS subjects smoked less than a week before the interview. Only 3 (1.1%) subjects had smoked in the past one to four weeks. This was made up of 1 (0.6%) non-AIDS and 2 (2%) AIDS subject(s). Twenty-seven subjects (representing 9.6% of the total), comprising 15 (8.3%) non-AIDS and 12 (11.9%) AIDS subjects had smoked over the previous one to six months. Also, 44 (15.7%) subjects had smoked during over the previous six months. This included 25 (13.9%) non-AIDS and 19 (18.8%) AIDS subjects. The numbers of subjects in each group who smoked in the time duration given above are provided in Appendix E (II).

A total of 167 (59.4%) subjects were on multivitamin and/ or mineral supplement. This included 63 (62.4%) AIDS subjects and 104 (57.8%) non-AIDS subjects. The number of subjects in the two groups who were using multivitamin and/ or mineral supplements is provided in Appendix E (III).

4.4 Weight and BMI

The non-AIDS males had a mean weight of 57.8 ± 9.5kg which was significantly (P<0.0001) higher than that of AIDS males, who had a mean weight of 45.1 ± 6.2kg. Also, the mean weight for non-AIDS females (55.2 ± 9.2kg) was significantly (P<0.0001) higher than that of the AIDS females, which was 41.4 ± 6.4kg. The mean weights of males and females in the non-AIDS group were not significantly different, but that for males in the AIDS group was significantly (P=0.004) higher than their female counterparts. The mean weights of the subjects in the two groups are presented in Table 4 (below).

Table 4: Mean weight and BMI of subjects

		Weight (kg)	BMI (kg/m^2)
Males	Non – AIDS	57.8 ± 9.5 (35.0 – 85.0)	23.8 ± 3.2 (16.9 – 33.1)
	AIDS	45.1 ± 6.2 (30.0 – 60.0)	18.3 ± 2.3 (12.0 – 23.0)
	P – value	< 0.0001	< 0.0001
Females	Non – AIDS	55.2 ± 9.2 (38.0 – 85.0)	23.9 ± 3.4 (14.8 – 34.3)
	AIDS	41.4 ± 6.4 (32.0 – 62.0)	18.0 ± 2.3 (14.2 – 24.2)
	P – value	< 0.0001	< 0.0001

Table 4 (above), also shows the mean BMI of subjects in the two groups. The mean BMI of non-AIDS male subjects was 23.8 ± 3.2kg/m^2, and this was significantly (P<0.0001) higher than that of the AIDS males who had a mean BMI of 18.3 ± 2.3kg/m^2. The non-AIDS females had a mean BMI of 23.9 ± 3.4kg/m^2 which was significantly (P<0.0001) higher than that of AIDS females who had a mean BMI of 18.0 ± 2.3kg/m^2. The mean BMI of males and females

in the non-AIDS group were not significantly different, and this was the same for the males and females in the AIDS group as well. In all, only 3.8% of the non-AIDS subjects were underweight, 65.6% of them were of normal weight, whilst 25.6% and 5.0% of them were overweight and obese, respectively. For the AIDS subjects, 55.4% of them were underweight, and the rest (44.6%) were of normal weight. The number of subjects in each group who belongs to each of the four BMI categories is provided in Appendix F.

4.5 Food Preferences

The HIV infection and associated clinical manifestations led to 117 (41.6%) subjects stopping the consumption of certain food items they used to consume. This was made up of 47 (26.1%) non-AIDS subjects and 70 (69.3%) AIDS subjects (Appendix G). Forty-six subjects (including 19 non-AIDS and 27 AIDS subjects) had stopped the consumption of highly spiced foods like 'hausa koko' and pepper soup. Of the 46 subjects, 34 (73.9%) stopped because of burning sensations in the mouth possibly due to oral lesions, 5 (10.9%) stopped because they encountered diarrhoea each time after consumption while 4 (8.7%) stopped because the strong aroma from the highly spiced foods put them off. Three subjects stopped because they had stomach cramps each time they consumed the specific food items.

Thirty-eight subjects (including 22 non-AIDS and 16 AIDS subjects) had stopped the consumption of vegetables like 'kontomire', 'okro', garden eggs and groundnut. Of this number, 21 (55.3%) stopped because they had diarrhoea after consumption, 15 (39.5%) stopped because they had stomach cramps after consumption, and 2 (5.3%) stopped because they vomited the item each time after consumption. Twenty-nine subjects (comprising of 9 non-AIDS and 20 AIDS subjects) had stopped consuming heavy food items like 'fufu', 'banku',

'kenkey' etc, because of swallowing problems (65.5%), stomach cramp (31.6%), while 15.8% of the subjects vomited these food items each time they consumed them. One subject, however, thought these food items worsen existing AIDS-related conditions.

Also, 17 subjects (9 non-AIDS and 8 AIDS subjects) had stopped taking beverages, sweets and milk because 8 (47.1%) had diarrhoea after consumption, 5 (29.4%) had stomach cramps and 4 (23.5%) vomited when the items were consumed. Ten subjects (including 3 non-AIDS and 7 AIDS) had stopped consuming oily foods because 8 (80.0%) of them vomited when the food items were consumed. One subject from each group had heart problems and became weak after consuming oily foods. Eight subjects (4 from each group) stopped the consumption of palm and groundnut soup because 6 (75%) of them had diarrhoea after consumption. One subject from each group was either put off by the aroma from these food items or felt that they make existing conditions worse. Appendix G (I) shows the number of subjects who stopped the consumption of each food item and reasons cited by these subjects (Appendix G II).

In all, 117 (41.6%) subjects had stopped consuming at least one food item, 35 (12.5%) subjects (including 19 non AIDS and 16 AIDS subjects) had stopped consumption of at least two items while 10 (3.6%) subjects (including 6 non-AIDS and 4 AIDS subjects) had stopped consuming at least three food items. One subject from non-AIDS group had stopped consuming four food items.

Also, HIV and its clinical manifestation, had led to the consumption of certain food items that subjects were not consuming earlier. In all, 49 (17.4%) subjects, made up of 24 (13.3%) non-AIDS and 25 (24.8%) AIDS subjects, had started consuming at least one food item due to present conditions. Only three subjects had started consuming two new food items.

Thirty-two subjects (comprising 16 subjects from each group) had started consuming garlic and other vegetables and fruits. Of this number, 18 (56.3%) subjects had started on the advice of doctors and/ or experts, 10 (31.3%) subjects were consuming these items because they thought it will enhance the building up of their immune system, 4 (12.5%) subjects had started on the advice of other PLWHA whilst one subject could not assign any particular reason for the consumption of these items. Seven subjects (4 non-AIDS and 3 AIDS subjects) had started consuming honey because it was added to herbal medicinal preparations for curing TB. Three subjects who did not use to consume bread had begun its consumption in order to manage diarrhoea. Tables showing the number of subjects who had started consuming particular food and non-food item(s) and the reasons for their consumption are shown in Appendices G (III) and (IV).

Ten subjects (comprising 4 non-AIDS and 6 AIDS subjects) consumed clay. Even though this is not a food item, they were consuming it to manage diarrhoea.

4.6 Daily Intake of Fat-Soluble Vitamins

The daily mean vitamin A intake by non-AIDS subjects was 959.1μg RE and 879.9μg RE for males and females respectively. These represent 95.7% and 110.0% of their respective RDAs. Likewise, daily mean intake of vitamin A by AIDS subjects was 524.5μg RE and 434.9μg RE for males and females, respectively. These represent 52.5% and 54.4% of RDA for the vitamin met respectively by these subjects. In all, 81 (49.4%) non-AIDS subjects met their RDA for vitamin A, whilst 24 only (23.8%) AIDS subjects met their RDA for vitamin A.

The mean daily vitamin D intake by male and female subjects from non-AIDS group was 3.5μg and 3.1μg respectively, corresponding to 69.6% and 61.2% of the RDA, respectively.

The daily vitamin D intake by males and females from AIDS group was 1.25μg and 0.95μg, respectively, which is equivalent to 25.0% and 19.0% of their respective RDA. On the whole, 53 (29.4%) non-AIDS patients met their RDA for vitamin D, while 3 (3.0%) AIDS patients met their RDA for this vitamin.

Non-AIDS males and females had daily vitamin E intakes of 4.9mg and 4.3mg, respectively, which is equivalent to 49.0% and 53.4% of their respective RDA. For the AIDS subjects, the average daily vitamin E intakes for males and females were 1.6mg and 1.4mg, respectively, representing 15.7% and 17.0% of their respective RDA. Twenty-two non-AIDS subjects (representing 12.2%) met their RDA for vitamin E, while only 1 (1.0%) AIDS subject met the RDA for vitamin E. Table 5 (below) shows the daily mean intakes of the fat-soluble vitamins by the subjects in each study group.

Table 5: Daily mean intakes of fat-soluble vitamins by the subjects

VITAMINS	HIV WITH NO AIDS		HIV WITH AIDS	
	Males Mean (%RDA)	Females Mean (%RDA)	Males Mean (%RDA)	Females Mean (%RDA)
Vitamin A (μg RE)	957.1 (**95.7**)	879.9 (**110.0**)	524.5 (**52.5**)	434.9 (**54.4**)
Vitamin D (μg)	3.5 (**69.6**)	3.1 (**61.2**)	1.25 (**25.0**)	0.95 (**19.0**)
Vitamin E (mg)	4.9 (**49.0**)	4.3 (**53.4**)	1.6 (**15.7**)	1.4 (**17.0**)

Fig.1 (below) illustrates the RDA for each fat-soluble vitamin met by subjects in each study group. Appendix H shows the number and percentage of subjects meeting at least 80% of the RDA for the various vitamins.

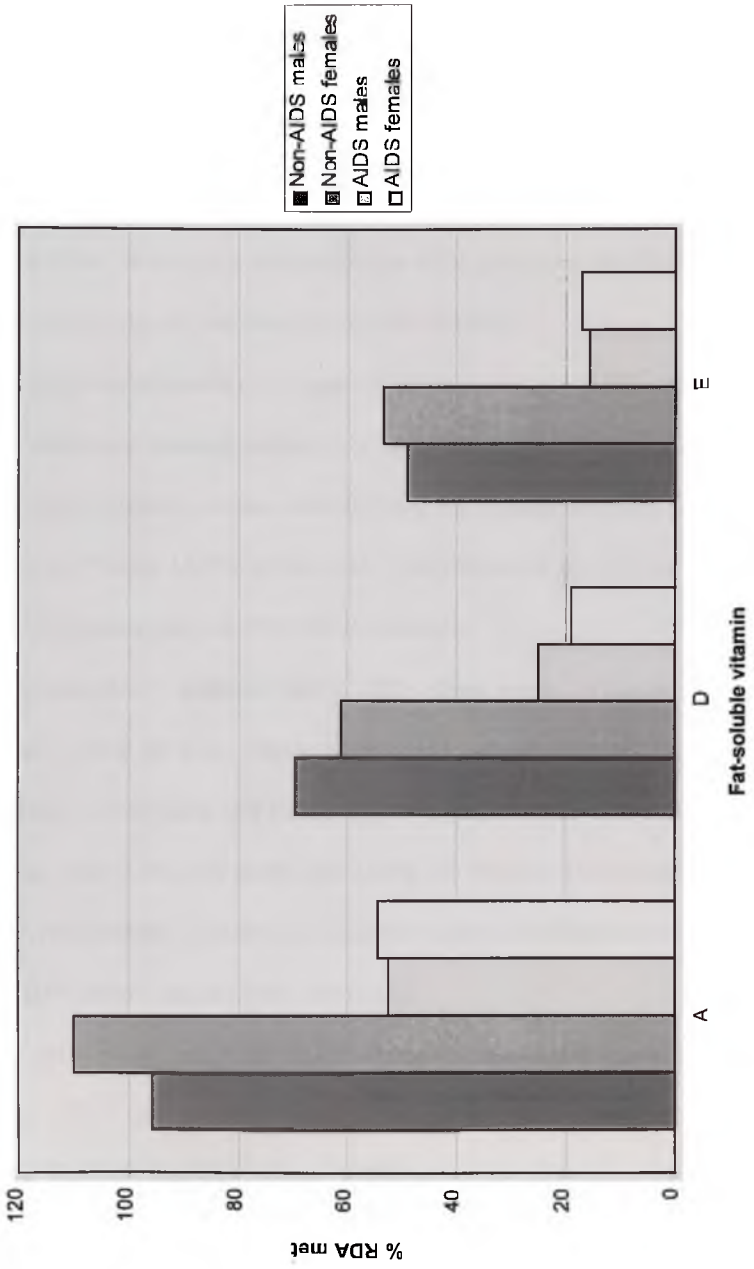


Fig. 1: Mean RDA (%) for fat-soluble vitamins met by subjects

4.7 Daily Intake of Water-Soluble Vitamins.

Male and female non-AIDS subjects had daily mean intakes of 0.66mg and 0.60mg of thiamine (vitamin B_1), respectively. These correspond to 44.0% and 54.5% of the RDA for male and female subjects, respectively. For AIDS subjects, the daily mean intakes of vitamin B_1 were 0.27mg and 0.23mg for male and female subjects, respectively, and correspond to 18.0% and 20.9% of the RDA. None of the subjects in the AIDS group met the RDA for vitamin B_1, but 10 (5.6%) non-AIDS subjects met the RDA for the vitamin.

The daily mean riboflavin (vitamin B_2) intakes by non-AIDS subjects were 0.64mg and 0.55mg for males and females, respectively, or 37.6% and 42.3% of the RDA for vitamin B_2. In the case of AIDS subjects, intakes were 0.25mg and 0.24mg for male and female, respectively representing 14.7% and 18.5% of the RDA. Only three subjects in the non-AIDS groups, and one in the AIDS group met the RDA for the vitamin.

Male non-AIDS subjects had a daily mean niacin (vitamin B_3) intake of 11.4mg compared to 10.0mg by their female counterparts. These represent 59.9% and 66.9% of the RDA for niacin met by male and female subjects, respectively. For AIDS subjects, daily mean niacin intakes were 4.1mg for males and 3.3mg for females. These represent 21.4% and 21.9% of the RDA, respectively. However, 41 (22.8%) of non-AIDS subjects met the RDA, while only 1 (1.0%) AIDS subject met the RDA for niacin.

The daily mean intake of vitamin B_6 by the non-AIDS group was 1.04mg for males, representing 52.0% of the RDA while the females had a daily mean intake of 0.89mg, representing 55.6% of the RDA. For the AIDS group, the daily mean intake was 0.41mg for the males and 0.36mg for the females. These represent 20.5% and 22.5% of the respective RDA for

the vitamin. Seventeen non-AIDS subjects (i.e. 9.4%) met their RDA for vitamin B_6 but only 1 (1.0%) AIDS subject did so.

For AIDS males and females, the daily mean intakes of vitamin B_{12} were 0.75μg (representing 37.5% RDA) and 0.57μg (representing 28.5% RDA), respectively. These were below values for the non-AIDS males and females, which were 1.87μg (indicating 93.5 % RDA) and 1.50μg (indicating 75.0% RDA). While 64 (35.6%) non-AIDS subjects met their RDA for the vitamin, only 5 (5.0%) AIDS subjects met their RDA.

Vitamin C intake was very good among the non-AIDS subjects. The mean intake for males was 69.7mg (indicating 116.2% RDA) and 66.2mg (indicating 110.3% RDA) for females. Among the AIDS subjects, however, intake levels were low. The male AIDS subjects had a daily mean intake of 28.8mg (representing 48.0% RDA) while the females had a mean intake of 19.2mg (representing 32.0% RDA). A total of 111 (61.7%) non-AIDS subjects met their RDA for vitamin C, but 14 (13.9%) of the AIDS subjects met their RDA for the vitamin.

Folate intake was inadequate for all the groups. Among the non-AIDS subjects, males had a daily mean intake of 108.9μg (i.e. 54.4% RDA) whilst females had 97.1μg (i.e. 53.9% RDA). Among the AIDS group, the daily mean intake for males was 50.6μg (i.e. 25.3% RDA) and 45.3μg (i.e. 25.2% RDA) for the females. None of the subjects in the AIDS group met the RDA for the vitamin, but 15 (8.3%) subjects in the non-AIDS subject group met their RDA. Table 6 shows the daily mean intakes of the water-soluble vitamins by the subjects and the percentage of RDA met.

Table 6: Daily mean intakes of water-soluble vitamins by subjects

VITAMINS	HIV WITH NO AIDS		HIV WITH AIDS	
	Males Mean (%RDA)	Females Mean (%RDA)	Males Mean (%RDA)	Females Mean (%RDA)
Thiamine (mg)	0.66 (**44.0**)	0.60 (**54.5**)	0.27 (**18.0**)	0.23 (**20.9**)
Riboflavin (mg)	0.64 (**37.6**)	0.55 (**42.3**)	0.25 (**14.7**)	0.24 (**18.5**)
Niacin (mg)	11.4 (**59.9**)	10.0 (**66.9**)	4.1 (**21.4**)	3.3 (**21.9**)
Vitamin B_6 (mg)	1.04 (**52.0**)	0.89 (**55.6**)	0.41 (**20.5**)	0.36 (**22.5**)
Vitamin B_{12} (µg)	1.87 (**93.5**)	1.50 (**75.0**)	0.75 (**37.5**)	0.57 (**28.5**)
Vitamin C (mg)	69.7 (**116.2**)	66.2 (**110.3**)	28.8 (**48.0**)	19.2 (**32.0**)
Folate (µg)	108.9 (**54.4**)	97.1 (**53.9**)	50.6 (**25.3**)	45.3 (**25.2**)

Fig.2 (below) illustrates the percentage RDA for each water-soluble vitamin met by subjects each group. Appendix H shows the number of subjects from each group who met at least 80% of RDA for each vitamin.

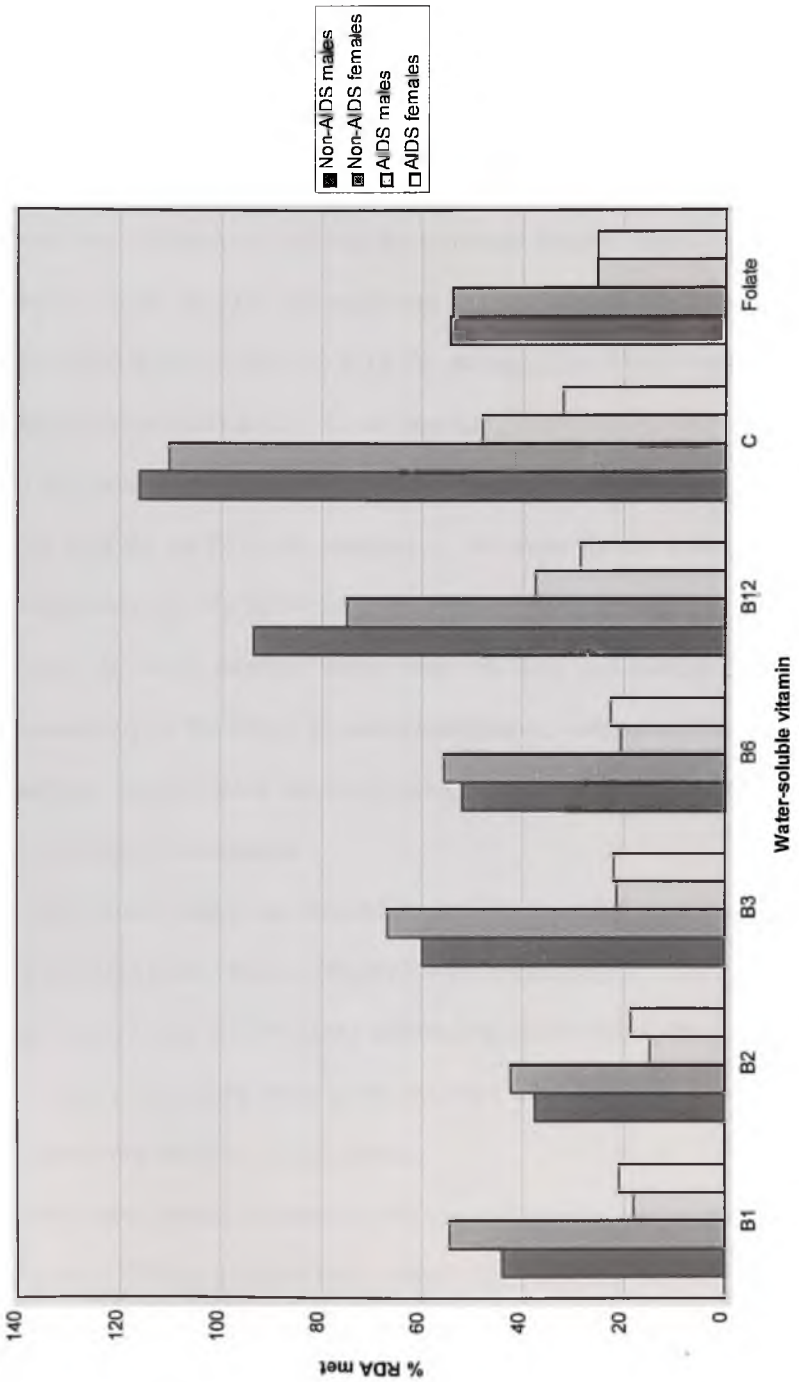


Fig. 2: Mean RDA (%) for water-soluble vitamins met by subjects

4.8 Daily Intake of Major Minerals

The daily mean intake of calcium among the non-AIDS group was 439.5mg for the males and 419.1mg for the females. These indicate the satisfaction of 54.9% and 52.4% of the RDA for calcium by male and female subjects, respectively. Among the AIDS subjects, the average intakes were 194.6mg and 169.6mg for males and females, respectively, corresponding to 24.3% and 21.2% of the RDA for males and females, respectively. Whereas none of the subjects in the AIDS group satisfied the RDA for calcium, 22 non-AIDS subjects (representing 12.2% of their total) satisfied the RDA for the mineral.

Both the males and females in the non-AIDS groups had mean intakes that were commensurate with the set RDA for phosphorus. The mean intakes among this group were 900.7mg (representing 112.6% RDA) for males and 804.9mg (representing 100.6% RDA) for females. Among the AIDS subjects, intakes were 463.5mg (representing 57.9% RDA) and 414.5mg (representing 51.8% RDA) for males and females, respectively. Only 15 (14.9%) of the AIDS subjects met the RDA for the mineral, while a total of 150 (83.3%) non-AIDS subjects met the RDA for phosphorus.

The daily mean intake for magnesium among non-AIDS males and females were 154.1mg (55.0% RDA) and 140.4mg (50.2% RDA), respectively. For the AIDS subjects, the mean intakes were 73.7mg (21.1% RDA) and 66.1mg (23.6% RDA) for males and females, respectively. None of the AIDS subjects met the RDA for magnesium, but 14 (7.8%) of the non-AIDS subjects met the RDA for the mineral.

The daily mean intakes of potassium for non-AIDS males and females were 1454.3mg (72.7%RDA) and 1325.3mg (66.3%RDA), respectively. Male and female AIDS subjects had daily mean potassium intakes of 737.0mg (36.8% RDA) and 662.0mg (33.1%RDA),

respectively. While 66 (36.7%) of the non-AIDS subjects met their RDA for potassium, only one male met the RDA for the mineral in the AIDS group.

For sodium, the daily mean intakes were 136.2mg (27.2% RDA) and 113.0mg (22.6% RDA) for AIDS male and females subjects respectively. For the non-AIDS males and females, the mean intakes were 309.3mg and 266.8mg, respectively. These correspond to 61.9% and 53.4% of RDA, respectively. The number of subjects who met their RDA for sodium was 35 (19.4%) for the non-AIDS subjects and only two for the AIDS subjects. The daily mean intakes of the major minerals by the subjects and the percentage RDA met for each major mineral are presented in Table 7 (below).

Table 7: Daily intake of major minerals by the subjects

MAJOR MINERALS	HIV WITH NO AIDS		HIV WITH AIDS	
	Males Mean (%RDA)	Females Mean (%RDA)	Males Mean (%RDA)	Females Mean (%RDA)
Calcium (mg)	439.5 (**54.9**)	419.1 (**52.4**)	194.6 (**24.3**)	169.6 (**21.2**)
Phosphorus (mg)	900.7 (**112.6**)	804.9 (**100.6**)	463.1 (**57.9**)	414.5 (**51.8**)
Magnesium (mg)	154.1 (**44.0**)	140.4 (**50.2**)	73.7 (**21.1**)	66.1 (**23.6**)
Potassium (mg)	1454.3 (**72.7**)	1325.3 (**66.3**)	737.0 (**36.8**)	662.0 (**33.1**)
Sodium (mg)	309.3 (**61.9**)	266.8 (**53.4**)	136.2 (**27.2**)	113.0 (**22.6**)

Fig.3 (below) illustrates the percentage of the RDA met by subjects in each group. Appendix I shows the number of subjects from each group who met at least 80% of RDA for each mineral.

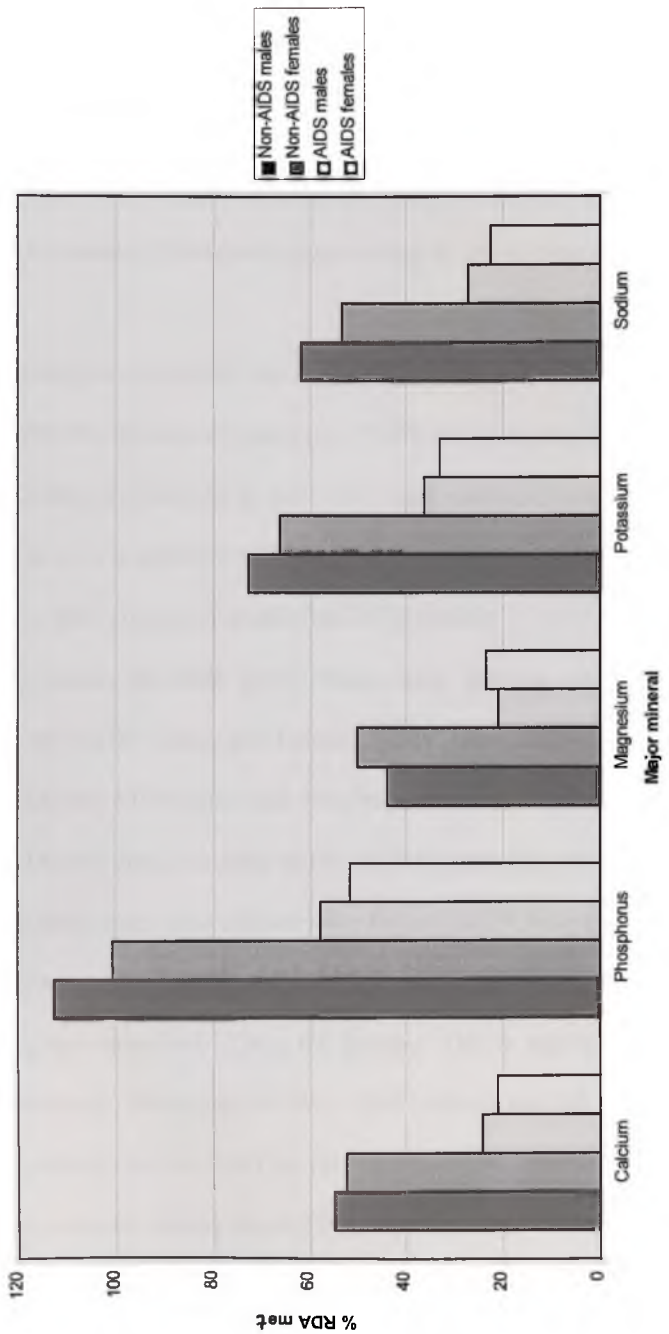


Fig. 3: Mean RDA (%) for major minerals met by the subjects

4.9 Daily Intake of Trace Elements

The daily mean iron intakes were 20.1mg (i.e. 201.4% RDA) and 17.4mg (i.e. 116.3% RDA) by non-AIDS male and female subjects, respectively. Among the AIDS subjects, mean intake was 9.1mg for males and 8.7mg for females. These indicate satisfaction of 91.2% and 58.1% of the RDA for iron, respectively. Forty-three AIDS subjects (representing 42.6% of their) and 166 non-AIDS subjects (representing 92.2% of their total) met the RDA for the trace element.

Among the non-AIDS males and female subjects, the daily mean intakes of zinc were 8.7mg (i.e. 86.9% RDA) and 7.6mg (i.e. 75.9% RDA) respectively. Likewise, among the AIDS group, the daily mean intakes by male and female subjects were 3.3mg and 2.9mg, respectively, equivalent to 33.3% and 29.1% of the RDA. On the whole, 87 (48.3%) non-AIDS subjects met the RDA for zinc, compared to only one AIDS subject.

For iodine, the daily mean intakes were 100.0μg (66.7% RDA) and 89.8μg (59.9% RDA) for non-AIDS males and females, respectively; and 43.6μg (29.1% RDA) and 35.7μg (23.8% RDA) for AIDS males and females, respectively. Only 34 (19.9%) non-AIDS subjects met the RDA for iodine, but none of the AIDS subjects met the RDA for iodine.

The daily mean intake of selenium for non-AIDS male and female subjects, were 33.4μg (47.7% RDA) and 27.8μg (50.6% RDA), respectively. For the AIDS subjects, the mean intake was 14.3μg for males and 12.9μg for females. This is equivalent to 20.4% and 23.5% of the RDA, respectively. While only 4 (4%) AIDS subjects met the RDA for selenium, 26 (14.4%) non-AIDS subjects met the RDA for this trace element.

Male subjects among the AIDS group had a daily mean copper intake of 0.44mg (i.e. 29.3% RDA) whilst the female AIDS subjects had a daily mean intake of 0.35mg (i.e. 23.3%

RDA). For the male and female non-AIDS subjects, intakes were 0.91mg (i.e. 60.7% RDA) and 0.84mg (i.e. 56.0% RDA), respectively. Twenty-seven non-AIDS subjects met the RDA for copper, compared to none of the AIDS subjects. Table (8) shows the daily mean intakes of the element by the subjects and the % RDA met for each trace element.

Table 8: Daily mean intake of trace elements by the subjects

TRACE ELEMENTS	HIV WITH NO AIDS		HIV WITH AIDS	
	Males Mean (% RDA)	Females Mean (% RDA)	Males Mean (% RDA)	Females Mean (% RDA)
Iron (mg)	20.1 (**201.4**)	17.4 (**116.3**)	9.1 (**91.2**)	8.7 (**58.1**)
Zinc (mg)	8.7 (**86.9**)	7.6 (**75.9**)	3.3 (**33.3**)	2.9 (**29.1**)
Iodine (µg)	100.0 (**66.7**)	89.8 (**59.8**)	43.6 (**29.1**)	35.7 (**23.8**)
Selenium (µg)	33.4 (**47.7**)	27.8 (**50.6**)	14.3 (**20.4**)	12.9 (**23.5**)
Copper (mg)	0.91 (**60.7**)	0.84 (**56.0**)	0.44 (**29.3**)	0.35 (**23.3**)

The mean RDA met for each trace element by male and female subjects in the two groups are provided in Fig.4 (below). Appendix I shows the number of subjects who met at least 80% of RDA for each trace element.

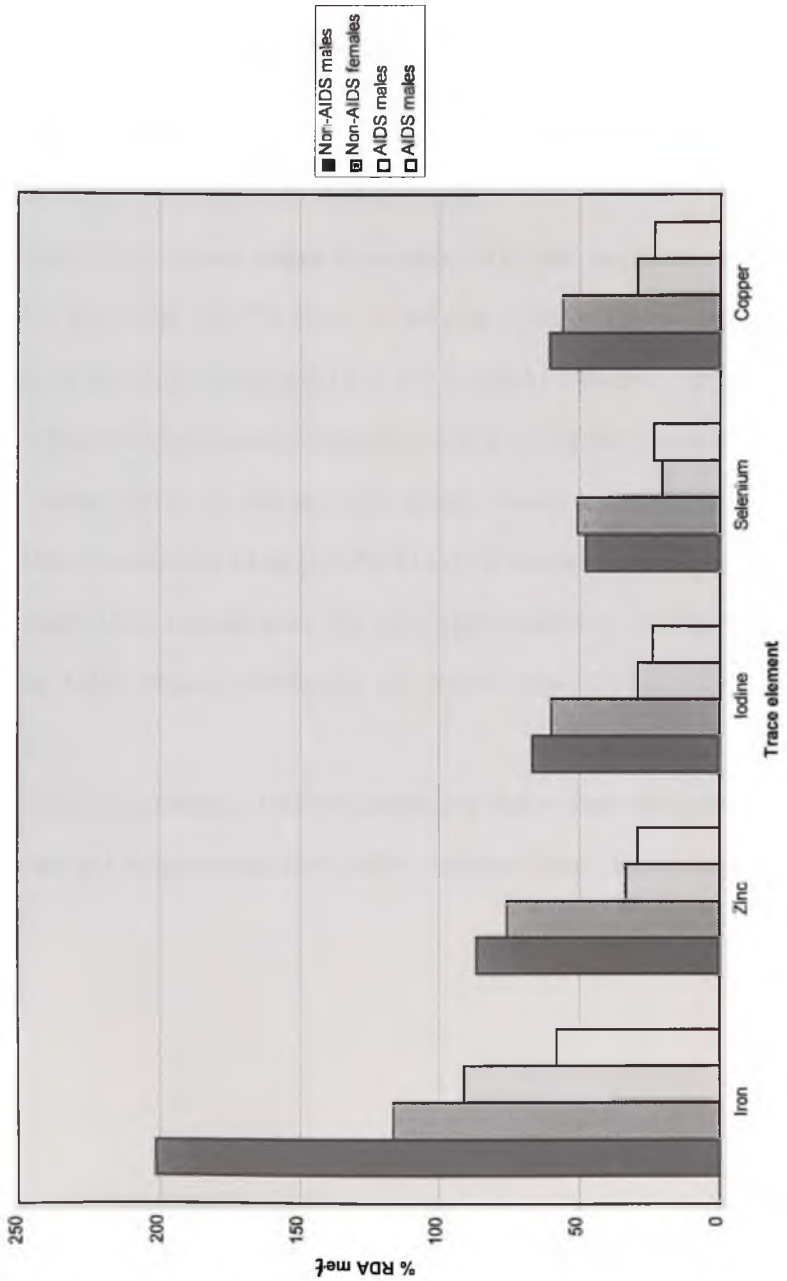


Fig. 4: Mean RDA (%) for trace elements met by subjects

4.10 Daily Intake of Energy, Protein and Dietary Fibre

Among the non-AIDS male and female subjects, daily mean intakes of energy were 1718.4Kcal (59.3% RDA) and 1514.6Kcal (68.8% RDA), respectively. Likewise, daily mean energy intakes were 815.6Kcal and 687.4Kcal for AIDS male and females, representing 28.1% and 31.2% of RDA for energy. None of the AIDS subjects met the RDA for energy, but 34 (18.9%) non-AIDS subjects met their RDA for energy.

The daily mean protein intakes among non-AIDS male and female subjects were 57.4g (91.2% RDA) and 50.6g (101.2% RDA). In the case of the AIDS male and female subjects, intakes were 22.1g (35.0% RDA) and 18.6g (37.2% RDA). However, 123 (68.3%) non-AIDS subjects met their RDA for protein, compared to only 6 (5.9%) AIDS subjects.

For dietary fibre, the average daily intakes among non-AIDS subjects were 17.5g (146.3% RDA) for males and 14.8g (123.4% RDA) for females. The daily mean intakes among male and female AIDS subjects were 7.9g (66.3%RDA) and 4.4g (36.9% RDA), respectively. Twenty-three AIDS subjects (22.8%) and 145 (80.5%) non-AIDS subjects met the RDA for dietary fibre.

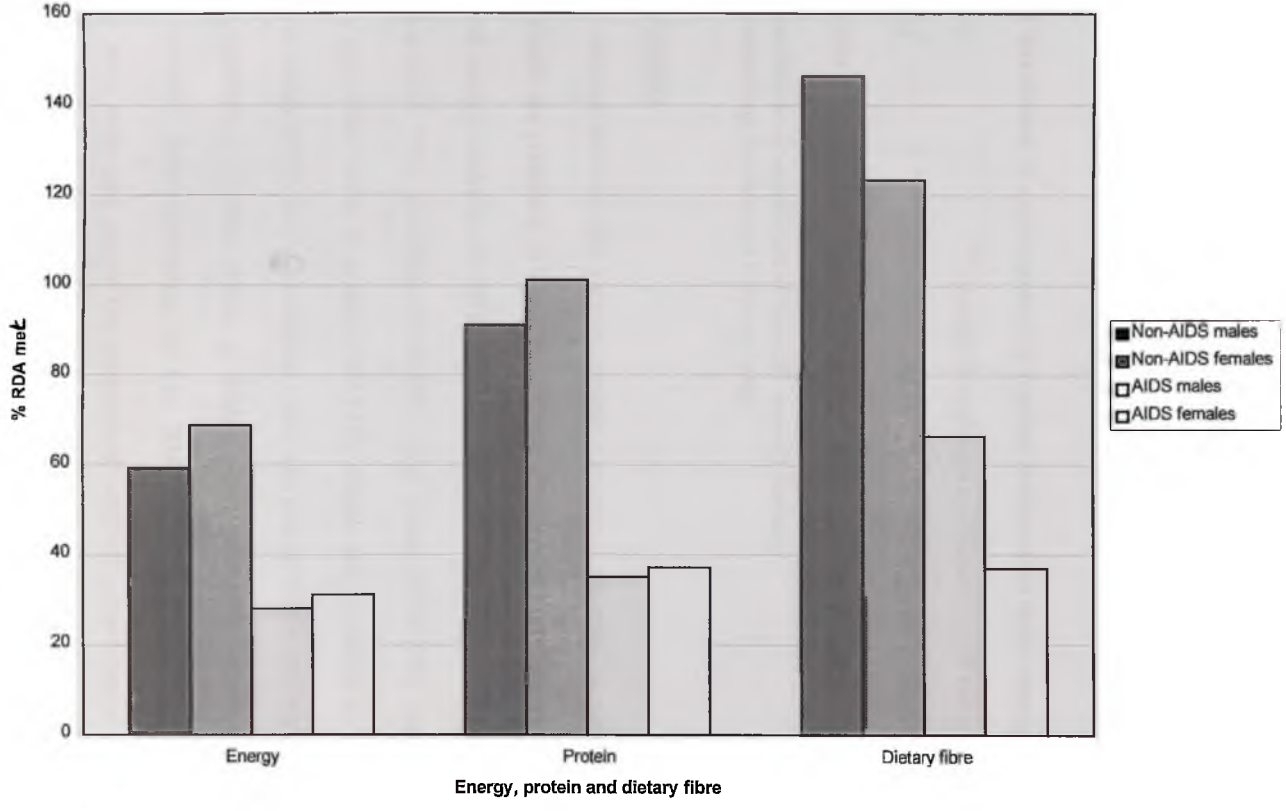
The daily mean intakes of energy, protein and dietary fibre among males and females in the two groups and the percentage RDA met by subjects in each group are provided in Table 9 (below).

Table 9: Daily mean intake of energy, protein and dietary fibre by the subjects

DIETARY INTAKE	HIV WITH NO AIDS		HIV WITH AIDS	
	Males Mean (% RDA)	Females Mean (% RDA)	Males Mean (% RDA)	Females Mean (% RDA)
Energy (Kcal)	1718.4 (**59.3**)	1514.6 (**68.8**)	815.6 (**28.1**)	687.4 (**31.2**)
Protein (g)	57.4 (**91.2**)	50.6 (**101.2**)	22.1 (**35.0**)	18.6 (**37.2**)
Dietary fibre (g)	17.5 (**146.3**)	14.8 (**123.4**)	7.9 (**66.3**)	4.4 (**36.9**)

Fig.5 (below) illustrates the mean RDA met for energy, protein and dietary fibre by male and female subjects in each group. Appendix J shows the number of subjects in each group who met at least 80% RDA for energy, protein and dietary fibre.

Fig. 5: Average % RDA of energy, protein and dietary fibre met by subjects



4.11 Associations between Energy intake and other factors

Anthropometric factors

Energy intake correlated positively and significantly with both BMI (r=0.687) and body weight (r=0.572). These correlations were significant after controlling for sex and disease stage. These are presented in Table 10 (below).

Table 10: Correlation of anthropometric indices factors with daily energy (Kcal) intake.

Factor	Pearson's correlation coefficient, r_p, (P value)	
BMI	0.687* (P<0.0001)	0.409*[a] (P<0.0001)
Weight	0.572* (P<0.0001)	0.241*[a] (P<0.0001)

* Correlation is significant at the 0.01 level (2-tailed).
[a] Controlling for Sex and Disease Stage

Clinical Symptoms

Correlations between the various clinical symptoms reported by subject and the energy intake of subjects are presented in Table 11 (below). The highest correlation was observed between Anorexia and energy intake (r=-0.407). Energy intake also correlated negatively and significantly with anaemia (r=-0.395), diarrhoea (r=-0.358), oral lesion (r=-0.336), sore throat (r=-0.286), tuberculosis and other forms of cough (r=-0.219), nausea and vomiting (r=-0.206) and with all forms of skin rashes (r=-0.160). Correlation's between energy intake and malaria and other forms of fever, headaches and other forms of bodily pairs and anal/vaginal discharges were not significant (p>0.01). After controlling for Sex, BMI and disease stage, energy intake correlated significantly only with oral lesion but correlations with all other clinical symptoms were not significant

Table 11: Correlation of reported symptoms with daily energy (Kcal) intake.

Factor	Spearman's correlation coefficient (P value)	
Anorexia	-0.407* (P<0.0001)	-0.099[b] (P=0.099)
Anaemia	-0.395* (P<0.0001)	-0.046[b] (P=0.448)
Diarrhoea	-0.358* (P<0.0001)	-0.001[b] (P=0.151)
Oral lesion	-0.336* (P<0.0001)	-0.115*[b] (P=0.010)
Sore throat	-0.286* (P<0.0001)	-0.095[b] (P=0.116)
Tuberculosis/ cough	-0.219* (P<0.0001)	-0.027[b] (P=0.656)
Nausea & vomiting	-0.206* (P<0.0001)	-0.013[b] (P=0.830)
Skin rashes	-0.160* (P=0.007)	-0.001[b] (P= 0.987)
Fever/ malaria	-0.042 (P=0.480)	-0.044[b] (P=0.470)
Bodily pains/ headache	-0.024 (P=0.692)	-0.005[b] (P=0.930)
Anal/ vaginal discharges	0.020 (P=0.733)	0.049[b] (P=0.417)

* Correlation is significant at the 0.01 level (2-tailed).
[b] Controlling for Sex, BMI and Disease Stage

Other factors

Table 12 (below) shows the correlations of energy intake with other clinical and lifestyle indicators. The strongest correlation was with the stage of the HIV infection, which correlated negatively and significantly with energy intake (r=-0.725). Energy intake also correlated negatively and significantly with subjects' avoidance of certain food items (r=-0.411). Correlations between energy intake and all other factors were not significant (p>0.01). After controlling for sex and BMI, the correlation between energy intake and disease stage was significant (p<0.01).

However, on controlling for sex, BMI and disease stage, correlations with all other factors were not significant (p>0.01).

Table 12: Correlation of various factors with daily energy (Kcal) intake.

Factor	Spearman's correlation coefficient, r_s, (p value)	
Stage of the infection	-0.725* (P<0.0001)	-0.484*[c] (P<0.0001)
Avoidance of certain foods	-0.411* (P<0.0001)	-0.129[b] (P=0.032)
Consumption of certain foods	-0.149 (P=0.012)	-0.043[b] (P=0.476)
Taking supplement	-0.078 (P=0.191)	0.058[b] (P=0.336)
Smoking	0.018 (P=0.770)	0.028[b] (P=0.643)
Alcohol	0.014 (P=0.821)	0.027[b] (P=0.655)

* Correlation is significant at the 0.01 level (2-tailed).
[c] Controlling for Sex and BMI
[b] Controlling for Sex, BMI and Disease Stage

4.12 Associations between Quality of Life and Intake of various Nutrients

The correlations of the levels of intake of various nutrients with the quality of life of subjects are provided in Table 13 (below). There were significantly positive correlations between quality of life and all the nutrients studied, with the exception of vitamin A. The strongest correlations were with iron (r=0.547), protein (r=0.545), energy (r=0.542), zinc (r=0.526), and niacin (r=0.510). After controlling for sex, disease stage and BMI, quality of life correlate significantly and positively with iron alone, correlations with all other nutrients were not significant at the 0.01 level.

Table 13: Correlation of quality of life with nutrient intakes

NUTRIENTS	Pearson's correlation coefficient, r_p, (P value)	
Vitamin A	0.139 (P=0.020)	0.079[b] (P=0.191)
Thiamine	0.424* (P<0.0001)	0.024[b] (P=0.693)
Riboflavin	0.436* (P<0.0001)	0.067[b] (P=0.254)
Niacin	0.510* (P<0.0001)	0.080[b] (P=0.186)
Vitamin B_6	0.464* (P<0.0001)	0.097[b] (P=0.107)
Vitamin B_{12}	0.355* (P<0.0001)	0.096[b] (P=0.112)
Folate	0.482* (P<0.0001)	0.116[b] (P=0.053)
Vitamin C	0.384* (P<0.0001)	0.107[b] (P=0.074)
Vitamin D	0.430* (P<0.0001)	0.030[b] (P=0.618)
Vitamin E	0.467* (P<0.0001)	0.062[b] (P=0.302)
Calcium	0.356* (P<0.0001)	0.012[b] (P=0.844)
Copper	0.414* (P<0.0001)	0.030[b] (P=0.622)
Iron	0.547* (P<0.0001)	0.161*[b] (P=0.007)
Magnesium	0.445* (P<0.0001)	0.056[b] (P=0.354)
Phosphorus	0.519* (P<0.0001)	0.116[b] (P=0.053)
Potassium	0.453* (P<0.0001)	0.071[b] (P=0.237)
Selenium	0.369* (P<0.0001)	0.001[b] (P=0.992)
Sodium	0.402* (P<0.0001)	0.009[b] (P=0.884)
Zinc	0.526* (P<0.0001)	0.106[b] (P=0.077)
Iodine	0.553* (P<0.0001)	0.114[b] (P=0.057)
Energy	0.542* (P<0.0001)	0.105[b] (P=0.082)
Protein	0.545* (P<0.0001)	0.099[b] (P=0.098)

* Correlation is significant at the 0.01 level (2-tailed).
[b] Controlling for Sex, BMI and Disease Stage

4.13 Associations between BMI and Intake of various Nutrients

Table 14 (below) shows the correlations between BMI and the intake of the various nutrients studied. BMI correlated significantly and positively with intake of all nutrients. Like quality of life, the strongest correlations were with protein ($r=0.718$), iron ($r=0.691$), energy ($r=0.687$), zinc ($r=0.682$) and niacin ($r=0.681$). After controlling for sex and disease stage, correlations with all nutrients (with the exception of vitamin C) were still positively significant ($p<0.01$).

Correlation between BMI and quality of life was positive and significant ($r=0.515$, $p<0.0001$), even after controlling for sex and disease stage ($r=0.162$, $p=0.007$).

Table 14: Correlation of BMI with nutrient intake

NUTRIENTS	Pearson's correlation coefficient, r_p, (P value)	
Vitamin A	0.303* (P<0.0001)	0.176*[a] (P=0.003)
Thiamine	0.601* (P<0.0001)	0.335*[a] (P<0.0001)
Riboflavin	0.575* (P<0.0001)	0.304*[a] (P<0.0001)
Niacin	0.681* (P<0.0001)	0.405*[a] (P<0.0001)
Vitamin B_6	0.587* (P<0.0001)	0.307*[a] (P<0.0001)
Vitamin B_{12}	0.414* (P<0.0001)	0.187*[a] (P=0.002)
Folate	0.592* (P<0.0001)	0.301*[a] (P<0.0001)
Vitamin C	0.388* (P<0.0001)	0.089[a] (P=0.141)
Vitamin D	0.693* (P<0.0001)	0.337*[a] (P<0.0001)
Vitamin E	0.607* (P<0.0001)	0.309*[a] (P<0.0001)
Calcium	0.491* (P<0.0001)	0.226*[a] (P<0.0001)
Copper	0.550* (P<0.0001)	0.259*[a] (P<0.0001)
Iron	0.691* (P<0.0001)	0.428*[a] (P<0.0001)
Magnesium	0.588* (P<0.0001)	0.303*[a] (P<0.0001)
Phosphorus	0.662* (P<0.0001)	0.381*[a] (P<0.0001)
Potassium	0.569* (P<0.0001)	0.266*[a] (P<0.0001)
Selenium	0.529* (P<0.0001)	0.277*[a] (P<0.0001)
Sodium	0.605* (P<0.0001)	0.373*[a] (P<0.0001)
Zinc	0.682* (P<0.0001)	0.403*[a] (P<0.0001)
Iodine	0.444* (P<0.0001)	0.309*[a] (P<0.0001)
Energy	0.687* (P<0.0001)	0.394*[a] (P<0.0001)
Protein	0.718* (P<0.0001)	0.447*[a] (P<0.0001)

* Correlation is significant at the 0.01 level (2-tailed).
[a] Controlling for Sex, and Disease Stage

4.14 Effect of BMI, weight and intake of nutrients on the quality of life of subjects

Table 15 (below) shows the univariate and multivariate regression coefficients and P values of BMI, weight and intake of some nutrients as determinants of quality of life of the subjects studied. Multivariate regression analysis indicates strong effects of BMI and intake of vitamins A and C on the quality of life of subjects.

Table 15: Univariate and multivariate regression coefficients and P values of some determinants of quality of life.

Determinant of quality	Coefficients of regression (P value)	
	Univariate	**Multivariate**
BMI	0.25 (<0.0001)	8.07×10^{-2} (0.050*)
Weight	8.47×10^{-2} (<0.0001)	1.76×10^{-2} (0.201)
Vitamin A	3.71×10^{-4} (0.02)	3.22×10^{-4} (0.032*)
Vitamin C	1.76×10^{-2} (<0.0001)	5.4×10^{-3} (0.045*)
Iron	0.155 (<0.0001)	5.3×10^{-2} (0.081)
Selenium	4.85×10^{-2} (<0.0001)	-1.38×10^{-2} (0.176)
Zinc	0.301 (<0.0001)	5.58×10^{-3} (0.947)
Protein	4.92×10^{-2} (<0.0001)	7.17×10^{-3} (0.649)
Energy	1.90×10^{-3} (<0.0001)	7.20×10^{-4} (0.146)

* Significant at the 0.05 level (2-tailed).

CHAPTER FIVE

DISCUSSION

5.1 Characteristics of Subjects

AIDS and non-AIDS subjects did not differ significantly in terms of most of their background data. This means that, differences in the nutrient intakes in these groups could not have arisen from differences in their background data. For instance, these two groups did not differ significantly in terms of their mean age; 36.1 years for non-AIDS and 36.7 years for AIDS subjects. Also, they did not differ significantly in terms of religion, marital status, tribe, and educational background. However, more AIDS subjects had lost their jobs. This is because the infection makes them weak and therefore unable to work as effectively as expected. Also, stigmatisations from other workers and/ or employers could lead to AIDS subjects losing interest in working.

In terms of care, more AIDS subjects received care than non-AIDS subjects. This may be due to the fact that, most often than not, AIDS subjects are weaker than non-AIDS subjects; they are therefore unable to cater for themselves. They would need people to cook for them, feed them and sometimes take them to see their doctor. In this study, most of the subjects receiving care, received care from their parents and spouse, as well as from their siblings and children. Most of these people giving care to HIV/AIDS patients were traders, who were mostly self-employed and therefore could make time to give care to their loved ones in distress.

5.2 Nutrient Intakes

Nutrient intakes were inadequate in both groups. However, it was worse among the AIDS group. The mean intake of all nutrients (with the exception of iron) by both male and female AIDS subjects was not commensurate with the recommended levels for each particular nutrient. This coupled with the fact that (on individual basis) the numbers of subjects in the AIDS group who were meeting their RDA for each nutrient were not appreciable, emphasises the point that levels of nutrient intakes were inadequate in the AIDS group. For instance, none of the AIDS subjects met the RDA for thiamine, folate, calcium, magnesium, copper and energy.

Among the non-AIDS subjects, however, levels of intake were adequate for vitamins A, B_{12} (males only) and C, phosphorus, iron, zinc (males only), protein and dietary fibre. Indeed, levels of intake of these nutrients were appreciable even though these do not in any way reflect the actual amounts that are available to the body because problems with nutrient absorption and utilization have widely been reported. Keating *et al.* (1995) reported that these may be due to increased intestinal permeability, whilst Babamento and Kotler (1997) suggested the possibility of the HIV infection itself, particularly of the intestinal cells, causing epithelial damage and malabsorption. Levels of intake of all other nutrients studied were not adequate. With respect to those nutrients that levels of intakes were adequate, the percentage of subjects meeting their RDA for those nutrients were as high as 92% for iron, 83% for phosphorus, 68% for protein and 62% for vitamin C.

It is apparently obvious that the levels of the specified nutrient intakes were better in the non-AIDS than in the AIDS group. This conforms to the findings from a study by Parisien *et al.* (1993) who observed lower intake of energy among symptomatic AIDS subjects. However, another study by Dworkin *et al.* (1993) found protein intakes to be similar for the two groups and

that these intakes exceeded RDA guidelines. HIV seropositive non-AIDS subjects could be as normal as seronegative subjects in various aspects. Depending on the stage of the infection, HIV seropositive non-AIDS subjects could be comparable to seronegative subjects with respect to their nutrient intakes. In fact, a case of HIV positive non-AIDS subjects having better intakes of specific nutrients than HIV seronegative subjects has been reported. In a study by Hogg *et al.* (1995) seropositive gay men had significantly higher intakes of energy (median, 2,198 against 1,971Kcal, p=0.019), and protein (median 94 against 81g, p=0.011), than their seronegative counterparts.

From this study, the most significant cause of the lower levels of nutrient intakes was attributed to inadequate consumption of food. This could be due to the presence of certain specific clinical complications associated with the HIV/AIDS infection. Anorexia was reported by about 27% of the subjects (mostly AIDS subjects), and this could be responsible for the inadequate intake of food. The level of anorexia (27%) in the present study was lower, compared to 45% reported in a study in Cote D'Ivoire by Castetbon *et al.* (1997). The difference could be due to possible differences in the characteristics of the two study subjects in terms of the stages of the infection, disposition of the subjects and other factors. In the current study, anorexia was more prevalent among the AIDS group than the non-AIDS group and this could be partly responsible for the differences in the nutrient intakes between these two groups. Anorexia could arise from sore throat, oral lesion, diarrhoea, vomiting and/ or tuberculosis, which were also more prevalent among the AIDS subjects than the non-AIDS subjects. In fact, in this study a correlation matrix (Appendix K) indicated that anorexia significantly and positively correlated with sore throat ($r = 0.140$, $p = 0.018$), oral lesion ($r = 0.126$, $p = 0.035$), diarrhoea ($r = 0.185$, $p = 0.002$), vomiting ($r = 0.157$, $p = 0.008$) and TB ($r = 0.164$, $p = 0.006$).

Sore throat and oral lesion could lead to inadequate intake of food. In the present study, this was evident because about 66% of the subjects that they had stopped the consumption of heavy foods, due to sore throat and/ or oral lesion. Burning sensations in the mouth and/ or difficulty in swallowing masticated food makes it practically impossible for these subjects to consume food in adequate amounts in order to meet their RDAs. Also, diarrhoea and vomiting could affect the intake of food. Indeed, most of the subjects, who had stopped consuming most of the other food items, had done so because they suffered from diarrhoea each time they consumed the specific food items. Diarrhoea presents more nutritional problems beyond just nutrient intake; it also affects nutrient utilization in the GIT.

In the present study, energy intake was used as an index of nutrient intake, and it correlated significantly and negatively with almost all the clinical symptoms reported by the subjects. These correlations were expected because most of these symptoms affect dietary intake. Energy intake correlated significantly and negatively with the presence of anorexia ($r=-0.407$, $P<0.0001$), and this supports the findings from a study by Sheehan and Macallan (2000) who found energy intake to be significantly related to appetite. It is clear that those who are not taking enough food will not be able to meet their energy requirements. As explained above, most of the other clinical symptoms reported independently affected appetite and subjects' urge to stop the consumption of heavy food items and other food items, which in tend lead to their inability to meet their energy requirements. Indeed, Niyongabo *et al.* (1999) reported that inadequate nutrient intake among HIV seropositive subjects was associated with tuberculosis ($P<0.0001$). The discomfort associated with coughing in the presence of tuberculosis could be responsible for the reduction in food intake.

Energy intake also correlated significantly and negatively with the stage of the infection. This may be due to the fact that AIDS subjects were presented with more clinical symptoms. Since

most of these symptoms affected dietary intake negatively, the negative correlation was expected. This means that, HIV positive subjects with no AIDS had a higher energy intake than subjects with AIDS. This finding conflicts with that of a study by Dannhauser *et al.* (1999). In that study, more than half the patients had low intake of vitamins C, B_6, D, A, calcium, iron and zinc, but there was no significant association between disease stage and nutrient intakes. This difference could be due to the fact that these researchers used CD4+ T-lymphocyte counts in staging the disease among their study subjects, which means that instead of only two stages in the current study, their study had three stages depending on the subject's CD4+ T-lymphocyte counts.

Energy intake also correlated significantly and negatively with avoidance of certain food items. This may be due to the fact that most of the subjects who avoided these food items had done so due to the presence of the very clinical symptoms that have been deem to influence energy intake negatively. Also, most of these subjects had stopped consuming heavy foods like 'fufu', 'kenkey' etc, which are the major sources of energy in the context of the typical Ghanaian diet.

Energy intake correlated significantly and positively with the anthropometric indices used in this study. The correlation was strong with weight ($r=0.572$, $P<0.0001$) and more so with BMI ($r=0.687$, $P<0.0001$). This is expected since energy intake accounts for body weight and composition. Subjects with adequate levels of energy intake are able to maintain their body weight to an appreciable level. On the other hand, subjects with inadequate levels of energy tend to lose weight. However, the study by Dworkin *et al.* (1990) found no significant correlations between specific anthropometric measurements and dietary intakes of protein or fat (major indicators of energy intake).

Energy intake did not correlate significantly with lifestyle habits like cigarette smoking, alcohol consumption and intake of nutrient supplements. This finding is not in agreement with a

study by Subar *et al.* (1990) among US nationals whose HIV status were unknown. The findings of that study revealed that cigarettes smokers had lower dietary intakes. This nonconformity could be due to the fact that the prevalence of cigarettes smoking, alcohol consumption and intake of nutrient supplements, among subjects in the current study were very low. For instance, in a study of the dietary attitudes and practices of low-income-African-Americans with AIDS by Doyle *et al.* (2001), 71% of the subjects were using multivitamin and/ or mineral supplements. In the current study, only about 59% of the subjects were on one multivitamin and/ or mineral supplement. However, other studies have reported lower percentage of subjects taking supplements. In a study by Bandy *et al.* (1993), only 46% of the subjects were using multivitamin and mineral supplements

Even though average levels of intakes of almost all the nutrients were higher in the male subjects than in the female subjects in both groups, the percentages of RDAs met for each nutrient were not significantly different. This is in view of the fact that, in most cases, the RDA set for males are higher than that set for females (Recommended Dietary Allowances, 1989). So, a higher level of intake by a male might not necessarily correspond to a relatively higher RDA met.

5.3 BMI and Weight

Comparing the weight and BMI of non-AIDS subjects with AIDS subjects revealed that non-AIDS males had average weight and BMI that were significantly higher than that of AIDS males. This result repeated itself for the females. However, there were no significant differences between the average BMI and weight of males and females in the same group. This means that in effect, the average weight and BMI of the subjects in the non-AIDS group was significantly higher than that of the subjects in the AIDS group. This result conforms to that of Castetbon *et al.* (1997) and Dannhauser *et al.* (1999) who observed that anthropometric values were significantly lower in

symptomatic patients than asymptomatic patients. Indeed, HIV infection is accompanied by subsequent emaciation. The level of emaciation is directly dependent on the stage of the infection. This renders AIDS subjects more emaciated than their HIV positive non-AIDS counterparts and this may be responsible for the differences in weight and BMI values observed for the two groups. In actual fact, weight loss is a major condition for the AIDS definition. However, whereas in the current study male and female subjects did not differ significantly in terms of their average BMI, the study by Dannhauser *et al.* (1999) reported that the men were leaner (BMI=18.9 kg/m^2) than the women (BMI=22.7 kg/m^2).

Emaciation can occur as a result of inadequate dietary intake or the direct results of the clinical manifestations of the HIV infection like diarrhoea. In this study, energy intake correlated significantly and positively with both BMI and weight, which suggests that emaciation can occur as a result of inadequate energy intake. Also, BMI correlated significantly and positively with all the other nutrients studied, which also suggests that subjects with adequate intakes of these nutrients are able to maintain their weight. The strongest correlations were with protein (r=0.718) and energy (r=0.687), which emphasises the role of PEM in HIV/AIDS-related weight loss and wasting. However, Macallan (1999) reported that HIV infection results in increased energy and protein requirements, therefore meeting the requirements set for HIV/AIDS-free populations might not necessarily be a panacea for PEM among HIV/AIDS individuals.

5.4 Clinical Symptoms and Quality of Life

The most frequent opportunistic infection reported by the subjects was diarrhoea. It was reported by about 54% of the subjects. Various studies from different parts of the world have reported different prevalence of diarrhoea among HIV/AIDS subjects. Studies from the highly

industrialised countries have reported lower prevalence of diarrhoea than those from the developing countries. These differences may have arisen due to the differences in the environments subjects in those studies find themselves. For instance, whilst a study in Germany by Schwenk *et al.* (1993) reported diarrhoea among 25% of the subjects, a Cambodia study by Senya *et al.* (2003) reported 41.2%. In Africa, Kelly (1998) reported that studies from Zaire, the Central African Republic, Uganda, and Tanzania suggest that 40 – 80% of people with HIV/AIDS suffer from diarrhoea at some stage. Previously, a study by De Cock (1989) in Tanzanian had reported a prevalence rate of 75%. This means that the prevalence obtained in the present study falls within the reported range in the African context. In the present study, diarrhoea was more prevalent among the AIDS group than the non-AIDS group ($P< 0.0001$).

Anorexia was present in 26.7% of the subjects. Anorexia is a common problem in HIV infection and occurs through several mechanisms, including local pathology in the oral cavity or oesophagus, central nervous system disease affecting eating mechanics or the perception of hunger. Secondary anorexia may be due to systemic infection, malabsorption, or medication, or to non-medical factors such as psychosocial problems, poverty, and isolation. Various studies have reported different prevalence rates of anorexia among HIV/AIDS patients. A study by Tindall *et al.* (1988) in Australia reported 56.4% of homosexual HIV/AIDS males having anorexia whilst the Germany study by Schwenk *et al.* (1993) reported 69.2%. In Africa, the study by Catestbon *et al.* (1997) in Cote D'Ivoire reported that appetite problems were present in 45% of the HIV/AIDS patients, particularly among the symptomatic patients. The lower rate reported in the present study could be due to underreporting by the subjects. The prevalence of anorexia was significantly higher among the AIDS group than the non-AIDS group ($P<0.0001$).

Anaemia was reported by 29.5% of the subjects. Anaemia is the most common haematological manifestation of the HIV/AIDS infection (Claster, 2002). The causes of HIV-related anaemia are multifactorial and include direct and indirect effects of HIV infection. HIV-related anaemia generally is due to reduced red blood cell (RBC) production, secondary to a variety of causes, but it may also involve nutritional deficiencies, increased RBC destruction, or a combination of these causes. Farinas (1998) reported that anaemia affects 15% of asymptomatic HIV-positive individuals and 85% of those with AIDS. In the present study, anaemia was present in 13.3% of non-AIDS and 58.4% of AIDS subjects. Anaemia impacts negatively on individuals, in that it leaves victims with diminished capacity to carry oxygen in their blood, thereby leading to fatigue, difficulty in breathing and increased heart rate.

Oral lesions of various causes and degrees were present in about 29% of the subjects. This percentage is small compared to 60.4% prevalence reported for HIV/AIDS subjects in South Africa (Arendorf *et al*, 1998). Likewise, a prevalence 40.2% for oral lesions was observed among AIDS subjects studied in Zambia (Hodgson, 1997). Outside Africa, various studies have reported higher percentages of oral lesions for HIV/AIDS subjects than the percentage reported in the present study. In a study in Thailand by Khongkunthian *et al.* (2001), oral lesions were reported by 38% of the HIV/AIDS subjects, whilst Campisi *et al.* (2001) reported 47% in a study in Italy. A study in Mexico by Ramirez-Amador *et al.* (1998) reported oral lesion for 75% HIV-positive subjects. However, the present study recorded that 47.5% of AIDS subjects suffered from oral lesion, a value which is lower than the 72% prevalence rate among AIDS patients in south India reported by Ranganathan *et al.* (2000).

In the present study, the prevalence of sore throat was 30.6%. This is lower than the prevalence rate 56.4 % reported in the Australian study by Tindall *et al.* (1988). However, the

AIDS patients reported a significantly higher prevalence of sore throat than non-AIDS subjects ($p<0.0001$).

Tuberculosis and other forms of coughing were present in 33.8% of the subjects. This is higher than a prevalence 26% reported in the Cambodia study by Senya *et al.* (2003), or 13% reported for AIDS patients in Cote d'Ivoire by Grant *et al.* (1997). Nevertheless, the AIDS patients reported significantly higher prevalence of tuberculosis than non-AIDS subjects ($P<0.0001$).

Malaria and fever of various causes were present in 22.4% of the subjects, but AIDS patients reported significantly higher prevalence of fever than the non-AIDS subjects ($p<0.0001$). However, the German study by Schwenk *et al.* (1993) reported that 30% of HIV/AIDS patients had fever. The report from the Australian study by Tindall *et al.* (1988) indicated 76%, and a Tanzanian study by De Cock (1989), reported fever prevalence of 79%.

Skin rashes of various forms were present in 48%. Headache and other forms of bodily pains were present in 24% of the subjects. About 21% of the subjects reported vomiting whilst about 14% of the subjects reported anal discharges. In all these clinical complications, AIDS subjects reported significantly higher prevalence rates than non-AIDS subjects ($p<0.0001$).

Quality of life scores correlated significantly and positively with levels of intakes of all the nutrients studied, with the exception of vitamin A. This may be due to the fact that the quality of life scores were calculated based on the number of clinical symptoms that each subject was free from. Since most of these clinical symptoms correlated negatively with energy intake, which was used as an indicator of nutrient intake, the positive correlations of the intakes of the various nutrients with quality of life scores were expected.

Quality of life and nutrient intakes operate in tandem, in that subjects with good quality of life are better placed to consume adequate amounts of meals to meet their RDAs. On the other

hand, subjects who do not meet their RDAs for the various nutrients, especially the micronutrients are more prone to the various opportunistic infections associated with HIV/AIDS and therefore are unable to maintain good quality of life.

Quality of life also correlated significantly and positively with BMI (Table 15). Weight loss could be a direct consequence of the impact of the infection and subsequent opportunistic infections. Subjects with more opportunistic infections (i.e. lower quality of life scores) tend to lose more weight and therefore have declined BMI. Subjects with better quality of life are therefore better placed to maintain a normal BMI, hence the significantly positive correlation even after controlling for potential confounders like sex and the stage of the disease.

The quality of life subjects were affected by BMI, weight and intakes of vitamins A and C, iron, zinc, selenium, protein and energy. However, multivariate regression analysis indicated that only BMI and intakes of vitamins A and C were the strong determinants of subjects' quality of life. P values show levels of significance ($p \leq 0.05$) for only these three parameters (Table 15). Indeed, vitamin A has been associated with the immunocompetence of PLWHA and their resistance to infections. This means that subjects with adequate intakes of vitamin A had a better immune status and were therefore protected from some of the clinical symptoms used to compute their quality of life scores in this study. Coutsoudis *et al.* (1995) in a study in South Africa reported that vitamin A supplementation reduced morbidity from diarrhoea in HIV-infected children. This means that adequate levels of intake of vitamin A could be protective against diarrhoea, which was the most prevalent clinical condition in this study. Vitamin C acts as an antioxidant (Piwoz and Preble, 2000) and also increases individual's resistance to infections (especially fever). This could explain it effect on the quality of life of the subjects.

CONCLUSIONS

From the results of this study, the following conclusions could be drawn;

1. AIDS subjects had inadequate dietary intake of all nutrients, except the males who met RDA for iron.

2. Non-AIDS subjects met RDAs for vitamins A and C, iron, phosphorus, protein and dietary fibre, and zinc (males only) and vitamin B_{12} (males only); but not vitamins B_1, B_2, Niacin, B_6, Folate, D and E; minerals Ca, Mg, K, Na, I_2, Se, Cu as well as energy.

3. Energy intake correlated significantly and positively with BMI and weight, and negatively with the stage of the infection and avoidance of certain food items.

4. Energy intake correlated significantly and negatively with the presence of anorexia, anaemia, diarrhoea, oral lesion and sore throat.

5. Quality of life of subjects correlated significantly and positively with their BMI and weight.

6. Quality of life of subjects correlated significantly and positively with intakes of all nutrients studied. The correlation was significant ($p<0.01$) for all the nutrients studied, except vitamin A. BMI of subjects correlated significantly and positively with intakes of all nutrients studied

7. BMI and intakes of vitamins A and C are strong determinants of the quality of life of HIV/AIDS subjects.

RECOMMENDATIONS

Experience from this study suggests the following recommendations;

1. Further studies be done in the area of food preferences of PLWHA, which can be used in planning for recipes for this population.

2. The use of CD4+ T-lymphocyte counts in HIV/AIDS disease staging be incorporated in future studies involving PLWHA. This will ensure a more accurate way of staging the disease.

3. The vitamin A and C nutritional status of PLWHA be looked at through biochemical analysis of blood and urine samples.

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APPENDICES

Appendix A: Present occupation of subjects

OCCUPATION	HIV WITH NO AIDS N=180 (% in bracket)	HIV WITH AIDS N = 101 (%)	TOTAL N = 281 (%)
None	46 (25.6)	73 (72.3)	**119 (42.3)**
Trader	60 (33.3)	11 (10.9)	**71 (25.3)**
Tailor / seamstress	17 (9.4)	1 (1)	**18 (6.4)**
Secretary	13 (7.2)	1 (1)	**14 (5.0)**
Teacher	12 (6.7)		**12 (4.3)**
Farmer	7 (3.9)	3 (3)	**10 (3.6)**
Driver / driver's mate	7 (3.9)	3 (3)	**10 (3.6)**
Painter / mason	5 (2.8)	3 (3)	**8 (2.8)**
Housewife / maid	2 (1.1)	4 (4)	**6 (2.1)**
Policeman / woman	3 (1.7)		**3 (1.1)**
Doctor / nurse	3 (1.7)		**3 (1.1)**
Caterer	1 (0.6)	1 (1)	**2 (0.7)**
Businessman / woman	2 (1.1)		**2 (0.7)**
Administrator		1 (1)	**1 (0.4)**
Student	1 (0.6)		**1 (0.4)**

Appendix B: Former occupation of subjects with no present occupation

OCCUPATION	HIV WITH NO AIDS N = 46 (% in bracket)	HIV WITH AIDS N = 73 (%)	TOTAL N = 119 (%)
None	8 (17.3)	9 (12.3)	**17 (14.3)**
Trader	17 (37.0)	30 (41.0)	**47 (39.5)**
Teacher	4 (8.7)	7 (9.6)	**11 (9.2)**
Driver / driver's mate	3 (6.5)	6 (8.2)	**9 (7.6)**
Secretary	2 (4.3)	5 (6.8)	**7 (5.9)**
Farmer	1 (2.2)	6 (8.2)	**7 (5.9)**
Tailor / seamstress	2 (4.3)	3 (4.1)	**5 (4.2)**
Painter / mason	3 (6.5)		**3 (2.5)**
Administrator	1 (2.2)	2 (2.7)	**3 (2.5)**
Housewife / maid		2 (2.7)	**2 (2.5)**
Mechanic		2 (2.7)	**2 (2.5)**
Caterer	1 (2.2)	1 (1.4)	**2 (2.5)**
Businessman / woman	2 (4.3)		**2 (2.5)**
Student	1 (2.2)		**1 (0.8)**
Traditional herbalist	1 (2.2)	-	**1 (0.8)**

Appendix C: Relations giving care to subjects

RELATION	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101 (%)	TOTAL N = 281 (%)
No care – giver	64 (35.6)	11 (10.9)	**75 (26.7)**
Parent	37 (20.6)	19 (18.8)	**56 (19.9)**
Spouse	32 (17.8)	18 (17.8)	**50 (17.8)**
Sibling	14 (7.8)	25 (24.8)	**39 (13.9)**
Child	11 (6.1)	13 (12.9)	**24 (8.5)**
Other family member	14 (7.8)	4 (4.0)	**18 (6.4)**
Friend	6 (3.3)	9 (8.9)	**15 (5.3)**
In – law	2 (1.1)	1 (1.0)	**3 (1.1)**
Pastor	-	1 (1.0)	**1 (0.4)**

Appendix D: Occupation of people from whom subjects derive care

OCCUPATION	HIV WITH NO AIDS N = 116 (% in bracket)	HIV WITH AIDS N = 90 (%)	TOTAL N = 206 (%)
None	8 (6.9)	10 (11.1)	**18 (8.7)**
Trader	42 (36.2)	29 (32.2)	**71 (34.5)**
Teacher	25 (21.6)	22 (24.4)	**47 (22.8)**
Farmer	10 (8.6)	11 (12.2)	**21 (10.2)**
Secretary	13 (11.2)	6 (6.7)	**19 (9.2)**
Businessman / woman	4 (3.4)	4 (4.4)	**8 (3.9)**
Tailor / seamstress	3 (2.6)	2 (2.2)	**5 (2.4)**
Nurse	3 (2.6)	1 (1.1)	**4 (1.9)**
Driver / driver's mate	2 (1.7)	1 (1.1)	**3 (1.5)**
Administrator	2 (1.7)	1 (1.1)	**3 (1.5)**
Policeman / woman	1 (0.9)		**1 (0.5)**
Painter / mason	1 (0.9)	-	**1 (0.5)**
Mechanic	1 (0.9)	-	**1 (0.5)**
Contractor	1 (0.9)		**1 (0.5)**
Housewife / maid		1(1.1)	**1 (0.5)**
Banker	-	1 (1.1)	**1 (0.5)**
Student	-	1 (1.1)	**1 (0.5)**

Appendix E (I): Last time subjects took Alcohol.

	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101 (%)	TOTAL N = 281 (%)
Less than a week	16 (8.9)	6 (5.9)	**22 (7.8)**
1 week – 1 month	18 (10.0)	6 (5.9)	**24 (8.5)**
1 mo+ to 6months	32 (17.8)	32 (31.7)	**64 (22.8)**
6 mo+	33 (18.3)	18 (17.8)	**51 (18.1)**
Never	81 (45.0)	39 (38.6)	**120 (42.7)**

Appendix E (II): Last time subjects smoked.

	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101 (%)	TOTAL N = 281 (%)
Less than a week	6 (3.3)	2 (2.0)	**8 (2.8)**
1 week – 1 month	1 (0.6)	2 (2.0)	**3 (1.1)**
1 mo+ to 6months	15 (8.3)	12 (11.9)	**27 (9.6)**
6 mo+	25 (13.9)	19 (18.8)	**44 (15.7)**
Never	133 (73.9)	66 (65.3)	**199 (70.8)**

Appendix E (III): Use of nutrient supplements among subjects

	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101 (%)	TOTAL N = 281 (%)
Taking supplements	104 (57.8)	63 (62.4)	**167 (59.4)**
Not Taking supplements	76 (42.2)	38 (37.6)	**114 (40.6)**

Appendix F: Distribution of subjects according to BMI their interpretations

BMI INTERPRETATION	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101 (%)
Underweight (< 18.4 Kg/m^2)	7 **(3.8)**	56 **(55.4)**
Normal (18.5 – 24.9 Kg/m^2)	118 **(65.6)**	45 **(44.6)**
Overweight (25.0 – 29.9 Kg/m^2)	46 **(25.6)**	–
Obese (> 30.0 Kg/m^2)	9 **(5.0)**	–

Appendix G (I): Food items subjects have stopped consuming.

	HIV WITH NO AIDS (N = 180)	HIV WITH AIDS (N = 101)	**TOTAL** (N = 281)
Highly spiced foods like 'hausa koko'	19	27	**46**
'Kontomire'/ 'okro'/ garden eggs/ groundnut	22	16	**38**
Heavy foods like 'fufu', 'banku', 'kenkey' etc	9	20	**29**
Beverages/ sweets/ milk	9	8	**17**
Oily foods	3	7	**10**
Palm/ groundnut soup	4	4	**8**
Grains like maize		3	**3**
'Gari' soakings	2	1	**3**
High protein foods like beans, 'agushi' etc	1	1	**2**
'Mpotompoto'	1		**1**
Bread	1		**1**
Coconut	1		**1**

Appendix G (II): Reasons given by subjects for stopping consumption of food items.

Food item	Reasons (number of subjects in bracket)
Highly spiced foods like 'hausa koko'	1. Burning sensations in the mouth (**34**) 2. Diarrhoea after consumption (**5**) 3. Put off by aroma from food item (**4**) 4. Stomach cramps/ discomfort (**3**)
'Kontomire'/ 'okro'/ garden eggs/ groundnut	1. Diarrhoea after consumption (**21**) 2. Stomach cramps/ discomfort (**15**) 3. Vomit food item after consumption (**2**)
Heavy foods like 'fufu', 'banku', 'kenkey' etc	1. Swallowing problems (**19**) 2. Stomach cramps/ discomfort (**6**) 3. Vomit food item after consumption (**3**) 4. Food item makes conditions worse (**1**)
Beverages/ sweets/ milk	1. Diarrhoea after consumption (**8**) 2. Stomach cramps/ discomfort (**5**) 3. Vomit food item after consumption (**4**)
Oily foods	1. Vomit food item after consumption (**8**) 2. Heart problems after consumption (**1**) 3. Gets weak after consumption (**1**)

Food item	Reasons (number of subjects in bracket)
Palm/ groundnut soup	1. Diarrhoea after consumption (**6**) 2. Put off by aroma from food item (**1**) 3. Food item makes condition worse (**1**)
Grains like maize	1. Food item makes condition worse (**2**) 2. Swallowing problems (**1**)
'Gari' soakings	1. Stomach cramps/ discomfort (**1**) 2. Swallowing problems (**1**) 3. Chest pains after consumption (**1**)
High protein foods like beans, 'agushi' etc	1. Diarrhoea after consumption (**1**) 2. Put off by aroma from food item (**1**)
Bread	1. Constipate after consumption (**1**)
Coconut	1. Vomit food item after consumption (**1**)
'Mpotompoto'	1. Diarrhoea after consumption (**1**)

Appendix G (III): Food items subjects had started consuming

	HIV WITH NO AIDS (N = 180)	HIV WITH AIDS (N = 101)	**TOTAL** (N = 281)
Fruits/ vegetables/ garlic	16	16	**32**
Honey	4	3	7
Bread	2	1	**3**
Clay**	4	6	**10**

** Not a food item but was being consumed

Appendix G (IV): Reasons given by subjects for starting to consume food items.

Food item	Reason (number of subjects in bracket)
Fruits/ vegetables/ garlic	1. Advice by doctors/ experts (**18**) 2. To build up my immune system (**10**) 3. Advice from other patients (**4**) 4. No particular reason (**1**)
Honey	1. In anti – TB herbal medicinal preparation (**7**)
Bread	1. To fight diarrhoea (**3**)
Clay**	1. To fight diarrhoea (**10**)

** Not a food item but was being consumed

Appendix H: Number of subjects obtaining at least 80 percent of RDA for vitamins.

VITAMIN	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101 (% in bracket))
Vitamin A	89 **(49.4)**	24 **(23.8)**
Vitamin D	53 **(29.4)**	3 **(3.0)**
Vitamin E	22 **(12.2)**	1 **(1.0)**
Thiamine	10 **(5.6)**	0 **(0.0)**
Riboflavin	3 **(1.7)**	1 **(1.0)**
Niacin	41 **(22.8)**	1 **(1.0)**
Vitamin B_6	17 **(9.4)**	1 **(1.0)**
Vitamin B_{12}	64 **(35.6)**	5 **(5.0)**
Vitamin C	111 **(61.7)**	14 **(13.9)**
Folate	15 **(8.3)**	0 **(0.0)**

Appendix I: Number of subjects obtaining at least 80 percent of RDA for minerals.

MINERAL	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101(% in bracket)
Calcium	22 **(12.2)**	0 **(0.0)**
Phosphorous	150 **(83.3)**	15 **(14.9)**
Magnesium	14 **(7.8)**	0 **(0.0)**
Potassium	66 **(36.7)**	1 **(1.0)**
Sodium	35 **(19.4)**	2 **(2.0)**
Iron	166 **(92.2)**	43 **(42.6)**
Zinc	87 **(48.3)**	1 **(1.0)**
Iodine	34 **(18.9)**	0 **(0.0)**
Selenium	26 **(14.4)**	4 **(4.0)**
Copper	27 **(15.0)**	0 **(0.0)**

Appendix J: Number of subjects obtaining at least 80 percent of RDA for energy, protein and dietary fibre.

DIETARY COMPONENT	HIV WITH NO AIDS N = 180 (% in bracket)	HIV WITH AIDS N = 101(% in bracket)
Energy	34 **(18.9)**	0 **(0.0)**
Protein	123 **(68.3)**	6 **(5.9)**
Dietary fibre	145 **(80.5)**	23 **(22.8)**

Appendix K: Correlation matrix of clinical symptoms.

	Diarrhoa	Skin ras	Anorexi	Ana discharg	Vomitin	Sor throat	Ora lesion	Fever/ malari	TB/ coug	Bod pain	Anemi
Diarrhoa											
r_s	1.00	.07	.185*	-.02	.184*	.209*	.183*	-.08	.205*	-.07	.330*
P valu		.18	.00	.70	.00	.00	.00	.14	.00	.23	.00
Skin rash											
r_s	.07	1.00	.10	.13	.00	.07	.07	.05	.14	-.03	.225*
P valu	.18		.09	.02	.91	.19	.20	.39	.01	.58	.00
Anorexia											
r_s	.185**	.10	1.00	-.05	.157*	.14	.12	.04	.164**	.04	.297*
P valu	.00	.09		.34	.00	.01	.03	.48	.00	.50	.00
Anal discharge											
r_s	-.02	.13	-.05	1.00	-.08	-.02	-.09	-.06	.01	-.03	.05
P valu	.70	.02	.34		.16	.72	.09	.25	.76	.60	.35
Vomit											
r_s	.184**	.00	.157*	-.08	1.00	.01	.04	-.05	.12	.11	.196*
P valu	.00	.91	.00	.16		.84	.42	.39	.03	.05	.00
Sore throat											
r_s	.209**	.07	.14	-.02	.01	1.00	.253*	-.04	.06	.04	.179*
P valu	.00	.19	.01	.72	.84		.00	.48	.28	.45	.00
Oral lesion											
r_s	.183**	.07	.12	-.09	.04	.253*	1.00	.01	.170*	-.12	.236*
P valu	.00	.20	.03	.09	.42	.00		.84	.00	.04	.00
Fever/ malaria											
r_s	-.08	.05	.04	-.06	-.05	-.04	.01	1.00	.03	-.08	-.03
P valu	.14	.39	.48	.25	.39	.48	.84		.60	.17	.61
TB/ cough											
r_s	.205**	.14	.164*	.01	.12	.06	.170*	.03	1.00	-.02	.411*
P valu	.00	.01	.00	.76	.03	.28	.00	.60		.62	.00
Body pains											
r_s	-.07	-.03	.04	-.03	.11	.04	-.12	-.08	-.02	1.00	-.05
P valu	.23	.58	.50	.60	.05	.45	.04	.17	.62		.39
Anaemi											
r_s	.330**	.225*	.297*	.05	.196*	.179*	.236*	-.03	.411*	-.05	1.00
P valu	.00	.00	.00	.35	.00	.00	.00	.61	.00	.39	

r_s Spearman's correlation coefficient

** Correlation is significant at the 0.01 level (2-tailed).

Appendix L: Semi – structured questionnaire for collecting information

DEPARTMENT OF NUTRITION AND FOOD SCIENCE
UNIVERSITY OF GHANA, LEGON.

THE ASSOCIATION BETWEEN NUTRIENT INTAKE, BODY ANTHROPOMETRY AND QUALITY OF LIFE OF HIV/AIDS PATIENTS IN GHANA.

A. BACKGROUND INFORMATION

A1. Date …… A2. Age…….. A3. Serial No …….. A4. Sex : M / F

A5. Study Area : (1) Korle Bu (2) Komfo Anokye

A6. Marital status: (1). Single (2). Married (3). Divorced/Separated (4). Widowed

A7. Religion: (1). None (2). Christian (3). Muslim

(4) Traditionalist (5) Other…….. ……

A8. Tribe: (1). Akan (2). Ewe (3). Ga (4). Northerner (5). Other……..

A9. Educational level: (1). None (2). Primary (3). Middle/JSS

(4). Secondary/Vocational/Post-secondary (5) Tertiary

A10. Occupation before infection: (1). None (2). Farmer (3). Teacher

(4).Trader (5). Secretary/ accounts clerk (6). Other……

A11. Current Occupation: (1). None (2). Farmer (3). Teacher

(4).Trader (5).Secretary/Accounts clerk (6). Other……

A12. Occupation of Caregiver (1). None (2). Farmer (3). Teacher

(4). Trader (5). Secretary/Accounts clerk (6). Other……

A13. Relationship to Caregiver (1). Spouse (2). Parent (3). Child

(4). Other family member (5). Friend (6). Other……

B. HOSPITAL AND BIOCHEMICAL DETAILS

B1 Date of admission…… B2. HIV diagnosis (years) ……

B3. Classification of disease stage: (1) HIV Positive with no AIDS

(2) HIV Positive with AIDS

B4. Reported complications: (1). None (2). Diarrhoea (3). Skin rashes (4). Anorexia

(5). Anal discharge (6). Vomiting (7). Sore throat (8). Oral lesion

(9)Anaemia (10) Tuberculosis (11) Other :….. ……

B5. Are you on any multivitamin and/ or mineral supplement? (1). NO (2). YES

B9. Supplement type and analysis ……...

……..

B10. When was the last time you smoked? ……..

B11. When was the last time you consumed alcohol? ……..

C. ANTHROPOMETRIC MEASUREMENT

C1. Weight on admission: ……..…… C2. Current weight: ……

C3. Height: …… C4. BMI:……

D. DIETARY SURVEY

D1. Do you have problems with eating? (1). NO (2). YES (If NO skip question D2)

D2. What problems? .....

D3. Have you stopped consuming any food item due to your present condition?

(1). NO (2). YES (If NO, skip question D4)

D4. Name these items and the reasons why you stopped consuming them

ITEM	REASONS

D5. Have you started consuming any food item that you were not consuming before your present condition? (1). NO (2). YES (If NO, skip question D7)

D6. Name these items and the reasons why you have started consuming them.

ITEM	REASONS

D7a. 24hr recall (*first weekday)*

MEAL PATTERN	**FOOD TYPE**	**QUANTITY**
BREAKFAST		
A.M. SNACK		
LUNCH		
P.M. SNACK		
SUPPER		
BEDTIME SNACK		

D7b. 24hr recall (*second weekday)*

MEAL PATTERN	**FOOD TYPE**	**QUANTITY**
BREAKFAST		
A.M. SNACK		
LUNCH		
P.M. SNACK		
SUPPER		
BEDTIME SNACK		

D7c. 24hr recall (*weekend*)

MEAL PATTERN	**FOOD TYPE**	**QUANTITY**
BREAKFAST		
A.M. SNACK		
LUNCH		
P.M. SNACK		
SUPPER		
BEDTIME SNACK		