Malnutrition, dietary diversity, morbidity and associated factors among schoolchildren in Kibwezi district, Kenya

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DECLARATION

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DEDICATION

This thesis is lovingly dedicated to my aunt Roselida and Uncle Lucas Aduol who have been my constant source of inspiration. These two people have made indemonstrable contribution towards my education. Without their love and support I would not have made it this far. God bless you!

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ABBREVIATIONS

AIDS Acquired Immunodeficiency Syndrome

ANP Applied Nutrition Program

ARIs Acute Respiratory Infections

BAZ BMI-for-age z-score

BMI Body Mass Index

CDC Centre for Disease Control

CDR Crude Death Rate

CRP C-Reactive Proteins

CVI Common Variable Immunodeficiency

DBM Double Burden of Malnutrition

DOMC Division of Malaria Control

ELISA Enzyme-linked immunosorbent assay

FAO Food and Agriculture Organization

GDP Gross Domestic Product

GoK Government of Kenya

HAZ Height-for-age z-score

Hb Hemoglobin

HIV Human Immunodeficiency Virus

HPLC High Performance Liquid Chromatography

ID Iron Deficiency

IDA Iron Deficiency Anemia

INSTAPA Improving Nutrition through Staples in Africa

IU International Unit

IVACG International Vitamin A Consultative Group

KDHS Kenya Demographic and Health Survey

KNBS Kenya National Bureau of Statistics

MI Micronutrient Initiative

MRDR Modified-Relative Dose Response

NGOs Non-governmental Organizations

PCD Partnership for Child Development

PEM Protein Energy Malnutrition

RBP Retinol-Binding Protein

RDA Recommended Dietary Allowance

TAWSB Tanathi Water Services Board

UNICEF United Nations Children's Fund

VAD Vitamin A Deficiency

VADD Vitamin A Deficiency Disorders

WAZ Weight-for-age z-score

WHO World Health Organization

OPERATIONAL DEFINITIONS

Anemia A condition characterized by hemoglobin concentration below established

cut off levels depending on age, sex and physiological status. In this study

anaemia is defined as Hb of <11.5 and <12g/dl for age groups 5-11 and 12-

14 years respectively

BMI-for-age Is a growth indicator that relates BMI to age. BMI-for-age is determined

using gender specific growth charts that place a child in percentile relative

to weight and height.

Stunting A condition where a child is too short for their age (<-2SD height-for-age)

Thinness A BMI-for-age of <2SD

Underweight A condition where a child has a low weight for their age (Z score<2SD

weight-for-age)

Micronutrient Defined in terms of anemia and vitamin A deficiency. A condition resulting

malnutrition from inadequate intake of foods rich in iron and vitamin A thus resulting in

sub-normal serum levels of the indicators for iron (hemoglobin) and vitamin

A (serum RBP).

Macronutrient Defined in terms of BMI-for-age (thinness), Height-for-age (stunting) and

malnutrition weight-for-age (underweight) z-scores of <2SDs.

Vitamin A Low serum levels of retinol binding protein (RBP) defined as <1SD serum

deficiency RBP z-score

ABSTRACT

In Kenya, information on the nutritional status of schoolchildren is scanty. This study assessed malnutrition levels (stunting, underweight, thinness, and anemia and serum RBP levels), dietary diversity and morbidity among children in Kibwezi district and served to provide baseline information for a randomized controlled trial of the effect of provitamin A biofortified cassava on vitamin A status of primary school children in Kenya.

A cross-sectional survey was conducted in three primary schools selected based on an estimated prevalence of Vitamin A Deficiency of 50%, size of the school and willingness to participate. Information on household socio-economic and demographic characteristics and children's morbidity burden was collected using pre-tested questionnaires and trained interviewers. A fourteen day recall period and a 24-hour recall period was used for morbidity and individual diversity score respectively. Blood for RBP and haemoglobin analysis was drawn by qualified and trained technicians. Descriptive statistics were computed for demographic and socio-economic data. Logistic regression and bivariate correlation analyses were performed to establish relationships. A P-value of <0.05 was considered significant in all the analyses.

In total, 423 pupils (52.2% males and 47.8% females) were included in the study. Mean age of the children was 9.3 ± 2.3 years. The mean household size of 7 (± 2.2) members was significantly higher than the national figure of 4.6 for rural areas ($t_{420} = -130.544$, p= 0.000). The age dependency ratio was 149.

Anemia was detected in 10% of the children of which 4.7% were mildly anemic, 5.0% moderately anemic and only 0.3% severely anemic. Non anemic children were less likely to fall below the 50th percentile of the population serum RBPP.

Prevalence of underweight was highest (31.7%) followed by thinness (28.9%) then stunting (18.0%). There was no significant association between the sex of a child and the nutritional status ($\chi^2 = 0.444$, df = 1, p =0.504). Children under the care of their mothers were less likely to have poor nutritional status.

The mean IDDS was 3.3 ± 1.0 food groups. At the 5% level, the proportions of children that had low, medium and high IDDS did not differ significantly between the schools (χ 2=8.367, df=4, P>0.05). The main source of income for the household was a significant predictor of individual dietary diversity (OR=1.4; 95%CI=1.3, 2.6). Children from households with permanent employment being main source of income were likely to have a higher IDDS.

Of the 423 children, 22.0% were sick in the two weeks preceding the survey of which only 52.2% sought medical intervention. The mean number of times a child was sick in the two weeks was 1.06 (± 0.25) times while the mean number of days per occasion of illness was 3.15 (± 2.4) days. Educational level of the caregiver, hemoglobin and dietary diversity score were strong predictors of morbidity status of a child (p<0.05).

The study concludes that anemia is of mild public health significance among schoolchildren. Malnutrition measured by stunting, underweight and thinness is widespread among the schoolchildren. There is poor diet quality evidenced by low individual dietary diversity. Educational levels of the caregiver and dietary diversity are a determinant of child morbidity. Moreover, there is a high rate of disease burden among schoolchildren.

CHAPTER ONE: INTRODUCTION

1.1 Background Information

Malnutrition refers to disorders resulting from an inadequate diet or from failure to absorb or assimilate dietary elements. The school-age period is nutritionally significant because this is the time to build up body stores of nutrients in preparation for rapid growth in adolescence (Kamari and Jain, 2005). In children, protein/calorie deficient diet results in underweight, wasting and lowered resistance to infection, stunted growth and impaired cognitive development and learning. The main nutritional problems facing the school-age child include among others stunting, underweight, vitamin A deficiency, anemia and iodine deficiency. In countries experiencing the 'nutrition transition', overweight and obesity are increasing problems in the school-age child. The main health problems facing schoolchildren are malaria, helminth infections, diarrheal diseases, respiratory infections, and the direct and indirect effects of HIV/AIDS (SCN, 2002).

Deficiency of micronutrients has long been recognized as a public health problem in developing countries. Globally, the most common micronutrient deficiencies are those of vitamin A, iron, and iodine, with an estimated 2 billion people (mostly women and children) at risk for these deficiencies (Ramakrishman, 2002). These micronutrient deficiencies impair growth, cognitive function, immune responses, and reproductive health.

Iron deficiency is the most prevalent and common micronutrient deficiency in the developing world (Tatala *et al.*, 1998; Asobayire *et al.*, 2001; Abalkhail & Shawky, 2002; Hashizume *et al.*, 2003). In particular, iron deficiency anemia leads to weakness, poor physical growth, and a

compromised immune system – decreasing the ability to fight infections and increasing morbidity. It is also thought to impair cognitive performance and delay psychomotor development. Macroeconomic estimates suggest that the average impact of iron deficiency anemia, through both physical and cognitive channels, could be as large as 4 percent of GDP in less developed countries (Horton and Ross, 2003). Through its impact on schooling, anemia could also be central to understanding the intergenerational transmission of poverty. Beside other vulnerable age groups, such as infancy and early childhood, adolescence is placed at a high-risk level for developing iron deficiency due to a combination of menstrual iron losses in girls and a rapid physical growth, especially in boys (Fomon *et al.*, 2003).

Poor diet quality and low dietary iron bioavailability are the principal factors that contribute to the increased incidence of iron deficiency (Tatala *et al.*, 1998). The bioavailability of heme iron, present in animal products, is high with absorption rates of 20–30%, whereas the bioavailability of non-heme iron is determined by the presence of enhancing or inhibiting factors (Hurrell, 1997).

Vitamin A deficiency (VAD) has for a long time continued to be a public health problem in many developing countries affecting about 190 million pre-school children globally (WHO, 2009a) and has been identified as the leading cause of pediatric blindness and a major determinant of severe infections and mortality among children in the developing world (McLaren & Frigg, 2001). In Africa, the prevalence of night blindness and serum retinol concentration of <0.70 μmol/l among pre-school children was 2% (2.55million children) and 44.4% (56.4 million) respectively (WHO, 2009a). Among the pregnant women, the prevalence was 9.8% (3.02 million) and 13.5% (4.18 million) respectively (WHO, 2009a). In Kenya VAD has been

classified as a moderate and severe public health problem using night blindness and serum retinol concentration respectively (WHO, 2009a). The prevalence of VAD is highest among children (14.7% - severe VAD and 61% -moderate VAD) (Mwaniki *et al.*, 1999). Such data is however missing on schoolchildren. This study was therefore formulated to bridge this gap in knowledge.

1.2 Statement of the Problem

The problem of malnutrition defined by low weight-for-age, low weight-for-height and low height-for-age, does not only affect children below five years but also school-age children (Stoltzfus *et al.*, 1997; PCD, 1998; Osei *et al.*, 2010; Daboné, 2011). Unfortunately, Demographic and Health Surveys (DHS), which provide nutritional status data at national level, do not include schoolchildren.

Furthermore, micronutrient deficiencies have been shown to be quite high in school-age children in the developed countries (Solon *et al.*, 2000; Singh and West Jnr, 2004, Maslova *et al.*, 2009; Osei *et al.*, 2010; Daboné *et al.*, 2011). However, the Kenya National Micronutrient Survey of 1999 as well as that of 2012 did not include this group but the rates could be high. The schoolage years are an opportune time to address the nutritional and health problems for several reasons. The school setting offers an ideal distribution system for public health interventions of many types (Savioli *et al.*,, 1992), including health education, micronutrient supplementation, and treatment or prevention of parasitic infections. To develop and target effective interventions to address the problems affecting the schoolchildren, a sound epidemiologic understanding of the problem is needed. In Kenya just like most of the developing world, such information is lacking.

1.3 Justification of the Study

Because schoolchildren undergo rapid growth, nutrient deficiencies at this age can lead to retarded growth, xeropthalmia, anemia, reduced immune function, and impaired motor and cognitive development (Black, 2003). Such outcomes adversely affect academic performance through reduced learning capacity and poor school attendance. Additionally, later in life, affected children have reduced work capacity.

Schoolchildren have not been considered to be at risk of malnutrition in many parts of the world Kenya included; and little is known about occurrence of malnutrition in this group. This limits the ability of policy makers to develop effective interventions to address the problem. Therefore, to develop effective interventions to combat malnutrition in this group of children, a sound understanding of nutrition condition is essential.

1.4 Study Aim

The aim of this study was to contribute to reduction of the levels of malnutrition in Kenya.

1.5 Study Purpose

This study served to provide information on malnutrition levels, dietary diversity and morbidity burden among children in Kibwezi district, Kenya and to provide baseline information for a randomized controlled trial of the effect of provitamin A biofortified cassava on vitamin A status of primary school children in Kenya.

1.6 Objectives

The main objective of the study was to estimate the prevalence of malnutrition, to describe the individual dietary diversity and morbidity burden and the associated factors among schoolchildren in Kibwezi District. This study therefore had the following specific objectives;

- 1. To describe the socio-demographic and economic characteristic of the study population
- 2. To estimate the prevalence of malnutrition among study children in terms of stunting, underweight and BMI-for-age
- 3. To determine the serum RBP of the study children as a proxy indicator for vitamin A malnutrition
- 4. To estimate the prevalence of anemia among the study children
- 5. To determine the individual dietary diversity among schoolchildren in Kibwezi district
- 6. To describe the morbidity pattern among schoolchildren in Kibwezi district
- 7. To establish factors associated with malnutrition, dietary diversity and morbidity burden among schoolchildren in Kibwezi

1.7 Hypotheses

Prevalence of anemia is of public health significance among schoolchildren in Kibwezi district

There is no difference in the levels of malnutrition between schoolchildren in Kibwezi and the levels in Eastern province

Individual dietary diversity score is dependent on household economic status.

Nutritional status in terms of stunting, underweight, thinness, Hb and RBP is dependent on household scocio-economic factors

1.8 Limitations of the Study

The study design was cross-sectional in nature; therefore, it is difficult to explain any seasonal variation in the micronutrient status of the children. Furthermore, it limits the ability to determine any causal relationships.

CHAPTER TWO: LITERATURE REVIEW

2.1 Introduction

The term malnutrition refers to disorders resulting from inadequate intake of nutrients from the diet or from failure to absorb or assimilate dietary elements. Malnutrition is a complex phenomenon. In the past, malnutrition was thought only to be "undernutrition," presumably protein-energy malnutrition (PEM) in the developing countries. In recent years, various vitamin and mineral deficiencies, including vitamin A, iron, iodine and zinc have been recognized as discrete types of malnutrition that adversely affect human health and contribute to disease and mortality (Rice *et al.*, 2004). A growing prevalence of over –nutrition manifested in obesity and its related chronic diseases have been observed in both pre-school and school age children (Prentice, 2006; Cattaneo *et al.*, 2010; Beroncello *et al.*, 2008). In developing countries, this rising epidemic along with the persistent underrnutrition and infections constitute the 'Double Burden of Malnutrition' (DBM) (FAO, 2006), a great concern for most of the African countries (Thiam *et al.*, 2006). The main nutritional problems facing the school-age child include stunting, underweight, anaemia and iodine deficiency and, based on information from recent surveys, vitamin A deficiency (SCN, 2002).

2.2 Macro-nutrient Malnutrition

Protein-energy malnutrition (PEM) is a common condition in children living in developing countries or belonging to socioeconomically underprivileged cohorts of the population of some industrial countries (Decsi & Koletzko, 2000). PEM predisposes children to various infectious diseases and contributes significantly to childhood morbidity and mortality. PEM is the major

cause of secondary immunodeficiency in the world (Cunningham-Rundles *et al.* 2004). Many studies have observed that PEM can lead to clinically significant immune deficiency and infections in children (Scrimshaw, 2003; Keusch, 2003; Neumann *et al.*, 2004). Amongst the effects that PEM has on immunocompetence, the most striking are: (i) atrophy of the lymphoid tissue, particularly in the thymus; (ii) a reduction in delayed cutaneous hypersensitivity; (iii) a reduction in the number of T cells, especially T helper cells; (iv) a decrease in thymulin activity; (v) decreased secretory immunoglobulin A antibody response; and (vi) a reduced concentration and activity of complement components and phagocyte dysfunction (Borelli & Nardinelli, 2001; Anstead et al., 2003; Niiya *et al.*, 2007; Fock *et al.*, 2007).

2.2.1 Stunting, Underweight and Thinness in Children

Stunting or low height-for-age is a physical indicator of chronic or long-term malnutrition and it often results in poor health and school performance, impaired physical and mental development and perpetuation of the cycle of poverty as it results in deficits in productivity in adulthood (Berkman *et al.*, 2002; Kossmann *et al.*, 2000). Stunting is a cumulative process of undernutrition originating from infancy and may worsen or persist during the school-age years (Casapia *et al.*, 2006; Khuwaja *et al.*, 2005). It appears that although children show evidence of growth into late adolescence, full 'catch-up' growth does not occur and linear growth retardation persists throughout the school years. Studies of catch-up growth in children show moderate levels of improvement, with 25-32% of the children stunted at age 2 years attaining the appropriate heights by school-age or pre-adolescence (Adair, 1999). This suggests that interventions in school-age children can supplement efforts in the preschool years to reduce levels of stunting and related effects on children's health and education (Dekker *et al.*, 2010). In

Guatemala, a study by Immink and Poyangayong (1999) on impoverished rural children who benefited from a poverty alleviation programme identified sanitation and housing conditions as the major risk factors for growth faltering in school-age children. Dekker *et al.* (2010) on the other hand found that stunting in schoolchildren associated strongly with socioeconomic and maternal nutritional status indicators whereas thinness did not associate with socioeconomic status.

Underweight (low weight-for-age) is an indicator of both chronic and acute undernutrition. Underweight among school-age children, like stunting, can reflect a broad range of insults such as prenatal undernutrition, deficiencies of macro- and micronutrients, infection and, possibly, inadequate attention by caregivers. In a prospective cohort study of school-age children in Bangladesh, diarrhoea was identified as the main cause of retarded weight gain and slow linear growth (Torres *et al.*, 2000).

Thinness (low BMI-for-age), which reflects acute malnutrition, is not as common as either stunting or underweight in school-age children. Nevertheless, wasting rates can change rapidly in crises, with school-age children, adolescents in particular, becoming severely malnourished in such situations (SCN, 2002).

2.2.2 Childhood Overweight and Obesity

Overweight and obesity is becoming increasingly prevalent in low-income countries where improvements in socioeconomic conditions and rapid urbanization are causing a 'nutrition transition' (SCN, 2002; Shoeps *et al.*, 2011). Obesity is a risk factor for many diseases such as certain cancers, hypertension, type II diabetes mellitus, dyslipidemia, metabolic syndrome, and

coronary heart disease (Marshall *et al.*, 2004; Stunkard *et al.*, 1999). A rapid shift in the composition of diet (higher fat and lower carbohydrate), reduced activity patterns (Robinson, 2001), and a subsequent shift in body composition characterize this transition (SCN, 2002). Available data suggest that the problem of obesity begins in preschool children, becoming more evident among school-age children (Martorell, 2002). Television viewing and lack of physical exercise has been associated with increased risk of developing obesity among the school-age children with a 12% greater risk for every extra hour of daily television (Hernandez *et al.*, 1999).

2.3 Micro-nutrient Malnutrition

Micronutrient malnutrition or "hidden hunger" is common in populations that consume poorquality diets that lack in diversity as their habitual diet is often lacking these nutrients (Shetty, 2011). Diets deficient in micronutrients are relatively high in intakes of staple food and cereal crops, but low in consumption of foods rich in bioavailable micronutrients such as animal and marine products, fruits, and vegetables.

Globally, close to two billion people suffer deficiencies of micronutrients mainly iron, iodine, zinc, and vitamin A (Black *et al.*, 2008), commonly referred to as micronutrients of public health significance. Deficiencies of these micronutrients impair cognitive development and lower resistance to disease in children and adults (UNICEF/MI, 2004), and increase the risk of morbidity and mortality of both mothers and infants during childbirth and in early childhood. Additionally, micronutrient deficiencies impair the physical ability and economic productivity of adults and lower school performance in children (Victoria *et al.*, 2008).

The global prevalence of micronutrient deficiency is remarkably high and it is estimated that a third of the world's population fail to meet their physical and intellectual potential due to micronutrient deficiencies (UNICEF/MI, 2004). The most vulnerable groups are infants and children, women in reproductive age and the elderly.

2.3.1 Vitamin A Deficiency (VAD)

The term vitamin A designates a group of retinoid compounds with the biologic activity of alltrans-retinol. Retinoids usually consist of four isoprenoid units with five conjugated carboncarbon double bonds (Solomons, 2001).

Vitamin A was discovered in 1913 (Rosenfield, 1997) when experiments showed that if the only fat present in diets of young animals was lard, their growth was retarded, and when butter was substituted the animals grew and thrived. A substance in butter but not in lard was found also in egg yolk and cod-liver oil. It was named vitamin A. It was later established that many products of vegetable origin had nutritional properties similar to those presented by vitamin A in foods of animal origin; they were found to contain a yellow pigment, carotene, which is converted to vitamin A in the body.

Two forms of vitamin A are available in the human diet: preformed vitamin A (retinol and its esterified form, retinyl ester) and provitamin A carotenoids (Johnso& Russell, 2010; Ross, 2010). Preformed vitamin A is found in foods from animal sources, including dairy products, fish, and meat (especially liver). By far the most important provitamin A carotenoid is beta-carotene; other provitamin A carotenoids are alpha-carotene and beta-cryptoxanthin. The body converts these plant pigments into vitamin A. Both provitamin A and preformed vitamin A must

be metabolized intracellularly to retinal and retinoic acid, the active forms of vitamin A, to support the vitamin's important biological functions (Ross, 2006; Ross, 2010). Other carotenoids found in food, such as lycopene, lutein, and zeaxanthin, are not converted into vitamin A.

The various forms of vitamin A are solubilized into micelles in the intestinal lumen and absorbed by duodenal mucosal cells (IoM, 2001). Both retinyl esters and provitamin A carotenoids are converted to retinol, which is oxidized to retinal and then to retinoic acid (Ross, 2010). Most of the body's vitamin A is stored in the liver in the form of retinyl esters.

2.3.1.1 Vitamin A Status and Maternal and Child survival

Vitamin A status appears to be considered an important factor in childhood morbidity and mortality in areas where infectious diseases are responsible for substantial childhood morbidity and mortality, such as in the developing countries. In children, vitamin A deficiency has been associated with increased risks of mortality and morbidity from measles and diarrheal infections (Villamor & Fawzi, 2000), blindness (Whitcher *et al.*, 2001), and anemia (Semba & Bloem. 2002). Among women, it is associated with high mortality related to pregnancy (Christian *et al.*, 2000; West *et al.*, 1999). Studies have demonstrated that supplementation with vitamin A substantially reduces overall mortality attributable to diarrheal diseases in children by up to 23-30% (Rahmathullah *et al.*, 1990; Hussey & Klein, 1990; Coutsoudis *et al.* 1991; West *et al.*, 1991, Glasziou & Mackerras, 1993,). In Nepal, a double-masked, cluster-randomized, placebo controlled trial showed that weekly supplementation of women of reproductive age with normal dietary levels of vitamin A or β-carotene could reduce pregnancy-related deaths by 40 and 49%, respectively (West *et al.*, 1999). A case-control study nested into this trial found that night blind

pregnant women, who were vitamin A deficient, were more likely to have symptoms of urinary and reproductive tract infections, lower and upper gastrointestinal infections and preeclampsia/eclampsia than were control women (Christian *et al.*, 1998), suggesting that vitamin A deficiency during pregnancy may predispose women to pregnancy-related complications.

2.3.1.2 Vitamin A Status and Immune Function

Several trials (both *in vitro* and *in vivo*) using animal models and human subjects have revealed that Vitamin A plays an essential role in a large number of physiological functions that encompass vision, growth, reproduction, hematopoiesis, and immunity. A study to examine the effect of weekly vitamin A or β -carotene supplementation on self-reported pregnancy and postpartum illness symptoms and labor and delivery complications among Nepali women found that symptoms of nausea, faintness and night blindness were lessened in late pregnancy due to supplement receipt. The incidence of acute lower respiratory tract infection and vaginal bleeding was lowered by 30–50% in the third trimester. Both vitamin A and β -carotene reduced symptoms of lower gastrointestinal infections such as loose stools in the postpartum period based on either 7-day or 3-month recall data. β -Carotene supplementation also reduced the prevalence of high fever in the 3 month period after birth (Christian *et al.*, 2000). These results demonstrate that Vitamin A plays a key role in normal immune function (Semba, 1994) and deficiency leads to depressed humoral and cellular immunity and heightened morbidity from infectious diseases.

In animal models, vitamin A deficiency is associated with a shift from type 2 cytokines responsible for generating humoral immunity, antibody production, and immunoglobulin

maturation to predominantly type 1 cytokines which are necessary for cellular immunity and cytotoxicity (Cantorna, Nashold, and Hayes, 1994). Reifen *et al.* (2002) demonstrated that vitamin A deficiency induces inflammation, fibrosis and morphological and histological changes in the colon and that colitis is amplified by VAD and ameliorated by supplementation of the vitamin. These results were in line with earlier results reported by Sauer *et al.* (1995) and Swamidas *et al.* (1999) that there exists an inverse relationship between vitamin A status and the inflammatory response. Similarly, Al-Awadi *et al.* (2000) reported histological alterations in colonic mucosa and infiltration of lymphocytes and neutrophils around the crypts in the lamina propria in VAD mice. A study to examine the possible role of vitamin A deficiency in Common variable immunodeficiency (CVI) (Spickett *et al.*, 1998; WHO, 1995a), showed that decreased vitamin A levels may be of pathogenic importance in CVI, influencing immunoglobulin production and inflammatory reactions in these patients (Auskrust *et al.*, 2000).

2.3.1.3 Assessing of Vitamin A Status and Deficiency

Two commonly used methods in assessing vitamin A status in populations are clinical and biochemical assessments. Clinical assessment involves assessing for the presence of any signs of xeropthalmia. Xeropthalmia is a term that is used to refer to clinical spectrum of ocular manifestations of VAD, from milder stages of night blindness and Bitot's spots, to potentially blinding stages of corneal xerosis, ulceration and necrosis (keratomalacia) (Sommer, 1995). The stages of xerophthalmia are regarded both as disorders and as clinical indicators of VAD, and thus can be used to estimate an important aspect of morbidity and blinding disability as well as the prevalence of deficiency. However, the most assessed stages are night blindness and Bitot's

spots by use of history and hand light examination of conjunctival surface respectively (WHO, 2009a).

Biochemical methods of assessing vitamin A status include measuring serum retinol concentration. Serum retinol concentration reflects an individual's vitamin A status, particularly when the body's reserves of vitamin A are limited, because serum retinol concentration is homeostatically controlled and will not drop until body stores are significantly compromised (de Pee and Dary, 2002). Serum retinol concentration is also affected by factors that affect release of holo-Retinol Binding Protein (RBP) from the liver. Such factors include infection, protein status, adequacy of other nutrients and organ disease. In general, these factors lower serum retinol concentration (Arroyave et al., 1979). Serum retinol is usually assessed by high-performance liquid chromatography (HPLC) or spectrophotometry. Although spectrophotometry is much simpler and less costly, it is also much less accurate; therefore, HPLC analysis is preferred. Indirect methods such as the relative dose response and modified relative dose response tests are more sensitive. The tracer dilution technique is the only method that provides a quantitative estimate of total body vitamin A pool size. However, these tests cannot be widely used since they are expensive. Direct measurement of liver reserves of vitamin A status through biopsy is rarely an option and therefore has no utility globally. Therefore, serum retinol concentration continues to be widely used to assess vitamin A status, with values below a cut-off of 0.7µmol/L representing VAD (Sommer and Davidson, 2002) and below 0.35µmol/L representing severe VAD. A cut-off point of 1.05µmol/L has been proposed to be used for pregnant and lactating women (West, 2002).

Serum Retinol Binding Protein (RBP) is another biochemical marker that can be used to measure vitamin A status. Serum RBP occurs in a 1:1:1 M complex with retinol and transthyretin (Soprano and Blaner, 1994). Because of the 1:1 complex, serum RBP concentration should reflect serum retinol concentration and therefore might be substituted for it as an indicator of vitamin A status. Serum RBP concentration would also be a very good biochemical indicator for determining whether VAD is a public health problem, because it is much easier to analyze and it correlates well with serum retinol concentration.

2.3.1.4 Strategies for Addressing Vitamin A Deficiency

The time-tested strategies universally promoted to combat micronutrient malnutrition have hitherto focused on dietary modification, supplementation and fortification of commonly consumed foods with micronutrients. Dietary modifications have long been recognized, and recently re-emphasized (Recommendations of the Seventh IVACG Meeting, 1983) as the preferred major long-term solutions to controlling vitamin A deficiency in a population. The major dietary sources of provitamin A (primarily beta-carotene) include the multitude of dark green leafy vegetables, and deep yellow fruits, vegetables, tubers, and roots. Red palm oil is the richest source of carotenoids, containing 0.5mg/ml of mixed and beta-carotenes. Preformed vitamin A exists either as retinol or, more typically, as one of its esters, and is found in egg yolk, fish liver and oils, animal liver, and dairy products. A strategy to improve dietary intake of these foods may draw upon any combination of agronomic, horticultural, educational, and socioeconomic inputs (Arroyave et al., 1976). The common goal is always to improve availability of these food sources of vitamin A, and gradually increase and maintain an acceptable level of their intake among the vulnerable segments of the population.

Fortification of a widely consumed food "vehicle" with vitamin A offers a second major intervention strategy which can be relatively inexpensive (WHO, 1974) and effective in increasing vitamin A intake throughout the year, reflected by sustained elevations in serum vitamin A (Arroyave *et al.*, 1979) and reductions in xerophthalmia among the vulnerable members of a population. Vitamin A fortification of foods such as margarine and dairy products, now practiced in some many nations around the world, has provided a strong technological basis for extending the feasibility of vitamin A fortification to numerous other potential food vehicles.

Periodic oral delivery with a standard 200,000 IU (60,000 mcg) dose of vitamin A as retinyl palmitate, and nearly always with 40 IU of vitamin E (to improve absorption), comprises the third and most direct intervention strategy (WHO, 1982). The physiologic objective of periodic dosing is to maximize liver reserves from a single, large, oral dose of vitamin A while minimizing the risk of acute toxicity.

2.3.2 Iron Deficiency Anemia (IDA)

Anemia is a widespread public health problem associated with an increased risk of morbidity and mortality, especially in pregnant women and young children (WHO, 2002). Iron deficiency anemia results from a variety of causes including inadequate iron intake, high physiologic demands in childhood and pregnancy, and iron losses from parasitic infections. Iron is a component in many proteins, including enzymes and hemoglobin, the latter being important for the transport of oxygen to tissues throughout the body (NAS, 2002).

Iron deficiency anemia (IDA) – that is low levels of hemoglobin (Hb) in combination with abnormal levels of other iron indicators such as transferrin saturation (iron stores) – can lead to

weakness, poor physical growth, increased morbidity, and delayed psychomotor development. In particular, animal studies suggest that iron deficiency early in life could inhibit the function of neurotransmitters, compromising later brain function.

The prevalence of iron deficiency varies widely depending on the criteria used to establish the diagnosis. According to WHO (2011), anemia affects 1.62 people globally corresponding to 24.8% of the population.

2.3.2.1 Development of iron deficiency anemia

Iron deficiency (ID) anemia develops in three overlapping stages (Cook & Finch, 1989). The first stage is the iron depletion stage characterized by absence of iron stores without loss of functional iron. This stage is not associated with any adverse physiological consequences. This stage can be assessed by measuring the concentration of serum ferritin which provides a quantitative estimation of the size of iron stores in the liver, bone murrow, and spleen (Cook & Finch, 1979; Lynch & Green, 2001). Each 1µg/L serum ferritin indicates about 8 mg storage iron in adults (Bothwell *et al.*, 1979) and 0.14 mg in children (Finch & Huebers, 1982). When serum ferritin levels in adults fall below 12µg/L iron stores are considered to be depleted (Institute of Medicine, 2002). The second stage known as early functional ID or ID erythropoesis is characterized by marginally inadequate supply of iron to bone marrow and other tissues (Lynch & Green, 2001). During this stage haemoglobin concentrations do not fall below levels considered to be indicative of aneamia and is therefore also called ID without aneamia. This stage can be determined by measuring the saturation of serum transferrin, the main iron-binding protein responsible for the transport of iron in plasma (Brody, 1999), which decreases from 30-

35% to below 16%. Further, free erythrocyte protoprophrin concentrations greater than 70μg/dL of erythrocytes indicate that the formation of heme from protoporphrin IX is impaired due to insufficient iron supply. Lastly, the lack of iron leads to an increase in transferring receptors on surface of all tissues resulting in soluble serum trasferrin receptor concentrations above 85mg/L (Institute of Medicine, 2002). The final stage of ID is iron deficiency aneamia (IDA), which is diagnosed based on hemoglobin concentrations below 120g/L in non pregnant women above 15 years of age and 110g/L in pregnant women and children aged between 6-59 months. Children aged 5-11 years and 12-14 years are considered to suffer from IDA when their Hb levels drop below 115g/L and 120g/L respectively. The critical levels for diagnosis of aneamia in men aged 15 years and above are slightly higher (130g/L) than all the other age groups (WHO, 2011).

2.3.2.2 Assessing anemia status

WHO/CDC (2004) expert consultation recommended addition of transferrin receptor in addition to hemoglobin and serum ferritin for assessment of iron status in places where infection is common. In situations with high prevalence of anemia a) clinical assessment, b) hemoglobin estimation and c) response to iron supplementation may suffice.

Clinical assessment: in moderate to severe anemia (WHO/CDC, 2004; Adaman and Dan., 2005)

-i) significant pallor of eyelids (difficult in children due to frequent conjunctivitis), tongue, nail
beds and palms (pale palm creases suggest severe anemia; ii) fatigue, low exercise capacity (mild
anemia can produce decreased exercise tolerance); iii) fissures at the corner of the mouth suggest
anemia; iv) nails, show pallor, flatness, softness to feel and later become spoon shaped
(koilonychia). It is rare in children < 6 yr of age, as hemoglobin is sacrificed to maintain tissue

growth; v) children develop irritability, pica for ice/mud/coal/substances containing lead; vi) findings of congestive failure indicate severe anemia, hemoglobin below 5.0g/dl; vii) iron deficiency affects mental functions (Agarwal., 2001) i.e. attention span, alertness and learning, and viii) deficiencies of vitamin B12 and folic acid are associated with psycho-neurological changes and pigmentation of mucous membranes and distal parts of the body. These clinical features are of diagnostic assistance for the individual patient with severe anemia to seek advice from a medical expert. However, clinical features fail to assess degree of anemia and therapeutic response. Thus, hemoglobin estimation is the criterion.

Normal hemoglobin and hematocrit distribution varies with age, gender, at different stages of pregnancy and with altitude and smoking. Given the wide range of hemoglobin concentrations in the population as a whole, an individual can have a substantial decrease in hemoglobin with the value still in the normal range.

Hemoglobin and hematocrit: can be used to assess anemia in community where prevalence is of public health significance (WHO/CDC, 2004). The problem arises in capillary blood collection the technician should be very careful. To obtain the best possible skin puncture sample, it is important to warm the extremity in order to facilitate a free flow of blood and to avoid any squeezing of the finger (WHO/CDC, 2004).

2.3.2.3 Causes of iron deficiency

Iron deficiency develops when the amount of iron absorbed from the diet is lower than the amount of iron needed to cover the physiological iron requirements. Consequently, the population groups most at risk of developing ID are those with the highest iron requirements.

These are infants, children, adolescents, and pregnant women who have additional iron needs due to growth as well as women of childbearing age who have higher iron losses due to menstrual blood loss.

Inadequate iron absorption can be caused by multiple factors. Low dietary iron intake is one of the reasons and can result from energy restriction (Hallberg, 2001) or a diet low in iron (Bothwell et al., 1989). There are two broad types of dietary iron; about 90% of iron from food is in the form of iron salts and referred to as non-heme iron. The extent to which this type of iron is absorbed is highly variable and depends both on the person's iron status and on the other components of the diet. The other 10% of dietary iron is in the form of heme iron, which derived primarily from the hemoglobin and myoglobin of meat. Heme iron is well absorbed, and its absorption less strongly influenced by the person's iron stores or the other constituents of the diet. There is little meat in the diet of most infants; therefore, most of their dietary iron is nonheme, and their intake is highly influenced by other dietary factors. Ascorbic acid enhances the absorption of non-heme iron, as do meat, fish, and poultry (Derman et al., 1980). Inhibitors of absorption include bran, polyphenols, oxalates, phytates, vegetable fiber, the tannins in tea, and phosphates (Charlton & Bothwell, 1989). Heme iron itself promotes the absorption of non-heme iron. For example, adults absorb approximately four times as much non-heme iron from a mixed meal when the principal protein source is meat, fish, or chicken than when it is milk, cheese, other dairy products, or eggs. Dietary quality, based on the content of enhancers and inhibitors of iron absorption in the diet, is frequently dependent on socio-economic factors (Taylor et al., 1995).

Iron deficiency can further result from increased blood loss or decreased iron absorption due to pathological causes. For example, hookworm and schistosomiasis can further cause heamturia (WHO, 1991; Stephenson, 1993; WHO, 1993). Pathological reasons for impaired iron absorption include achlorhydria and gastric surgery (Hallberg *et al.*, 1993b). Although the mechanism is not fully understood yet, Barabino (2002) showed that the gastric *Helicobacter pylori* causes IDA.

2.3.2.4 Prevention of Iron Deficiency

Strategies to prevent iron deficiency and iron deficiency aneamia include supplementation, dietary modification/education and food fortification. Supplementation with iron tablets is the most widely used approach to control iron deficiency (Allen, 2002a). Iron supplements can be used to treat individuals with ID/IDA, or routinely to prevent ID/IDA in population groups at high risk of developing ID/IDA, such as pregnant women, infants, and young children. However, while iron supplementation has an advantage of enabling the targeting of high-risk population groups, it has major disadvantage of requiring an effective system of health care delivery.

Dietary modification is the other strategy that has been employed to address ID and IDA. Such strategies include food-based interventions, for example, promotion of food processing techniques which decrease the content of iron absorption inhibitors in the diet, for example phytic acid content by soaking, germination, or fermentation of whole grain cereals and pulses. Further, increasing the intake of foods and beverages rich in enhancers of iron absorption with meals containing non-heme iron is assumed to improve iron status by increasing iron absorption. Similarly, decreasing the intake of foods and beverages containing inhibitors is also expected to

improve iron status. Interestingly, cooking in iron pots is also promoted in order to increase the iron content of the diet (Ruel & Levin, 2001).

The third strategy, food fortification with iron compounds, is generally considered the best-long term approach to prevent iron deficiency (Cook & Reusser, 1983). The advantage of food fortification is that it reaches most segments of the population and does not require the cooperation of the individual.

2.4 Common Health Problems in Childhood

The poor, particularly children in low-income countries, carry the greatest burden of morbidity and mortality. Much of this burden results from hazards within their homes or their immediate environment (Cairncross *et al.*, 1996). High levels of malnutrition, and its known synergistic relationship with immune function exacerbate their vulnerability to disease particularly diarrhoeal disease, helminth infections, acute respiratory infections (ARIs) and malaria. For the urban poor in low-income countries, there is a double jeopardy as they find themselves exposed to both "traditional" diseases of poor sanitation and overcrowding and the "modern" diseases of chronic heart and lung disease (SCN, 2002).

Diarrrheal diseases are primarily a symptom of intestinal infections caused by a range of viral, bacterial, and parasitic organisms. If severe or persistent, the fluid loss and dehydration associated with diarrhoea may be life threatening, especially in infants and young children, the malnourished, or people with impaired immunity. WHO estimates that 3.3 million children die from intestinal infections such as cholera, typhoid or infectious hepatitis every year. The total number of diarrhoeal episodes may be as high as 4000 million (WHO, 1996).

Parasitic helminth infections are a major public health problem in developing countries affecting all age groups. The prevalence of these infections rises to a maximum in childhood and settles to a stable asymptote in the adult population (SCN, 2002). However, for the age-intensity (or worm burden) profile, there is a marked convexity, such that it is the school-age child who has the most intense infections (the greatest worm burden). As morbidity is directly related to the intensity of infection, it follows that it is this age group who is particularly susceptible (Bundy et al., 1992). World Bank (1993) estimated that for girls and boys aged 5-14 years in low-income countries, intestinal worms alone accounted for an estimated 12 and 11% respectively of the total disease burden of this age group, making this the single largest contributor to the disease burden of this group. Children with chronic worm infections and large numbers of worms may be stunted and underweight, which can lead to long-term retardation of mental and physical development, and even death in severe cases. Moreover, worms can cause malnutrition through inappropriate nutrient utilization and malabsorption and aneamia through loss of blood. An inverse relationship between infection intensity and heamoglobin levels have been shown by a number of studies (Lwambo et al., 1992; Brock & Cammish, 1997; Beasley et al., 1999).

Malaria is probably the world's most important parasitic infectious disease, occurring in tropical and subtropical countries. It is caused by four species of Plasmodium parasite; *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale* and *Plasmodium malariae*. Malaria can cause iron deficiency and anaemia (SCN, 2002) through haemolysis, erythrophagocytosis and dysentrythropoiesis (Means, 1994).

2.5 Knowledge Gaps

There is increasing evidence, with resulting international concern, that there exists high level of nutritional deprivation combined with the heavy burden of disease in school-age children which has negative consequences for a child's long term overall development. This has prompted an increased focus on the diverse needs of the school-age child. A clear understanding and awareness of the heavy burden of malnutrition and disease among school-age children is of great importance. However, the true picture and extent of the burden is still lacking since not much is documented in detail yet. This study was designed to address this gap by providing information on the nutrition status, dietary diversity and morbidity burden among schoolchildren.

CHAPTER THREE: METHODOLOGY

3.1 Introduction

This chapter reviews the description of the study setting in terms of geographical location, the topographical features, climatic conditions and the demographical and socio-economic features of the population. The chapter also reviews the research methodology employed: sample size determination, sampling procedures and data collection tools and procedures. In addition, this chapter includes data quality and analytical methods employed.

3.2 Study Setting

3.2.1 Geographical Location of the Study Area

The study was done in three primary schools in rural Kibwezi District (Annex 7), one of the dryland Districts in South Eastern Kenya, comprising Kibwezi, Makindu, Machinery and Mtito Andei Divisions. The district borders Kajiado District to the West, Taita District to the South, Mutomo District to the East and Nzaui and Makueni Districts to the North. The District covers an area of 3954.6 km² and lies within longitude 37° 49° 11.14" East and 38° East and latitude between 2° 17° 11.21" South and 2° South (TAWSB, 2011)

3.2.2 Topographic Features

The district lies within an arid zone rising from 300 m above sea level at the lowlands of Mtito Andei to 1100M above sea level on Chyulu Hills. These hills form the main land features in the district. They are of volcanic rocky formation and are situated to the Western side of the District. There is also the smaller Mbui Nzau Hill on the northern side. The rest of the district consists

mainly of low-lying grassland, which receives little rainfall but has an enormous potential for ranching and/or livestock keeping (TAWSB, 2011).

3.2.3 Climatic Conditions

The district has two rainy seasons: March/April Rains and November/December rains. The November/December rains are the most reliable in this region though nationally these are the short rains. The average rainfall is 351.9 mm to 687.4 mm per annum. The evapo-transpiration rate is recorded at 2000mm per year. The district is prone to frequent droughts and the only economic activity the community engages in is mainly agro-pastoral activities as well as small-scale irrigation along the permanent rivers Kiboko, Kibwezi and Athi River (ACF International, 2011)

3.2.4 Demographic and Socio-economic Features

Kibwezi District has an average population size of 248,704 persons of whom 123,069 are male while 125,635 are female. The district has approximately 52,979 households with an average household size of 5 persons, while the average human population density is 63 persons per km² (KNBS website, accessed on 07/05/2013).

Poverty levels are very high in the district with 64.3% of the population living below the poverty line and a food poverty level of 57.2% (GoK, 2009). The crude death rate (CDR) in the district stands at 13.9 deaths per 1000 persons while under-five mortality rate is 84 deaths per 1000 live births (GoK, 2009).

The district has 213 primary schools and 55 public secondary schools with a net primary enrolment rate of 66.52 percent while the drop-out rate in secondary schools is recorded at 1.4% from an enrolment of 10,371 students (GoK, 2009). In terms of health facility endowment; the area is served by one District Hospital, four Health Centres, and 18 Dispensaries. Of importance is the HIV/AIDs prevalence which stands at 9% (GoK, 2009).

The district thrives on its natural resource-base for the following activities: agriculture, mining and quarrying. The resources available in the district include: land, water, forestry, wildlife, minerals, sand, limestone, granite, gypsum, quartz and coal. Access to water has remained elusive due to under exploitation of the resource in a sustainable manner. The coverage of the water supply stands at 40 percent with about 25 percent of the population covered by a piped water line. The average distance to the nearest water source during the rainy season is 3.5 km while during dry spell it increases to 8 km (ACF International, 2011). In the recent past the district started experiencing increased sand harvesting, quarrying activities, deforestation, and charcoal burning and overgrazing resulting in destruction of the water catchments (GoK, 2009).

3.3 Research Methodology

3.3.1 Study Design

This study assumed a cross-sectional design with some retrospective components. Both descriptive and analytic components were also employed in the study.

3.3.2 Study Population

The study focused on schoolchildren aged 5 years to 15 years currently enrolled in three primary schools namely Thange, Muusini and Kithasyu Primary Schools. Apparently healthy children aged 5-15 years enrolled in the schools at the time of study were all eligible to participate. However, only those children whose parents/caregivers gave a written consent were included in the study. Prior to the study, the children were tested for malarial parasites and deworming drugs administered to them. Children found to be positive for malaria were treated before being recruited into the study.

Eligible children with a history of infectious or systemic diseases (e.g. tuberculosis, sickle cell anaemia) were also excluded from the study. Pupils in standard eight were also excluded from the study since they were doing examinations at the time of study.

3.3.3 Sample Size Determination

A representative sample size was determined using a formula as in the box below (Fischer et al.,

1991).

$N=z^2pq/d^2$

Where \mathbf{n} = the desired minimum sample size

Z= the standard normal deviation which is 1.96 at 95% confidence interval

P= prevalence of VAD estimated at 50%

 \mathbf{q} = 1-p (proportion of school-age children with adequate vitamin A status (0.72).

d= the degree of accuracy desired set at 5%

Therefore,

 $\mathbf{n} = (1.96)^2 (0.5 \times 0.5)$

 0.05^{2}

=384 children

With a 10% attrition rate (38 children), the minimum sample size was 422 children.

3.3.4 Sampling Procedure

This study was part of a randomized controlled study designed to measure the effect of provitamin A biofortified cassava on vitamin A status of primary school children in Kibwezi district (CassavitA project). Primary school children were chosen as study subjects since they did not receive routine vitamin A supplementation by government regulation (unlike children <5 years). Moreover they were relatively easy to target with a food-based intervention study from a logistical point of view. Three primary schools from Kibwezi district were selected for the intervention study based on the prevalence of VAD, size of the schools and willingness to participate. A sampling frame of children from the three schools was prepared from which the representative sample for the study was selected. Caregivers of the sampled children were called to school for administration of the questionnaire.

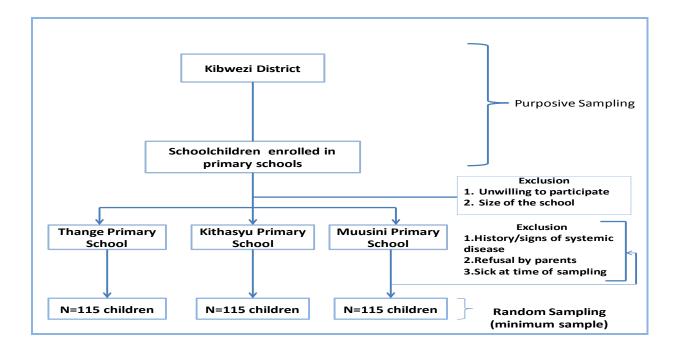


Figure 1: Sampling procedure flow diagram

3.4 Data Collection

3.4.1 Data Collection Tools

Four semi-structured questionnaires (Annex 2-5) were used to collect the necessary information from the caregivers of the children. Before administration, the questionnaires were translated to the local language then back translated to the original language. Pre-testing of the questionnaires and the results of the pretest used to modify the tool to collect the information required.

3.4.2 Recruitment and Training of Research Assistants

Three research assistants from the area with a minimum C+ (plus) in Kenya Certificate of Secondary Education (KCSE), fluent in the local dialect (Kikamba) and of good moral behavior were recruited to administer the questionnaires. They were adequately trained on administration of the questionnaire. During the training, demonstration on taking anthropometric measurements and role-play as well as standardization of messages was ensured (Annex 6).

3.4.3 Data Collection Procedures and Methods

3.4.3.1 Data quality assurance

Data quality was ensured by training of enumerators, calibration of equipment, onsite data cleaning, field supervision by principal investigator, pretesting of the questionnaire and use of qualified staff for collection and analysis of the blood samples.

3.4.3.2 Demographic, socio-economic and morbidity data

A semi-structured questionnaire was used for collecting data on demographic and socioeconomic characteristics of the study households such as the household size, marital status, sex and level of education of the caregiver. A fourteen-day recall period of common childhood illness such as diarrhea, cough, fever etc was used to gather information on morbidity incidence among the children.

3.4.3.3 Dietary diversity data

Data on individual dietary diversity was collected using a sixteen-food group dietary diversity questionnaire based on a twenty-four hour recall period (FAO, 2011). Questions were directed to the mothers or primary caregivers, but the target child was also present during the interview to assist in indicating foods eaten outside the home. Subjects were asked to mention all the foods and beverages eaten during the preceding 24-hours (from the time they woke up the previous day up to the time they woke up again on the day of interview).

The individual dietary diversity was determined by summing all the food groups consumed by the child. Nine food groups (Starchy foods, legumes, fruits and vegetables, dark green leafy vegetables, vitamin A rich vegetables and fruits, milk, meat and fish, eggs and organ meats) were used in the analysis.

3.4.3.4 Anthropometric data

Weight and height were measured according to WHO guidelines (de Onis *et al.*,, 2004). Weight was measured to the nearest 0.1 kg with an electronic Seca® scale (Seca GmbH & Co. KG,

Hamburg, Germany) with children wearing only light clothing and without shoes. Two measurements were taken and the mean value used in the analyses. If the difference between the two measures exceeded 1kg, a third weight was taken. The scale was calibrated after every 20 measurements.

Height of the children was measured using a Seca stadiometer (Seca GmbH & Co. KG, Hamburg, Germany). The stadiometer was placed on a flat ground with the base then the subject asked to stand against it with feet together (without shoes) on the base. The shoulders, the buttocks and the heels had to touch the vertical rod. The children standing with their eyes in the Frankfort horizontal plane, the height was measured to the nearest 0.1 cm and recorded twice. Similarly, when the difference between the two measures was higher than 0.5 cm, a third measure was taken and the mean of the two closest values was used in the analyses.

Age of the children was estimated from their birth certificates, clinic cards, baptism cards, school records, calendar of events or recall by the caregiver/child.

The Weight and height measurements were converted to BMI-for-age, weight-for-age and height-for-age using WHO AnthroPlus for personal computers (v 1.0.4). The indices were expressed as z-scores using the international reference population (WHO, 1995b). Children were classified as stunted, thin or underweight if the respective z-scores fell below -2SD of the reference population for the age and sex (WHO, 2009b). Weight-for-age z-scores were computed for children aged 5-9years only since Anthroplus couldn't determine the WAZ for children older than 9 years.

3.4.3.5 Biochemical samples collection and handling

A 10mL blood sample from venipuncture was taken from each child by technicians using BD contact activated lancets (BD Microtainer). The blood sample was collected in BD pink microtainers with EDTA and flow tops connection, immediately stored at 4° and shielded from light. On the same day, the blood was analysed for haemoglobin, malaria, and C-reactive Protein (CRP) respectively. Afterwards, the blood cells were separated from serum using a desktop centrifuge (3000 rpm, 10 minutes, room temperature) in a field laboratory, and 0.2 ml aliquots of serum were placed in liquid nitrogen for shipping and further analysis in the laboratory of the Division of Human Nutrition, Wageningen University, the Netherlands.

a. Serum Retinol Binding Protein Concentration

RBP serum as a proxy indicator of serum retinol to determine vitamin A status was measured by a highly sensitive two-site enzyme linked immunoassay (ELISA) (K-Assay, KT 504, Seattle, US). An iMark Microplate Absorbance Reader connected to an external computer by a USB interface was used to read the microplate absorbance. The results were expressed in nanograms per millilitre (ng/ml). Intra-assay variation was determined by measuring RBP level of samples in duplicate to confirm that the measurement was valid. Furthermore, inter-assay variation was also determined by measuring the same quality control samples over 10 consecutive assays.

Data on serum RBP was used to calculate the mean and standard deviation. The children were put into tertiles; lower, medium and high using the serum RBP.

b. Hemoglobin Determination and Diagnosis of Anemia

Haemoglobin from $20\mu l$ blood was measured using the HemoCue machine (HemoCue HB 201, Angelholm, Sweden) on battery power. The quality of hemoglobin measurements were ensured by measuring a normal value (7.5 \pm SD 0.4 mmol/gram) and a low value (5.0 \pm SD 0.2 mmol/gram) Certified Reference Material (HB Eurotrol) before and after hemoglobin sample measurements.

Age-specific criteria (Table 1) were used to determine the prevalence of anemia in the two age groups; 5-11 years and 12-14 years (WHO, 2011).

Table 1: Classification of IDA

Age Group	Aneamia					
	Non-aneamia	Mild	Moderate	Severe		
5 -11 years	≥115g/L	110-114g/L	80-109g/L	<80g/L		
	(≥7.1mmol/L)	(6.8 - 7.0mmol/L)	(5.0 - 6.8mmol/L)	(<5.0mmol/L)		
12- 14 years	≥120g/L	110-119g/L	80-109g/L	<80g/L		
	(≥7.3mmol/L)	(6.8 – 7.3mmol/L)	(5.0 - 6.8 mmol/L)	(<5.0mmol/L)		

Adopted from WHO, 2011

3.5 Ethical Considerations

The CassavitA study protocol of which the current this study was part of was approved by National Ethical Review Committee of Kenyatta National Hospital and the University of Nairobi and was conducted with the permission of the District Commissioner and his respective officers.

Written informed consent form was sought from the parents/caregivers of the children (Annex 8). Verbal consent was obtained from the children. Permission was obtained from the school administration before carrying out the survey in the school. While in the field, the data collectors observed appropriate professional ethics through their dressing mode and language. The subjects were assured of confidentiality of the information

3.6 Statistical Analysis

Data was entered in MS Access as soon as they become available. Subsequent analysis was done using Statistical Package for Social Scientists version 16 (SPSS Inc). Data from open-ended questions were pre-coded before entry.

Descriptive statistics (means, percentages, standard deviations and range) were computed for demographic and socio-economic data. Analyses were stratified by age, sex, school, dietary diversity and other characteristics. A p-value of <0.05 was considered significant in all the analyses. Chi-square and independent t-tests was used for comparison of prevalence anemia, low RBP and malnutrition between different groups. Analysis Of Variance (ANOVA) was used to compare the means of three or more groups.

A bivariate correlation analysis was done to identify factors that were associated with the independent variables Hb, RBP, HAZ, WAZ, BAZ, IDDS and Morbidity status. Binary logistic regression were performed in order to establish the relationship between Hb, HAZ, WAZ, BAZ, serum RBP and different variables. Odds ratios were used to estimate the relative risk of anemia and low RBP levels in the different gender and age categories.

Students t-tests and Analysis of Variance (ANOVA) were used to compare the means for significant difference between the different categories.

CHAPTER FOUR: RESULTS

4.1 Introduction

This chapter presents results from the study. The results are organized as per the specific objectives of the study and are based on analysis of data from on 423 schoolchildren aged 5-15 years old from 398 households. Among the three schools, Thange primary school had the highest number of children in the study (167, 39.5%)) followed by Kithasyu primary school (133, 31.4%)) then Muusini primary school (123, 29.1%)) in the second and third position respectively.

4.2 Socio-demographic and Economic Characteristics of the Study Population

4.2.1 Socio-demographic Characteristics of the Study Households

Table 2 shows the socio-demographic characteristics of the households studied. The mean household size was 7 people (SD 2.2). The smallest household had 3 members while the largest household had 16 members. Most (64.1%) of the households had more than 7 members. Children aged below 15 years constituted the greatest proportion of the population (48.8%) while those aged above 65 years constituted the smallest proportion (4%). The dependency ratio of the study population was 149. Majority (85.6%) of the households studied practice monogamous type of marriage. All the households had access to a toilet facility. The most common type of toilet facility was pit latrine without slab (77.9%). More than one-third (35.3%) of the households shared the toilet facilities with other households. On average, one toilet facility was shared by at least 3 households.

Table 2: Socio-demographic characteristics of the study households

Characteristics (N=398)		%
Household size	≤7 members	64.1
	>7 members	35.9
Treat water	Yes	25.9
	No	74.1
Type of toilet	Flush toilet	0.9
	Ventilated Improved pit latrine	3.1
	Pit latrine with a slab	18.1
	Pit latrine without a slab	77.9
Main source of drinking water	Rainy season (%)	Dry season (%)
Piped water	6.2	11.6
Tube well/borehole	25	43.8
Dug well protected	4.3	6.9
Dug well (unprotected)	7.6	10.7
Surface water	14	26.9

Tube well/borehole was the main source of water during both the rainy season and dry season. Only 25.9% of the households treated their drinking water before use. Al most one-half (49.1%) of the households took less than 30 minutes to get their drinking water from the source. Only 0.7% of the households had water within the premises. The rest (50.2%) travelled for 30 minutes or more to collect water from the source.

4.2.2 Socio-economic Characteristics of the Study Population

The main source of income for most households in the study was casual labor (44.7%) while the least common income source was destitute (gifts/begging) (1.2%) Both crop and livestock farming were important economic activities in the area. Although most of the households (97%) practice crop farming, only 6.8% of them had food from their farm at the time of study. 76.3% of the households studied keep livestock with poultry being reared by most (62.5%) followed by goats (56.2%), cattle (26.7%) and sheep (7%). Table 3 shows the socio-economic characteristics of the study households.

Table 3: Socio-economic characteristics of the study households

Characteristics of households	% (N=398)		
Own livestock	76		
Producing own food	91.4		
Having adequate food to last till next harvest	10.2		
Owning some means of transport			
Owning at least one mobile phone			
Household main source of income			
✓ Casual labor	44.9		
✓ Crop sales	22.5		
✓ Salaried employment	13.2		
✓ Trade	12.3		
✓ Remittances	4.5		
✓ Animal/animal product sale	1.4		
✓ Destitute/gift/begging	1.2		

4.2.3 Socio-demographic Characteristics of the Respondents

Three hundred and ninety eight (398) respondents were interviewed during the study. Of the respondents interviewed 84.7% while only 15.3% were males. Majority (52.2%) of the respondents were aged 20-35 years. Most of the respondents (58.4%) had completed 5-8 of primary school while only 3.3% had attained above secondary level of education. Table 4 shows the socio-demographic characteristics of the respondents.

Table 4: Selected Socio-demographic Characteristics of the respondents

Characteristics	Percent (N=398)
Relationship to the child	
Mother	69.7
Father	10.4
Grandmother	10.4
Others (uncles/sister/aunt)	9.5
Age category	
<20 years	1.2
20-35 years	52.2
36-50 years	34.5
50+ years	12.1
Educational levels	
None	6.9
Completed 1-4 of primary school	8.5
Completed 5-8 of primary school	58.4
Attended Secondary School	9.2
Completed Secondary School	11.8
Above Secondary School	3.3
Others (Adult education, Vocational training)	1.9

4.2.4 Characteristics of the Study Children

The study covered 423 pupils (52.2% males and 47.8% females) from three primary schools in Kibwezi district. The mean age of the study children was 9.3 years (SD 2.3). The oldest was 15 years while the youngest was 5 years old. By birth order of the children, 48% were first or second born in their households while the rest (52%) were third born or higher. The greatest share of the study children were from lower primary school especially standard one (18%) and st two (18.7%) as shown in figure 2.

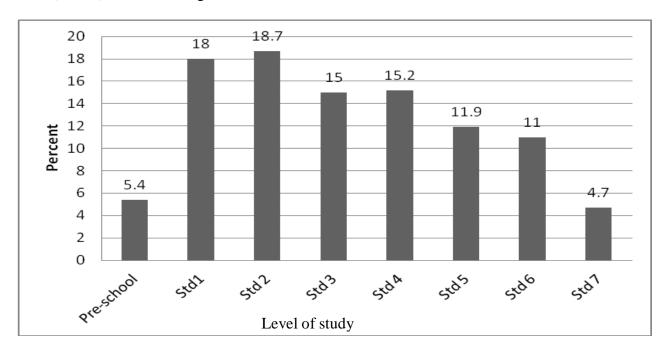


Figure 2: Distribution of study children by level of study

4.4 Vitamin A malnutrition among the Study Children

The mean RBP was 5.15 ± 1.48 ng/ml while the median range were 4.90ng/ml and 10.22ng/ml respectively. The lowest RBP reading was 1.429ng/ml while the maximum value was 11.656ng/ml. Table 4 below shows the mean RBP values by age group of the children. Younger children (5 -11 years) had a significantly lower mean RBP than that of older children (11 - 15 years) (p= 0.001).

Table 5: Mean serum RBP by age groups

Age (years)	Mean	SD	Student t-test
5 -11	5.04	1.4	F ₄₂₀ =11.99,
12 - 15	5.70	1.8	p=0.001
All	5.14	1.5	

A comparison of mean RBP by school showed that Kithasyu primary school had a significantly higher mean serum RBP from the other two schools the three schools ($F_{2,419}$ =3.52, p = 0.030)

Most (60.7%) of the children had serum RBP measurements less than the mean serum RBP. There was no significant difference (P=0.113) between the prevalence of low RBP (serum RBP levels below the population mean serum RBP) between boys (14.9%) and girls (9.5%). The relative risk of having serum RBP value of below the 50th percentile for male and females was 0.781 and 1.317 showing that girls were about twice likely to fall below the 50th percentile. Children consuming <5 food groups were 1.45 times likely to fall below the 50th percentile for serum RBP than those consuming >5 food groups (OR = 1.45, 95% CI: 0.638 to 3.294). Children

who consumed foods from vitamin A rich groups had significantly higher mean serum RBP values (mean=1.60ng/mL) than those who did not consume foods from vitamin A rich foods (mean= 1.40ng/mL), (p=0040). There was a significant difference in the distribution of the children in the 3 tertile groupings (p=0.001) by school. Kithasyu primary school had the greatest number of children in the lower quartile (39.4%).

Table 6: Distribution of the study children by the RBP percentile groups and schools

School	Lower tertile (%)	Medium tertile (%)	High tertile (%)
Muusini (n=123)	34.1	22.8	43.1
Kithasyu (n=132)	39.4	39.4	21.2
Thange (n=167)	27.5	36.5	35.9
All (N=422)	33.2	33.4	33.4

Univariate analysis showed that age of the child, age of the care giver, main source of the household income and ownership of toilet facility were significantly associated with serum RBP levels (p=0.003, $R^2=0.234$). (Annex 10).

Table 7 shows the distribution of the children by the RBP percentile groups. There was a significant difference in distribution of the children in the different percentile groups between the older and younger children (p=0.013).

Table 7: Distribution of the children by the RBP percentile groups and selected variables

		Perce	Percentile Group of RBP			
		Lower tertile	Medium tertile	High tertile	$(\chi 2)$	
Age group	5-11 years (n=351)	35.3	34.2	30.5	0.013	
	12-15 years (n=71)	22.5	29.6	47.9		
Thinness	Thin (n=85)	41.2	36.5	22.4	0.068	
	Normal (n= (n=183)	31.1	32.8	36.1		
Stunting	Stunted (n=75)	38.7	29.3	32.0	0.517	
	Normal (n=346)	32.1	34.4	33.5		
Underweight	Underweight (n=121)	38.0	32.2	29.8	0.394	
	Normal (n=300)	31.3	34.0	34.7		
IDDS	Low DDS($n=254$)	33.5	32.3	34.3	0.688	
	High DDS (n=168)	32.7	35.1	32.1		
Anemia	No anemia (n=379)	32.2	34.6	33.2	0.339	
	Anemia (n=42)	40.5	23.8	35.7		

4.5 Prevalence of Anemia among the Study Children

The mean Hb level was 8.0 ± 1.2 mmol/L and 8.2 ± 1.1 mmol/L for age groups 5-11years and 12-14 years respectively and 8.2 ± 1.2 mmol/L and 8.0 ± 1.2 mmol/L for the male and female children, respectively. Table 8 shows the mean Hb of the children by levels of study. There was no significant difference in the mean Hb levels of the children in different levels of study (F $_{7}$, $_{414}=1.121$, p=0.349). In all the study levels, there was no significant difference in the mean Hb levels between the male and female children (p>0.05).

Table 8: Mean hemoglobin levels of the children by levels of study

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-school	24	6.50	10.90	7.9	1.0
Standard 1	73	6.10	12.10	8.0	1.3
Standard 2	79	4.40	12.80	8.1	1.3
Standard 3	64	5.70	12.00	7.9	1.1
Standard 4	64	6.60	14.10	8.2	1.2
Standard 5	51	6.60	12.30	8.5	1.2
Standard 6	47	6.80	11.60	8.1	0.8
Standard 7	20	7.00	9.20	8.1	0.5
All levels	422	4.40	14.10	8.1	1.2

Overall, the mean Hb was 8.1 ± 1.2 mmol/L (Table 8). Anemia was detected in 10% of the children of which 4.7% were mildly anemic, 5.0% moderately anemic and only 0.3% severely anemic. No cases of severe anemia were detected among the older children (12-14years) (Table 9).

Table 9: Prevalence of anemia among study children by age and gender

		5-11 years			12-14 years		
Gender	Normal	Mild	Moderate	Severe	Normal	Mild	Moderate
	(%)	(%)	(%)	(%)	(%)	(%)	(%)
Male (n=202)	90.9	4.8	3.6	0.6	78.4	8.1	13.5
Female (n=220)	90.3	4.9	4.9	0	97.1	0	2.9
Total (N=422)	90.6	4.9	4.3	0.3	87.5	4.2	8.3

At 5% confidence level there was no significant difference in the prevalence of anemia between the male and female children (χ^2 =0.888, df = 2, P =.346) as well as between the older (12-15yeras) and younger children (5-11 years) (χ^2 =.629, df = 1, p =.428). Non-anemic children were less likely to fall below the 50th percentile of serum RBP than anemic children were (OR = 0.876, 95% CI: 0.463, 1.658). Table 10 shows the relative risk of a child being anemic in relation to various variables. A bivariate correlation analysis showed no significant correlation between the IDDS and serum Hb (r=0.010, p=0.846)

Table 10: Odds ratios (ORs) of occurrence of anemia under different exposure factors

		CI (95%)	
Variable	OR	LCL	UCL
IDDS (<5/≥5groups)	0.957	0.358	2.558
RBP (<50 th percentile/>50 th percentile)	0.876	0.463	1.658
Underweight (Yes/No)	2.178	0.940	5.048
Stunted (Yes/No)	0.929	0.412	2.097
Thin (Yes/No)	2.000	0.786	5.092
Sick in last 2 weeks (Yes/No)	1.827	0.745	4.478

4.6 Nutritional Status of the Study Children

The mean weight for the children was 23.8 ± 6.7 kg with the lightest child weighing 13kg while the heaviest child weighed 54.5kg. The mean height of the children surveyed was 127.4 ± 12.7 cm. The tallest child was 164.5cm while the shortest was 97.5 cm. As shown in figure 5 of the three nutritional indicators, underweight had the highest prevalence (31.7%) followed by thinness (28.9%) and stunting (18%). There was no significant association between the sex of a child and the nutritional status ($\chi^2 = 0.444$, df = 1, p =0.504).

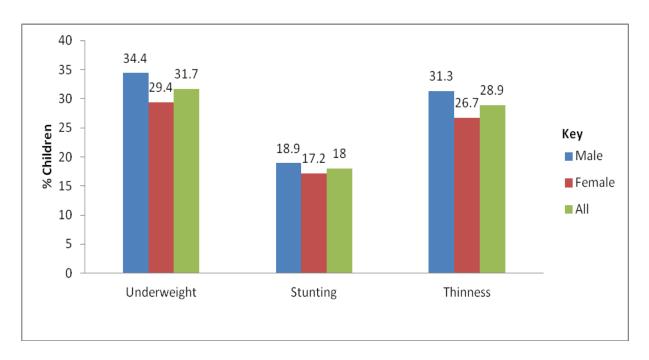


Figure 3: Prevalence of macronutrient malnutrition among the children

4.6.1 Weight-for age (Underweight)

Underweight was computed only for children aged below 10 years old (N=260). Overall, 31.7% of the children were underweight. 5.6% of the children were severely underweight while 26.1% were moderately underweight (Table 11). Children aged 6 years old had the highest prevalence of severe underweight (9.3%). The results show that the prevalence of underweight increased with age with the children who were 8 years old having the highest prevalence of underweight (40%). The mean weight-for-age z-score for the children was -1.18 (SD, 0.89).

Table 11: Distribution of the children by weight-for-age Z score

	N			Weight	-for-age		
Years		%<-3SD	(95% CI)	%<-2SD	(95% CI)	Mean z-score	SD
							0.8
All	260	5.6	(1.9%, 7.4%)	31.7	(20.1%, 32.3%)	-1.18	9
							0.8
5	33	0	(0%, 1.5%)	12.1	(0%, 24.8%)	-1.25	2
							0.9
6	54	9.3	(0.6%, 17.9%)	27.8	(14.9%, 40.7%)	-1.51	8
							0.9
7	62	6.5	(0%, 13.4%)	35.5	(22.8%, 48.2%)	-1.62	4
							1.0
8	50	6	(0%, 13.6%)	40	(25.4%, 54.6%)	-1.66	1
9	61	0	(0%, 0.8%)	0	(0%, 0.8%)	-0.01	0.1

4.6.2 Height-for-age (Stunting)

The study found out that 18% of the children were stunted of whom 14.5% were moderately stunted while 3.6% were severely stunted. There was no significant difference (P>0.05) in the height-for-age Z score in the three schools (p=1.00).

Table 12: Distribution of the children by height-for-age Z-score

Age group	N	% < -3SD	(95% CI)	% < -2SD	(95% CI)	Mean z-score	SD
All	422	3.6	(1.7%, 5.4%)	18.0	(14.2%, 21.8%)	-1.1	1.1
(5-9)	260	2.7	(0.5%, 4.9%)	13.5	(9.1%, 17.8%)	-0.91	1.13
(10-14)	162	4.9	(1.3%, 8.6%)	25.3	(18.3%, 32.3%)	-1.4	0.98

4.6.3 BMI-for-age (Thinness)

BMI-for-age was used to measure the prevalence of thinness in the children. 23.2% of the children were found to be moderately thin while 5.7% were severely thin. Overall, 28.9% of the children had low BMI for their age. Only 0.5% of the children were found to be overweight (>1SD). There was no significant difference in the BMI-for-age z-scores between the male and female children (p=0.054) and also among the schools (p=0.836).

Table 13: Distribution of the children by BMI-for-age

Years	N	% < -3SD	(95% CI)	% < -2SD	(95% CI)	Mean z score	SD
All	422	5.7	(3.4%, 8%)	28.9	(24.5%, 33.4%)	-1.47	0.95
(5-9)	260	4.6	(1.9%, 7.4%)	26.2	(20.6%, 31.7%)	-1.44	0.92
(10-14)	162	7.4	(3.1%, 11.7%)	33.3	(25.8%, 40.9%)	-1.52	1.01

4.6.4 Anthropometric status by school

As shown in table 14, Thange Primary School a significantly lower prevalence of stunting (p=0.025). There was no significant difference in the prevalence of underweight (p=0.717) and thinness (p=0.836) amongst the three schools. The prevalence of thinness was highest in Thange Primary School (40.7%) followed by Muusini Primary School (27.6%) and Kithasyu primary School (15.2%). Stunting was highest in Kithsyu primary school (25%). (Table 13)

Table 14: Anthropometric indices by school

		Weight-for-age (%)		Height-	for-age (%)	BMI-for-age (%)		
School	N	< -3SD	< -2SD	< -3SD	< -2SD	< -3SD	< -2SD	>+1SD
All	423	2.8	31.7	3.6	18	5.7	28.9	0.5
Muusini	123	3.3	16.3	1.6	20.3	5.7	27.6	0.8
Kithasyu	133	0	9	5.3	25	0	15.2	0.8
Thange	167	4.8	17.4	3.6	10.8	10.2	40.7	0

4.7 Individual Dietary Diversity Score (IDDS)

The individual dietary diversity scores were calculated based on 9 food groups (FAO, 2011). The mean individual dietary diversity score was 3.3 (SD, 1.008). Children consumed between 1 and 6 food groups with the majority 38.3% consuming 3 food groups out of the possile 9 food groups included in the analysis. Children consuming \leq 3 food groups were considered to have low IDDS while those consuming 4 or 5 and \geq 6 were considered to have medium and high IDDS respectively. Based on this classifition, only 1.9% of the children had high IDDS. Most of the children (60.3%) had low IDDS while 37.8% of the children had medium IDDS.

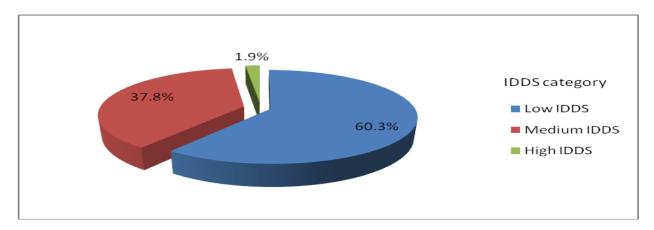


Figure 4: Distribution of the children by IDDS category

At the 5% level, the proportions of children that had low, medium and high IDDS did not differ significantly between the schools (χ 2=8.367, df=4, P>0.05). Figure 5 shows the distribution of the study children by school as per the number of food groups consumed.

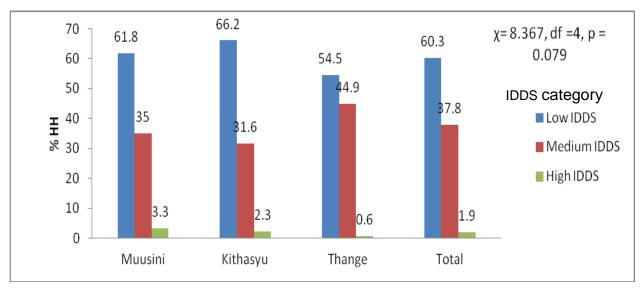


Figure 5: Distribution of study children by IDDS category and school

As shown in figure 6, the main food groups consumed by the children were starches and legumes. Generally, foods of animal origin were least consumed with no child consuming organ meats while only 7.1% and 0.7% consumed meat and fish and eggs respectively. Vitamin A rich fruits and vegetables were consumed by less than one –third of the children and vegetables contributed the most to the consumption of this food group.

Only 55.8% of the children studied consumed foods rich in vitamin A. Of these, 46.3% consumed plant foods rich in vitamin A while only 17% consumed vitamin A rich animal foods.

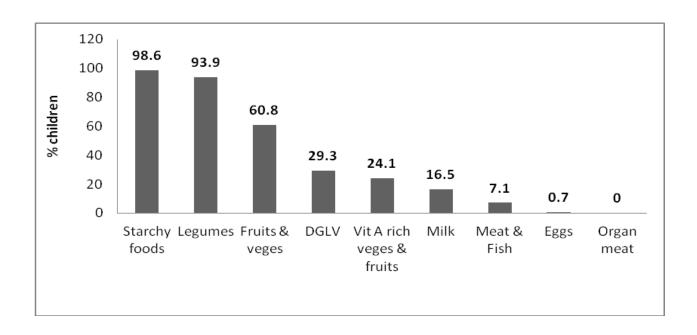


Figure 6: Percent consumption of individual food groups by the study children

4.8 Morbidity Experience

About 22.0% of the children involved in the study reported to have been sick in the immediate two weeks preceding the interview day. Of these, only 52.2% reported to have received health related treatment. Acute respiratory infections had the highest prevalence compared with the other forms of illnesses. Cough was the most common type of illness reported by most of the study children (26.2%) while bloody stool was the least reported type of illness (0.2%). The study found out that the mean number of times a child became sick within the previous two weeks was 1.06 times (SD, 0.247). The mean number of days per occasion of sickness was 3.15 days (SD, 2.4). None of the children was found positive for malaria and infection using the CRP test.

Table 15: Distribution of the study children by morbidity experience

Type of illness		Prevalence	No. of times		Days of illness			
	n	(%)	Mean	Std. Dev	Min	Max	Mean	Std. Dev
Cough	108	26.2	1.06	0.11	1	14	3.8	2.5
Headache	60	14.4	1.05	0.22	1	9	2.1	1.7
Fever	24	5.1	1.08	0.28	1	14	4.0	3.1
Joint body pains	18	4.5	1.05	0.30	1	9	4.0	2.5
Rashes	19	4.3	1.00	0.00	1	14	5.2	4.3
Diarrhea	17	4.0	1.24	0.56	1	9	2.6	1.8
Red watery eyes	10	2.6	1.18	0.60	1	13	4.4	3.6
Short Breath	8	1.9	1.00	0.00	1	9	4.0	2.7
Bloody stool	1	0.2	1.0	-	4	4	4.0	-

4.9 Factors associated with the nutritional status, dietary diversity and morbidity experince of schoolchildren in rural Kibwezi district

Significant models were obtained using logistic regressions of the predictors of child thinness $(F_{23,\ 235}=680.851,\ p<0.000,\ adjusted\ R^2=.984)$, anemia $(F_{24,\ 233}=2.37,\ p<0.001,\ R^2=0.113)$, Underweight $(F_{23,235}=1\ 796.6,\ p<0.000)$ with adjusted R square 0.994), and Stunting $(F_{23,\ 235}=1114.2,\ p<0.000,\ adjusted\ R^2=.990)$. Among the key predictors of anemia are morbidity status of the child, weight-for-age z score, height-for-age z-score and the BMI-for-age z-score. Children who were sick in the two weeks are more likely to have lower Hb levels. Age of the child has a negative relationship with stunting and thinness levels of a child. Table 16 shows significant variables included in the regression models for prediction.

Table 16: Predictors of malnutrition of children in Kibwezi district

Predictor variable	Beta	P- value
Anemia		
Child sick in last two weeks (yes)	-0.3	0.003
WAZ	-2.436	0.002
HAZ	1.928	0.002
BAZ	1.423	0.004
<u>Underweight</u>		
Caretaker relationship to the child (mother)	2.649	0.009
Age of child	0.012	0.022
HAZ	0.785	0.000
BAZ	0.614	0.000
Stunting		
Caretaker relationship to the child (mother)	0.017	0.010
RBP level	0.014	0.040
Age of child	-0.014	0.040
BAZ	-0.774	0.000
WAZ	1.261	0.000
Thinness		
Caretaker relationship to the child (mother)	0.023	0.007
RBP level	0.019	0.033
Age of child	-0.020	0.017
HAZ	1.604	0.000
WAZ	-1.259	0.000

A regression analysis showed that main source of income was a predictor of individual dietary diversity of the children (OR=1.4; 95%CI=1.3, 2.6). In predicting the factors associated with the dependent variable child morbidity, three independent variables were significant; educational levels of the caregiver (p=0.001, OR=0.328, 95% CI: 0.175, 0.617), hemoglobin levels (p=0.003, OR=0.698, 95% CI: 0.550, 0.881) and IDDS (p=0.013, OR=0.678, CI=0.497, 0.919).

Using the enter method, a significant model emerged ($F_{14, 245}$ =2.650, p < 0.001. Adjusted R square = 0.363. Significant predictors of serum RBP were individual dietary diversity of vitamin A rcih foods, birth order of the child, weight-for-age, height-for-age and BMI-for-age z-scores. Table 17 shows a summary of the model.

Table 17: Summary of the regression model of the predictors of serum RBP

	Unstand Coeffi		Standardized Coefficients		
Model	B Std. Error		Beta	t	Sig.
(Constant)	2.763	1.365		2.025	.044
Hb	.002	.075	.001	.021	.983
Gender	.280	.176	.097	1.593	.112
Birth order	.393	.192	.136	2.050	.041
Total household size	.016	.045	.023	.355	.723
Ownership of transport means	305	.188	104	-1.617	.107
Treat water	.171	.211	.050	.811	.418
Child ill in the past 14 days	.298	.322	.087	.927	.355
WAZ	-2.424	1.180	-1.606	-2.054	.041
HAZ	1.743	.794	1.353	2.194	.029
BAZ	1.654	.749	1.065	2.209	.028
Child age	.080	.042	.119	1.911	.057
IDDS	.205	.156	.141	1.313	.190
IDDS (Vitamin A)	504	.207	250	-2.432	.016
Days of Illness	.025	.080	.029	.315	.753

Dependent Variable: RBP

CHAPTER FIVE: DISCUSSION

5.1 Introduction

Malnutrition has been reported to be a serious nutritional problem of school-going children in the developing countries including Kenya and is known to be serious in families of low socio-economic status (Williams, 1994). The present study aimed at investigating the prevalence of micro- and macro- nutrient malnutrition and to describe the individual dietary diversity and morbidity burden among schoolchildren in Kibwezi District.

5.2 Socio-demographic and Economic characteristics

Most of the women attend primary school and only 6.9% having no education. This is comparable to the nation results reported in KDHS 2008-09 in which 8.9% of women had no education. The mean household size of 7 people found in this study is significantly higher than the national figure of 4.7 and 4.2 reported by 2009 population and housing census (KNBS, 2010) and the KDHS 2008-09 respectively (p=0.000) (KNBS and ICF Macro, 2010). The proportion of population above 65 years compare with that reported in the KDHS 2008-09 (KNBS and ICF Macro, 2010). The age dependency ratio of 149 is higher than the national figure of 96 reported by KNBS and ICF Macro (2010) and 82.14 reported by World Bank (2012) probably because the proportion of population below 15 years was high since the study targeted children aged 5-15 years. The study finds a gender parity rate in school enrolment of 0.91. This rate is in agreement with the national figure of 0.97 but slightly lower than the rate reported for Eastern province (KNBS and ICF Macro, 2010).

The study reveals that majority (of the households do not treat their water before drinking (74.1%). These results are consistent with those reported by ACF-International (2011). This was despite the fact that unsafe water formed the predominant source of water.

The main type of toilet facility used by majority of the households is pit latrine without slab. KNBS and ICF Macro (2010) have reported similar results for rural areas and ACF-International (2011) for Makueni county.

5.3 Micronutrient Malnutrition

A high prevalence of micronutrient malnutrition at school age is not uncommon in developing countries (Hall *et al.*, 2001). The current study did not calculate the prevalence of VAD. In Northern Ethiopia, the prevalence of VAD was 51.1% in a study conducted in 1997 in 824 pupils aged 6-9 years (Kassaye *et al.*, 2001). Two different studies in Burkina Faso by Dabone' *et al.* (2011) and Zeba *et al.* (2006) found the prevalence of VAD among schoolchildren to be 40.5% and 40%, respectively. This trend has been reported by Dabone' *et al.* (2011) and Kassaye *et al.* (2001).

The mean Hb of 8.118 (±1.15) mmol/L found in this study is significantly higher than that of children 6- 59months (6.76mmol/L) (p =0.000) and of children aged 10 -14 years (7.94mmol/L) (p =0.002), both reported by the Kenya Malaria Indicator Survey (DOMC/KNBS and ICF Macro, 2011). The prevalence of 10% of mild to severe anemia in the current study is much lower than the 70% prevalence reported by Mwaniki *et al.* (1999) and 69% reported in Kibwezi among children 2-36 months of age (Verhoef *et al.*, 2001). Other similar studies among schoolchildren in different parts of the world have found relatively higher prevalence of anemia. Using a cutoff point of <11g/dl, Stoltzfus *et al* (1997) found that the overall prevalence of anemia was 62.3 % among 3595 schoolchildren from

Pemba Island and Zanzibar. Many numerous studies (Oski, 1993; Jain and Jain, 2012) have reported above 50% prevalence of anemia in schoolchildren. In Egypt (Population Council, 2008), nearly half (47%) of the adolescents suffer from anemia. The results are however comparable to 9% prevalence of malaria among children 10 -14 years reported by the 2010 Malaria Indicator Survey in Kenya (DOMC, KNBS and ICF Macro, 2011). The current study shows no significant difference in the prevalence of anemia between the different gender as reported by Verma et al. (1998). In his study of the prevalence of anemia in school children, Dabone' et al. (2011) too did not find significance difference in the prevalence of anemia between the boys and girls indicating that both gender are affected the same way. Although there are many causes of anemia, inadequate intake of iron, folate, vitamin B12 or other nutrients usually accounts for the majority of cases in many populations. For populations living in malaria endemic regions, malaria is one of the leading causes of anemia. Other causes of anemia include thalassemia, sickle cell disease and intestinal worms. The prevalence of anemia among the children surveyed is attributable to the inadequate diet especially the low intake of foods of animal origin, which are rich in the haem iron; and poor socio economic status. In the current study, the children were screened for the presence of malarial parasites and given deworming drugs before being included in the study.

5.4 Macronutrient malnutition

Stunting (long duration malnutrition) is widely believed to occur mainly in early childhood and through a cumulative process. Children stunted at school age are likely to have been exposed to poor nutrition since early childhood and that the degree of stunting tends to increase throughout the school-age years. This study found the prevalence of stunting to be lower than those reported by Lwambo *et al.*, (2000) in Tanzania (42.5%) and Zulkifli *et al.*, (2000) in Malasyia (40.2%). Similarly, these stunting level reported in this study is lower than

the national level of 35.3% and 41.9% reported for Eastern province by KDHS 2008-09 (KNBS and ICF Macro, 2010) among children aged 5 years and below. This shows that most of the children gain their linear growth during their adolescence. These result were, however, comparable to the 21% reported by Parranga *et al.*,(1996). These findings were also similar as in South Africa, where stunting (20%) and underweight (10%) remain a public health problem in children (Labadarios *et al.*, 2008). The results also reflect those of Srivastava *et al.*, (2012) who found the prevalence of wasting and stunting among school age children to be 33.3% and 18.5% respectively. The stunting levels in the current study however are much lower than the 48% prevalence reported by Shahabuddin *et al.*, (2000) in Bangladesh. The high levels of stunting found in this study reflect accumulated levels of malnutrition throughout the seasons when food is scarce in this area.

Underweight among school-age children, like stunting, can reflect a broad range of insults such as prenatal undernutrition, deficiencies of macro- and micronutrients, infection and, possibly, inadequate attention by caregivers. In this study, the prevalence of underweight is 31.7%, and is even higher than the prevalence rate among the children under five years old as reported for Eastern province (18.9%) by KDHS 2008-09 (KNBS and ICF Macro, 2010).

The current study indicates that boys are more likely to have a higher stunting, thinness and underweight prevalence than girls. These results are similar to a number of the studies in Africa that have found boys to be at a higher risk of undernurition (Parraga *et al.*, 1996, Shahabuddin *et al.*, 2000, El Hioui *et al.*, 2011) Studies conducted in Ecuador (Sebastion and Senti, 1999) and in Tanzania (Lwambo *et al.*, 2000) show that boys were more commonly affected than girls. One of the largest studies (Partnership for Child Development, 1998) of anthropometric status of rural school children in low income countries (Ghana, Tanzania, Indonesia, Vietnam and India) found the overall prevalence of stunting and underweight to be

high in all five countries, ranging from 48 to 56% for stunting and from 34 to 62% for underweight. Boys in most countries tended to be more stunted than girls and in all countries, boys were more underweight than girls were.

5.5 Individual dietary diversity score

Dietary diversity consists of the total number of foods or food groups that contribute to the overall diet of an individual over a reference period (FAO, 2011). Dietary diversity in terms of food groups better predicts diet quality than that based on individual food items (Ruel, 2003). Dietary diversity assessed in this study consists of simple count of food groups that individuals consumed over a 24-hour reference period. A more diversified diet has been shown to increase intake of energy as well as micronutrients in developing countries (Onyango et al, 1998; Hatloy, et al., 1998; Tarini et al., 1999; Gina et al., 2007; Nti, 2011). The low diversity observed in this study is an indication of poor nutritional quality and as a result inadequate nutrient intake. A possible reason for the low diversity is poor weather conditions characterized by low and erratic rainfall that does not favor agricultural production. Kibwezi district is characterized by frequent crop failures and poor food security due to droughts (GoK, 2009). Although other studies have shown dietary diversity to correlate with nutritional indicators (Onyango et al., 1998; Hatloy, et al., 1998; Nti, 2011), this study does not find any such significant correlation. This may be so due the fact that the current study does not take into account the quantity consumed by the child. With the simple count of food groups consumed, foods consumed in very small quantities that may not contribute adequate nutrients to meet the requirements of an individual are also included during the analysis. The current study also used 24 hours as the recall period as opposed to other studies that have used up to one week as the recall period or repeated recalls. A single recall as opposed to a repeated one does not really show the typical/usual nutrient intake and as a result may not reflect on the child's nutritional status.

All the children in the study consume foods made from cereals/roots/tubers within 24 hours. Consumption of animal rich proteins is lowest in all the children with the highest consumed animal protein being milk (16.7%) and none consuming organ meats. This kind of diet is typical of many developing nations. The implication of such diets is inadequate intake of micronutrients given that animal products are the richest sources of micronutrients for instance the preformed vitamin A in the case of vitamin A and the heme-iron in the case of iron. The result is continued deprivation of micronutrient resulting in high levels of stunting as the case observed in this study. The study found that household source of income is a predictor for the dietary diversity of the children. This result is in agreement with other studies that have shown that an increase in dietary diversity is associated with socio-economic status and household food security (Hoddinot & Yohannes, 2002; Hatloy *et al.*, 2000).

5.6 Morbidity Experience

Morbidity among school children have not been widely documented in many studies involving schoolchildren. Children are especially susceptible to a host of diseases and infections that compromise their health and immunity and, in turn, their nutritional status. In this study, it is realized that 22% of the children had been ill in the reference period. This is quite high given the fact that illnesses affect school attendance and academic performance. The mean number of days of illness of 3 days is likely to affect the school attendance and subsequently the academic performance. The most common symptoms reported are those of respiratory system. Diarrhea remains a problem with 4% of the children having experienced diarrhea in the reference period. The study also reveals that the educational level of the

caregiver is significant predictors of morbidity status of a child. This finding is similar with the result obtained from another study in Ghana and Nigeria, where the prevalence of diarrhea varies according to education of mothers which was relatively high among children of mothers with no education (Oadi and Kuitunen, 2005; Yilgwan and Okolo, 2012; Mohammed *et al.*, 2013). This is probably because education provides the knowledge of the rules of hygiene, feeding and weaning practices, and the interpretation of symptoms, which enhances timely action on childhood illness.

CHAPTER SIX: CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

Based on the findings of this study, it is evident that malnutrition remains a problem among schoolchildren. Both acute and chronic malnutrition remains a problem even in children who are older than five years. The prevalence of anemia is of mild public health significance based on the WHO cut-offs (WHO, 2011).

The study has shown that morbidity status, stunting, underweight and thinness are significant predictors of anemia in children. Stunting, thinness and underweight are all associated with a child's age and relationship of the child to the caregiver.

The individual dietary diversity is not associated with a child's micronutrient status, as the dietary diversity among schoolchildren is low and only a very small portion consume foods from animal origin. Hence, both food quantity and quality consumed by children are inadequate for proper growth and good health. Household's main source of income is a significant determinant of a child's dietary diversity.

Acute respiratory infections are prevalent among schoolchildren. Many children do not seek medical care during illness. Educational levels of the caregiver, and dietary diversity of a child determines a child's morbidity status. Low hemoglobin levels is associated with illness among the children.

6.2 Recommendations

The prevalence of micronutrient deficiency should be of concern and underlines the compelling need for corrective and preventive measures in schools, which should no longer be neglected in favor of pre-schoolchildren. Therefore, interventions that address the nutrition of schoolchildren and the household at large should be put in place to address the problem.

Projects that improve household income like employment creation should be put in place in order to improve the dietary diversity and nutrient intake of the children.

Malnutrition and childhood diseases are interconnected and mutually reinforce one another. It is therefore extremely important that childhood diseases are identified, and appropriately treated, to contain the effect of the disease on child health. Parents and caregivers should be given education on the importance of seeking medical care during child illness.

REFERENCES

- Abalkhail B and Shawky S (2002). Prevalence of daily breakfast intake, iron deficiency anemia and awareness of being anemic among Saudi school students. Int J Food Sci and Nutr, 53(6): 519–28.
- ACF-International (2011). Integrated Smart Survey Report. Kibwezi, Makindu And Kathonzweni Districts & Kalawa, Nguu, Mulala, Kiou and Malili Divisions, Makueni County, Kenya
- Adair LS (1999). Filipino children exhibit catch-up growth from age 2 to 12 years. J Nutr; 129: 1140-1148
- Adaman JW Dan L (2005). Anemia and polycythemia. Section-10 Hematological Alterations. In Harrison's Principles of Internal Medicine 16th ed. Vol. 1, editors Kasper DL Fauci AS Longo DL Braunwald E Hauser SL Jameson JL; pp 334-336 McGraw Hill, N. Y.
- Agarwal K N. Iron and brain: neurotransmitter receptors and magnetic response spectroscopy.

 <u>Brit J Nutr</u> 2001; 85 Suppl 2 S147-150.
- Al-Awadi FM, Karam SM and Khan I (2000). Vitamin A deficiency in mice enhances the colonic level of purine enzyme activity. Ann. Nutr. Metab. 44: 150–156.
- Anstead GM, Chandrasekar B, Zhang Q & Melby PC (2003). Multinutrient undernutrition dysregulates the resident macrophage proinflamatory cytokine network, nuclear factor-kB activation, and nitric oxide production. *J Leuk Biol*; **74**: 982–991.
- Arroyave G, Auilar JR, Flores F, and Guzman MA (1979). "Evaluation of Sugar Fortification with Vitamin A at the National Level," Scientific Publication No. 384, Pan American Health Organization, Washington, D.C.Asobayire F, Adou P, Davidsson L, Cook J and Hurrell R (2001). Prevalence of iron deficiency with and without concurrent anemia in population groups with high prevalence of malaria and other infections. Am J Clin Nutr, 74(6): 776–782.
- Aukrust P, MuÈ ller F, Ueland T, Svardal AM, Berge RK and Frùland SS, (2000). Decreased vitamin A levels in common variable immunodeficiency: vitamin A supplementation in vivo enhances immunoglobulin production and downregulates inflammatory responses. Eur J Clin Invest; 30 (3): 252-259

- Barabino A (2002). Helicobacter pylori-related iron deficiency anemia: A review. Helicobecter; 7: 71-75
- Beasley NM, Tomkins AM, Hall A, Kihamia CM, Lorri W, Nduma B, Issae W, Nokes C, and Budy DA(1999) The impact of population level deworming on the heamoglobin levels of school children in Tanga, Tanzania. Trop Med and Int Health 4: 744-750.
- Berkman DS, Lescano AG, Gilman RH, Lopez SL, Black MM (2002). Effects of stunting, diarrheal disease, and parasitic infection during infancy on cognition in late childhood: a follow-up study. Lancet; 359:564-571
- Bertoncello C, Cazzaro R, Ferraresso A, Mazzer R, Moretti G. (2008). Prevalence of overweight and obesity among school-aged children in urban, rural and mountain areas of the Veneto Region, Italy. Public Health Nutr.;11(9):887–890. doi: 10.1017/S1368980007001152.
- Black MM, (2003). Micronutrient Deficiencies and cognitive functioning. J Nutr; 133:3927-1331.
- Black RE, Allen LH, Bhutta ZA, Caulfield LE, de Onis M, Ezzati M, Mothers C, Rivera J, Maternal and Child Undernutrition Study Group (2008). Maternal and Child Undernutrition: global and regional exposures and health consequences. Lancet 371: 243-260.
- Borelli P & Nardinelli L (2001). Protein-calorie malnutrition: decrease in the peritoneal macrophage's respiratory burst. *Braz J Pharm Sci*; **37**: 51–60.
- Bothwell TH, Baynes RD, MacFarlane BJ and MacPhail AP (1989). Nutritional iron requirements and food iron absorption. J Internal Med. 226, 357-365
- Brock C and Cammish N (1997). Factors Affecting Female Participation in Seven Countries. DFID.
- Brondy, T. (1999). Nutritional biochemistry. 2nd ed. Academic Press, San Diego, California.
- Bundy DAP, Hall A, Medley GF and Savioli L (1992) Evaluating measures to control intestinal parasitic infections. World Health Statistics Quarterly 45:168-179.

- Cairncross S, Blumenthal U, Kolsky P, Moraes L and Tayeh A.(1996). The public and domestic domains in the transmission of disease. Trop Med and Int Health 1:27-34.
- Cantorna MT, Nashold FE, and Hayes CE, (1994). In vitamin A deficiency multiple mechanisms establish a regulatory T helper cell imbalance with excess Th1 and insufficient Th2 function. J. Immunol. 152:1515–1522.
- Casapia M, Joseph SA, Nunez C, Rahme E and Gyrkos TW (2006). Parasite risk factors for stunting in grade 5 students in a community of extereme poverty in Peru. Int J Parasitol; 36:741-747
- Cattaneo A, Monasta L, Stamatakis E, Lioret S, Castetbon K, Frenken F, Manios Y, Moschonis G, Savva S, Zaborskis A, Rito AI, Nanu M, Vignerova J, Caroli M, Ludvigsson J, Koch FS, Serra-Majem L, Szponar L, van Lenthe F and Brug J. (2010). Overweight and obesity in infants and pre-school children in the European Union: a review of existing data. Obes Rev.;11(5):389–398. doi: 10.1111/j.1467-789X.2009.00639.x
- Charlton R W and Bothwell T H (1989). Iron absorption. Annual Review of Medicine, 34:55-68.
- Christian P, West KP, Khatry SK, Kimbrough-Pradhan E, LeClerq SC, Katz J, Shrestha SR, Dali SM and Sommer A (2000). Night blindness during pregnancy and subsequent mortality among women in Nepal: effects of vitamin A and β-carotene supplementation. Am J Epidemiol, 152:542-547.
- Christian P., West K. P., Jr., Khatry S. K., Katz J., Shrestha S. R., Pradhan E. K., LeClerq S. C. and Pokhrel R. P. (1998). Night blindness of pregnancy in rural Nepal: Nutritional and health risks. Int. J. Epidemiol. 27: 231–237.
- Cook JD and Finch CA (1979). Assessing iron status of a population. Am J Clin Nutr 32, 2115-2119
- Cook JD and Reusser ME (1983). Iron fortification: an update. Am J Clin Nutr. 38, 648-659

- Coutsoudis A, Broughton M and Coovadia HM (1991). Vitamin A supplementation reduces measles morbidity in young African children: a randomized, placebo-controlled, double-blind trial. Am. J. Clin. Nutr. 54: 890-895.
- Cunningham-Rundles S, McNeely D and Ananworanich JM (2004). Immune response in malnutrition. In Stiehm ER, Ochs HD, Winkelstein JA (eds): *Immunologic Disorders in Infants and Children*. Philadelphia, Saunders.
- Daboné C, Delisle HF and Receveur O (2011). Poor nutritional status of schoolchildren in urban and peri-urban areas of Ouagadougou (Burkina Faso). Nutr J 10:34.
- de Onis M, Onyango AW, Van den Broeck J, Chumlea WC, Martorell R (2004).

 Measurement and standardization protocols for anthropometry used in the construction of a new international growth reference. Food Nutr Bull. 2004 Mar;25(1 Suppl):S27-36.
- de Pee S, and West CE (1996). Dietary carotenoids and their role in combating vitamin A deficiency: a review of the literature. Eur J Clin Nutr 50: 538-553.
- de Pee S and Dary O (2002): Biochemical indicators of vitamin A deficiency: serum retinol and serum retinol binding protein. J Nutr, 132(9 suppl):2895S-2901S.
- Decsi T and Koletzko B. (2000). Effects of Protein-Energy Malnutirtion and Human Immunodeficieny Virus-1 Infection on essential Fatty acid metabolism in children. Nutr; 16:447-453.
- Dekker LH, Mora-Plazas M, Marin C, Baylin A and Villamor E, (2010). Stunting associated with poor socioeconomic and maternal nutrition status and respiratory morbidity in Colombian schoolchildren. Food Nutr Bull 31 (2): 242-250.
- Derman DP, Bothwell TH, MacPhail AP, Torrance JD, Bezwoda WR, Charlton RW and Mayet FG. (1980). Importance of ascorbic acid in the absorption of iron from infant foods. Scand J Hematol, 25(3): 193-201.
- DOMC /Ministry of Public Health and Sanitation], Kenya National Bureau of Statistics, and ICF Macro (2011). 2010 Kenya Malaria Indicator Survey. Nairobi, Kenya: DOMC, KNBS and ICF Macro.

- El Hioui M, Azzaoui FZ, Ahmani AOT and Aboussaleh Y (2011). Nutritional Status and School Achievements in a Rural Area of Anti-Atlas, Morocco. Food Nutr Sci, 2011, 2, 878-883
- Elk G V (1985). Iron deficiency. 2nd ed.: American Academy of Pediatrics. USA. P: 213-220.
- FAO (2006). The double burden of malnutrition: Case studies from six developing countries. FAO Food and nutrition paper No84, Rome.
- FAO (2011). Guidelines for Measuring Household and Individual Dietary Diversity. FAO, Rome, Italy.
- FAO/WHO (1992). International Conference on Nutrition. FAO, Rome.
- Finch CA and Huebers H (1982) Perspectives in iron metabolism. New England J of Med 306, 1520-1528
- Fischer, AA., Lang, JE., Stoeckel, JE and Townsend, JW. 1991. Hand book for family planning operations, Research Design 2nd Edition. The population council, NewYork.
- Fock RA, Vinolo MAR, Rocha VMS, Rocha LCS and Borelli P (2007). Protein-energy malnutrition decreases the expression of TLR-4/ MD-2 and CD14 receptors in peritoneal macrophages and reduces the synthesis of TNF-α in response to lipopolysaccharide (LPS) in mice. *Cytokine*; 40(2 Supl): 105–114.
- Fomon S, Drulis J Nelson S, Serfass R, Woodhead J and Ziegler E (2003). Inevitable iron loss by human adolescents, with calculations of the requirement for absorbed iron. J Nutr, 133: 167–172.
- Gina LK, Maria RP, Chiara S, Guy N and Inge B (2007). Dietary Diversity Score Is a Useful Indicator of Micronutrient Intake in Non-Breast-Feeding Filipino Children. J Nutr.; 137:472-477.
- Glasziou PP, Mackerras DE.(1993). Vitamin A supplementation in infectious diseases: a meta-analysis. British Med J, 1993, 306:366–370.
- GoK (2009). Makueni District Project Annual progress report July 2007 June 2008.

- GoK. (2002). Makueni District Development Plan for the period 2002-2008. Government Printers, Nairobi.
- Hall A, Bobrow E, Brooker S, Jukes M, Nokes K, Lambo J, Guyatt H, Bundy D, Adjei S, Wen ST, Subagio H, Rafiluddin MZ, Miguel T, Moulin S, de Graft Johnson J, Mukaka M, Roschnik N, Sacko M, Zacher A, Mahumane B, Kihamia C, Mwanri L, Tatala S, Lwambo N, Siza J, Khanh LN, Khoi HH and Toan ND (2001): Anaemia in schoolchildren in eight countries in Africa and Asia. Public Health Nutr, 4(3):749-756.
- Hallberg L(2001.) Perspectives on nutritional iron deficiency. Annual Review of Nutrition 21, 1-21
- Hallberg L, Sandstrom B and Aggett PJ (1993). Iron, zinc and other trace elements In Human nutrition and dietetics, pp 174-188 [JS Garrow and WPT James, editors] Edinburgh [etc] Churchill Livingstone
- Hashizume, M., Kunii, O., and Sasaki S(2003). Anemia and iron deficiency among schoolchildren in the Aral Sea region, Kazakhstan. J Trop Pediatric, 49 (3): 172–177.
- Hatloy, A., Hallund, J., Diarra, MM and Oshaug A. (2000). Food variety, socioeconomic status and nutritional status in urban and rural areas in Koutiala (Mali). *Public Health Nutrition* 3:57-65.
- Hatløy, A., L. E. Torheim, and A. Oshaug (1998). "Food variety.A good indicator of nutritional adequacy of the diet? A case study from an urban area in Mali, West Africa. Eur J Clin Nutr; 52:891-898.
- Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM and Parra-Cabrera S.(1999). Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico City. Int J Obesity and Related Metabolic Disorders 23(8): 845-54.
- Hoddinott, J and Yohannes, Y. (2002). *Dietary diversity as a food security indicator*. FANTA 2002, Washington DC. http://www.aed.org/Health/upload/dietarydiversity.pdf
- Horton S and Ross J (2003). "The Economics of Iron Deficiency." Food Policy 28(1): 51-75.

- Hurrell R F (1997). Bioavailability of iron. Eur J Clin Nutr, 51: S4–S8.
- Hussey GD and Klein M (1990). A randomized, controlled trial of vitamin A in children with severe measles. New England J Med; 323: 160–164
- Immink MD and Payongayong E (1999) Risk analysis of poor health and growth failure of children in the central highlands of Guatemala. Social Sci & Med 48: 997-1009
- Institute of Medicine (2002). Iron: In Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington, DC, National Academy Press
- Institute of Medicine (IoM) (2001). Food and Nutrition Board. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington, DC: National Academy Press.
- Jain N and Jain VM (2012). Prevalence of anemia in school children. Medical Practice and Review Vol. 3(1), pp. 1-4 (http://www.academicjournals.org/MPR)
- Johnson EJ and Russell RM (2010). Beta-Carotene In: Coates PM, Betz JM, Blackman MR, et al., eds. Encyclopedia of Dietary Supplements. 2nd ed. London and New York: Informa Healthcare; 115-20.
- Kamari S and Jain R (2005). Assessment of school children from rural Bihar. Ind J. Nutr. Dietet., 42:326-334
- Kassaye T, Receveur O, Johns T, Becklake MR (2001): Prevalence of vitamin A deficiency in children aged 6-9 years in Wukro, northern Ethiopia. Bull World Health Organ, 79(5):415-422.
- Kenya National Bureau of Statistics (KNBS) and ICF Macro. 2010. *Kenya Demographic and Health Survey 2008-09*. Calverton, Maryland: KNBS and ICF Macro.
- Keusch GT (2003). The history of nutrition: malnutrition, infection and immunity. *J Nutr*; **133**: 336–340.

- Khuwaja S, Selwyn BJ and Shah SM, (2005). Prevalence and correlates of stunting among primary school children in rural areas of southern Pakistan. J Trop Pediatr; 51:72-77
- KNBS (2010). Kenya 2009 Population and Housing Census Highlights.
- KNBS (www.knbs.or.ke /finddata.php /Census Volume 1 Question 1 Population

 Households and Density_by_Sublocations_-_2009) accessed on 07/05/2013
- KNBS and ICF Macro (2010). Kenya Demographic and Health Survey 2008-09. Calverton, Maryland: KNBS and ICF Macro
- Kossmann J, Nestel P, Herrera MG, El Amin A, Fawzi WW. (2000). Undernutrition in relation to childhood infections: a prospective stidy in Sudan. Eur J Clin Nutr; 54:463-472.
- Kumar MV, Sunvold G D, Scarpace PJ (1999). Dietary vitamin A supplementation in rats: suppression of leptin and induction of UCP1 mRNA. J Lip Res 1999, 40:824-29.
- Labadarios D, Swart R, Maunder EMW, Kruger HS, Gericke GJ, Kuzwayo PMM, Ntsie PR, Steyn NP, Schloss I, Dhansay MA, Jooste PL, Dannhauser A, Nel JH, Molefe D and Kotze YJVW (2008): Executive summary of the national food consumption survey fortification baseline (NFCS-FB-1) South Africa, 2005. S Afr J Clin Nutr, 21(Suppl 2):245-300.
- Livingstone DEW, Jones G, Smith K, Jamieson PM, Andrew R Kenyon CJ, Walker BR (2000). Understanding the role of glucocorticoids in obesity: tissuespecific alterations of corticosterone metabolism in obese zucker rats. Endocrinol, 141:560-563.
- Lwambo NJ, Brooker S, Siza JE, Bundy DA & Guyatt H (2000): Age patterns in stunting and anaemia in African schoolchildren: a cross-sectional study in Tanzania. Eur. J. Clin. Nutr. 54, 36–40.
- Lwambo NJS, Bundy DAP and Medley GFH (1992) A new approach to morbidity risk assessment in hookworm endemic communities. Epidemiol Infection. 108: 469.
- Lynch SR and Green A (2001). Assessment of nutritional anemias: In Nutritional Anemias, pp 24-43 [U Ramakrishnan, editor] Boca Raton CRC Press LLC

- Marshall SJ, Biddle SJH, Gorely T, Cameron N, Murdey I (2004). Relationship between media use, body fatness and physical activity in children and youth: A meta-analysis. Int J Obes, 28:1238-1246.
- Martorell R (2002) Obesity in the developing world. In: Coballero B and Popkin B. The Nutrition Transition: Diet- Related Diseases in the Modern World.
- Maslova E, Mora-Plazas M, Forero Y, Lopez-Arana S, Baylin A, and Villamor E, (2009). Are Vitamin A and Iron deficiencies re-emerging in urban Latin America? A survey of Schoolchildren in Bogota, Colombia. Food Nutr Bull; 30:103-111.
- McLaren DS, Frigg M (2001). Sight and Life Manual on Vitamin A Deficiency Disorders (VADD) 2nd edition. Basel: Task Force Sight and Life.
- Means, R.T. (1994): Pathogenesis of anaemia of chronic diseases: a cytokine mediated anaemia. *Stem cells*. 13:32 7
- Mohammed S, Tilahun M, Tamiru D (2013). Morbidity and Associated Factors of Diarrheal Diseases Among Under Five Children in Arba-Minch District, Southern Ethiopia, 2012. *Science Journal of Public Health*. Vol. 1, No. 2, pp. 102-106. doi: 10.11648/j.sjph.20130102.19
- Mwaniki, DL, Omwenga, AM, Muniu EM, Mutunga JN, Akelola R, Shako BR, Gotink MH/Perfect (1999). Aneamia and Status of Iron, Vitamin A and Zinc in Kenya: National Micronutrient Survey Report.
- National Academies of Sciences (2002). Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc: A Report of the Panel on Micronutrients. National Academy Press: Washington DC.
- Neumann CG, Gewa C & Bwibo NO (2004). Child nutrition in developing countries. *Pediatr Ann*; **33**: 658–674.
- Niiya T, Akbar SM, Yoshida O, Miyake T, Matsuura B, Murakami H, Abe M, Hiasa Y, Onji M.(2007). Impaired dendritic cell function resulting from chronic undernutrition disrupts the antigen-specific immune response in mice. *J Nut* 2007; **137**: 671–675.

- Nti CA (2011). Dietary Diversity is Associated with Nutrient Intakes and Nutritional Status of Children in Ghana. Asian J Med Sci (2); 105-109
- Oadi KO, Kuitunen M. Childhood diarrheal morbidity in the Accra Metropolitan Area, Ghana: Socio-economic, environmental and behavioral risk determinants. J Health Pop Dev Countries 2005:33-46.
- Onyango, A., K. G. Koski, and K. L. Tucker (1998). "Food diversity versus breastfeeding choice in determining anthropometric status in rural Kenyan toddlers. Intl J Epidemiol; 27:484-489.
- Osei A, Houser R, Bulusu S, Joshi T, and Hamer D, (2010). Nutritional Status of primary schoolchildren in Garhwali Himalayan illages of India. Food Nutr Bull;31:221-233.
- Oski F (1993). Iron deficiency in infancy and childhood. N Eng J Med, 329(3): 190–193.
- Parraga MI, A. Assis AM, Prado MS, Barreto ML, Reis GM, King CH and Blanton RE. (1996). "Gender Differences in Growth of School-Age Children with Schistosomiasis and Geohelminth Infection," Am J Trop Med Hyg, Vol. 5, pp. 150-156.
- PCD (1998). The anthropometric status of school children in five countries in the Partnership for Child Development. Proceedings of the Nutrition Society; 57:149-158.
- Population Council, (2008). Transitions to adulthood, a national survey of adolescents in Egypt. Available from http://www.popcouncil.org/ta/transition/health.html
- Prentice AM, (2006). The emerging epidemic of obesity in developing countries. Int J Epidemiol. 35(1):93–99. doi: 10.1093/ije/dyi272
- Rahmathullah, L, Underwood, BA., Thulasiraj, RD., Milton, RC., Ramaswamy, K, Rahmathullah, R. & Babu, G. (1990). Reduced mortality among children in southern India receiving a small weekly dose of vitamin A. N. Engl. J. Med. 323: 929-935.
- Ramakrishman U (2002). Prevalence of micronutrient malnutrition worldwide. Nutr Rev; 60(5 pt 2):S46
- Recommendations of the Seventh IVACG Meeting, Dakar, Senegal, January 31-February 4, 1983.

- Reifen R, Nur T, Ghebermeskel K, Zaiger G, Urizky R and Pines M (2002). Vitamin A Deficiency Exacerbates Inflammation in a Rat Model of Colitis through Activation of Nuclear Factor-κ B and Collagen Formation. J. Nutr. 132: 2743–2747.
- Rice AL, West Jr KP, BlackR (2004). Vitamin A deficiency. In Comparative Quantification of Health Risks: Global and Regional burden of disease attributable to selected major risk factors. Vol. 1. Eds. Ezzati M, Lopez ADL, Rodgers A, and Murray CJL, WHO, Geneva, Switzerland. 212-256.
- Robinson TN, (2001). Television viewing and childhood obesity. Pediatr Clin North Am, 48:1017-25.
- Rosenfield L (1997). Vitamine Vitamin. The early years of discovery. Clinical Chemistry. New York University Medical Center, Department of Pathology, 560 First Ave., New York, NY 10016. 43: 4. 680-685.
- Ross A (2006). Vitamin A and Carotenoids. In: Shils M, Shike M, Ross A, Caballero B, Cousins R, eds. Modern Nutrition in Health and Disease. 10th ed. Baltimore, MD: Lippincott Williams & Wilkins:351-75.
- Ross CA (2010). Vitamin A. In: Coates PM, Betz JM, Blackman MR, et al., eds. Encyclopedia of Dietary Supplements. 2nd ed. London and New York: Informa Healthcare; 778-91.
- Ruel MT (2003). Operationalizing Dietary Diversity: A Review of Measurement Issues and Research Priorities. J Nutr 133:3911S-3926S.
- Ruel MT and Levin CE (2001). Food-based approaches: In Nutritional Anemias, pp 153-183 [U Ramakrishnan, editor] Boca Raton CRC Press LLC
- Sakamuri VP, Ananthathmakula PA, Veettil GN, and Ayyalasomayajula V, (2011). Vitamin A decreases pre-receptor amplification of glucocorticoids in obesity: study on the effect of vitamin A on 11beta-hydroxysteroid dehydrogenase type 1 activity in liver and visceral fat of WNIN/Ob obese rats. Nutr J 10:70.

- Sauer K, Knoblich JA, Richardson H, Lehner CF (1995). Distinct modes of cyclin E/cdc2c kinase regulation and S-phase control in mitotic and endoreduplication cycles of Drosophila embryogenesis. Genes Dev. 9(11): 1327--1339
- Savioli L, Bundy DAP and Tomkins A (1992). Intestinal parasitic infections: a soluble health problem. Trans R Trop Med Hyg; 86: 343-4
- SCN (2002). School-age children: their health and Nutrition. SCN News December, 2002.
- Scrimshaw NS (2003). Historical concepts of interactions, synergism and antagonism between nutrition and infection. *J Nutr*; **133**: s316–s21.
- Sebastion MS, Senti S (1999): The health status of rural school children in Amazon Basin of Ecuador. J Trop Pediatr, 45:379-382.
- Semba RD (1994). Vitamin A, immunity, and infection. Clin Infect Dis;19: 489–99.
- Semba RD and Bloem MW (2002). The aneamia of Vitamin A deficiency: epidemiology and pathogenesis. Eur J Clin Nutr 56: 271-281.
- Shahabuddin AKM, et al (2000) Adolescent nutrition in a rural community in Bangladesh. *Indian Journal of Pediatrics* 67(2): 93-98.
- Shetty P, (2011). Addressing Micronutrient Malnutrition to achieve Nutrition Security. In Combating Micronutrient Deficiencies: Food-based approaches. Eds. B Thompson and L. Amorosa. CABII publishing, Wallingford. 28-41.
- Shoeps OD, de Aberu LC, Valenti VE, Nascimento VG, de Oliveire AG, Gallo PR, Wajnsztejn R and Leone C. (2011). Nutritional status of pre-school children from low income families. Nutr J; 10:43
- Singh V, West KP Jr, (2004). Vitamin A deficiency and xeropthalmia among school aged children in South Asia. Eur J Clin Nutr; 58:1342-1349
- Solomons, N. W. (2001). Vitamin A and carotenoids, In B. A. Bowman and R. M. Russell (ed.), Present knowledge in nutrition. ILSI Press, Washington, D.C. p.127 -145.

- Solon SF, Klemm RDW, Sanchez I, Darnton-Hill I, Craft NE, Christian P, and West KP Jr, (2000). Efficacy of a vitamin A–fortified wheat-flour bun on the vitamin A status of Filipino schoolchildren. Am J Clin Nutr;72:738–744.
- Sommer A, (1995). Vitamin A deficiency and its consequences: a field guide to detection and control, 3rd ed. Geneva, World Health Organization.
- Sommer A, Davidson FR, (2002). Assessment and control of vitamin A deficiency: the Annecy Accords. J Nutr, 132: 2845S–2850S.
- Sommer A, West KP Jr, (1996). Vitamin A deficiency: health survival, and vision. New York: Oxford University Press.
- Soprano, D. R. & Blaner, W. S. (1994) Plasma retinol-binding protein. In: The Retinoids: Biology, Chemistry, and Medicine, 2nd ed. (Sporn, M. B., Roberts, A. B. & Goodman, D. S., eds.), 257–281. Raven Press, New York, NY.
- Spickett GP, Farrant J, North ME, Zhang J-G, Morgan L, Webster ADB. (1998). Common variable immunodeficiency: how many diseases? Immunol Today;18:325±8.
- Srivastava A, Mahmood SE, Srivastava PM, Shrotriya VP and Kumar1 B (2012). Nutritional status of school-age children A scenario of urban slums in India. Arch Public Health 70:8.
- Stephenson L (1993) The impact of schistosomiasis on human nutrition. Parasitology 107 Suppl, S107-123
- Stoltzfus RJ, Albonico M, Tielsch JM, Chwaya HM and Savioli L. (1997) Linear growth retardation in Zanzibari school children. J Nutr; 127: 1099-1105
- Stunkard AJ, Berkowitz RI, Stallings VA, Schoeller DA (1999): Energy intake, not energy output, is a determinant of body size in infants. Am J Clin Nutr 1999, 69:524-30.
- Styen NP, NEL JH, Nantel G, Kennedy G, Labadarios D, (2006). Food Variety and Dietary Diversity Scores in children: are they good indicators of dietary adequacy: Public Health Nutr 9(5):644-50.

- Swamidas, G. P., Basaraba, R. J. & Baybutt, R. C. (1999). Dietary retinol inhibits inflammatory responses of rats treated with monocrotaline. J. Nutr. 129: 1285–1290.
- Tarini, A., S. Bakari, and H. Delisle (1999). "The overall nutritional quality of the diet is reflected in the growth of Nigerian children." Sante; 9; 23-31
- Tatala S, Svanberg U and Mduma B (1998). Low dietary iron availability is a major cause of anemia. Am J Clinical Nutr, 69: 171–178.
- TAWSB (2011). Environmental Impact Assessment for Construction for Umanyi Mtito Andei Water Supply Project.
- Taylor PG, Mendez-Castellanos H, Martinez-Torres C, Jaffe W, Lopez de Blanco M, Landaeta Jimenez M, Leets I, Tropper E, Ramirez J and Garcia Casai MN (1995) Iron bioavailability from diets consumed by different socioeconomic strata of the Venezuelan population. J Nutr 125, 1860-1868
- Tchum SK, Tanumihardjo SA, Newton S, De Benoist B, Owusu SA, Arthur FKN, and Tetteh A, (2006). Evaluation of vitamin A supplementation regimens in Ghanaian postpartum mothers with the use of the modified relative-dose response test. Am J Clin Nutr; 84:1344 –1349.
- Thiam I, Samba K, Lwanga D (2006). Diet related chronic disease in the West Africa Region. in Diet-related chronic diseases and the double burden of malnutrition in West Africa. SCN News. 33:6–10.
- Torres AM, Peterson KE, de Souza ACT, Orav EJ, Hughes M and Chen LC. (2000) Associations of diarrhoea and upper respiratory infections with weight and height gains in Bangladeshi children aged 5 to 11 years. Bulletin of the World Health Organization 78: 1316-1323.
- UNICEF/MI (2004). Vitamin and Mineral Deficiency: A World Progress Report.

 Micronutrient Initiative, Ottawa, Ontario.
- Verhoef H, West CE, Ndeto P, Burema J, Beguin Y, Kok FJ. Serum transferrin receptor concentration indicates increased erythropoiesis in Kenyan children with asymptomatic malaria. Am J Clin Nutr 2001;74: 767–75.

- Verma M, Chhatwal J, Kaur G (1998). Prevalence of Anaemia among Urban School Children of Punjab. (www.indianpediatrics.net/dec/htm.)
- Victoria CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, Sachdev HS, Maternal Maternal and Child Undernutrition Study Group (2008). Maternal and Child Undernutrition: consequences for adult health and human capital. Lancet 371: 340-357.
- Villamor, E., and W. Fawzi (2000). Vitamin A supplementation: implications for morbidity and mortality in children.J. Infect. Dis. 182:S122-S133.
- West KP (2002). Extent of Vitamin A Deficiency among preschool children and women of reproductive age. J Nutr, 132(Suppl):2857-2866.
- West, K. P. J., Pokhrel, R. P., Katz, J., LeClerq, S. C., Khatry, S. K., Shrestha, S. R., Pradhan, E. K., Tielsch, J. M., Pandey, M. R. & Sommer, A. (1991) Efficacy of vitamin A in reducing preschool child mortality in Nepal. Lancet338: 67–71.
- West, K. P., Jr., Katz, J., Khatry, S. K. LeClerq, S. C., Pradhan, E. K., Shrestha, S. R., Connor, P. B., Dali, S. M., Christian, P., Pokhrel, R. P. & Sommer, A., on behalf of the NNIPS-2 Study Group (1999). Double blind, cluster randomized trial of low dose supplementation with vitamin A or β-carotene on mortality related to pregnancy in Nepal. Br. Med. J. 318: 570–575.
- Whitcher, J. P., M. Srinivasan, and M. P. Upadhyay (2001). Corneal blindness: a global perspective.Bull. WHO. 79:214-221.
- WHO (1974). "Current Approaches for the Prevention of Vitamin A Deficiencies-Food Fortification," presented at a Joint WHO/USAID Meeting on the Control of Vitamin A Deficiency, NUT/WP/74.15, WHO, Jakarta, Indonesia, November 25-29.
- WHO (1982). Control of vitamin A deficiency xerophthalmia. Report of a Joint WHO/UNİCEF/USAID/Helen Keller International IVAGC Meeting 1982, WHO Technical Report Series, Report No: 672.
- WHO (1991). Hookworm infections and anaemia Approaches to prevention and control. Geneva, Switzerland WHO

- WHO (1993). The control of Schistosomiasis. WHO Tech Rep Ser No 830 Geneva, Switzerland WHO
- WHO (1995a) The Global Prevalence of Vitamin A Deficiency. Micronutrient Deficiency Information System (MDIS) Working Paper 2: WHO/NUT/95 3:World Health Organization, Geneva, Switzerland
- WHO (1995b). WHO Expert Committee Physical status: The use and interpretation of anthropometry. Report of a WHO Expert Committee. World Health Organization, Geneva, Switzerland
- WHO (1996) World Health Report: Fighting Disease, Fostering Development. WHO: Geneva.
- WHO (2001). Iron Deficiency Anaemia: Assessment, Prevention and Control. World Health Organization: Geneva
- WHO (2002). The world health report 2002: reducing risks, promoting healthy life. Geneva: WHO.
- WHO (2009a). Global Prevalence of Vitamin A Deficiency in Populations at Risk 1995–2005. WHO Global Database on Vitamin A Deficiency. Geneva.
- WHO (2009b). WHO AnthroPlus for personal computers Manual: Software for assessing growth of the world's children and adolescents. Geneva: http://www.who.int/growthref/tools/en/
- WHO (2011) Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. Vitamin and Mineral Nutrition Information System. Geneva, World Health Organization.(WHO/NMH/NHD/MNM/11.1)(http://www.who.int/vmnis/indicators/haemoglobin.pdf
- WHO/CDC (2004). Best indicators to assess iron deficiency, a major cause of anemia. 7th May 2004. http://www.who.int/.(Dr Bruno de Benoist-email:debenoist@who.int.).
- Widdowson EM. and Spray CM (1951). Chemical development in utero, Arch Disease in Childhood, 77(1):205-214.

- Williams SR(1994). Essential of Nutrition and Diet Therapy. Seventh Ed, St Louis, Baltimore, London, pp 134-149.
- World Bank (1993) World Development Report: Investing in Health. Oxford: Oxford University Press.
- Yilgwan CS and Okolo S N (2012). Prevalence of diarrhea disease and risk factors in Jos University Teaching Hospital, Nigeria. Ann Afr Med; 11: 217-21.
- Zeba AN, Prevel YM, Some IT, Delisle HF(2006). The positive impact of red palm oil in school meals on vitamin A status: study in Burkina Faso. Nutr J, 5:17.
- Zimmermann MB, and Hurrell RF (2007). Nutritional Iron Deficiency. Lancet 370: 511-520.
- Zulkifli A, Anuar AK, Atiya AS and Yano A (2000): The prevalence of malnutrition and geohelminth infections among primary school children in rural Kelantan. *Southeast Asian J Trop Med Public Health*, **31**(2):339–345.

ANNEXES

Annex 1: Informed Consent Form

Study Name: Malnutrition, dietary diversity, morbidity and associated factors among

schoolchildren in Kibwezi district, Kenya

Investigator: Francis Odhiambo Oduor

Reg No: A56/70114/2011

MSc. Applied Human Nutrition

University of Nairobi

Purpose of the research

The purpose of this project proposal is to provide information on the vitamin A status of

school-age children from Kibwezi district; and how it relates with morbidity patterns,

anthropometric status, and dietary intake of the children.

Your part in the research

You are requested to co-operate in this study by answering the questions in the questionnaire

and providing any other information as pertains to the study. You are also requested to allow

your child to participate in the study. Little blood samples will be drawn the children during

the study.

Possible benefits

The benefits from this study may not be directly anticipated but the results may be useful to

the relevant stakeholders (GoK and NGOs) in filling the gaps in service provision and

formulation of appropriate interventions to target the children of this age group.

83

Your child will get a free medical check up, and will also be checked for infection and malaria. You will be given all the immediate results from the tests that your child goes through and if needed your child will be referred to the health facility.

Possible Risks

There are no foreseen risks associated with the study.

Compensation

Your participation is voluntary and therefore, you will not receive any form of compensation.

Your Rights as a participant:

This study is protected by human ethics committee, the ministries of health, Kenya which approved this study through KNH/ERC

Volunteer Agreement

I have read the consent form describing benefits, risks and procedures for this study on *Micronutrient and macronutrient malnutrition, dietary diversity and morbidity burden* among schoolchildren in rural Kibwezi district, Kenya and I voluntarily agree to participate.

Name	Signature
Date	
For official use only	
I certify that the nature and purpose, the perpendicular participating in this study have been explain	otential benefits and possible risks associated with ned to the above individual
Date	Signature

Annex 2: Personal Information Questionnaire

Name child School: Standard Standard Birth date: Proof by (circle) Medical record Birth certificate Verbal recall Baptism card Birth announcement Other:	Date		Form fil	lled by		Id number	
Birth date: Proof by (circle) Medical record Birth certificate Applies and Birth announcement Birth announcement	Name child				1		1
Baptism card Birth announcement Birth announcement	School:					Standard	
Gender Male / Female Village Location Name caretaker Age caretaker to child form the child of	Birth date:	Proof by (circle)		Medical	record	Birth certificate	Verbal recall
Village Name caretaker Age caretaker Age caretaker Child Birth order of the child			Baptism	n card		Other:	
Name caretaker Age caretaker Age caretaker Child Relation of the caretaker to child Other: (specify) Birth order of the child Grand father Child of Age 1st wife Age 1st wife Age 2nd wife Age above 59 Education of the same pot) None Completed 1-4 of primary school Adult education Completed secondary school Adult education Age 50+ Sob+ ob+	Gender	Male / Female					
Age caretaker	Village				Location		
Relation of the caretaker to child Relation of the caretaker to child Grandmother Other: (specify) Birth order of the child Gth Other: (specify) Ist 2nd 3d 4th 5th Child of Ist Child of Ist Other: Child of Ist Age below 15 Age above 59 Education cof caretaker Age 15-59 Age above 59 Completed 1-4 of primary school Completed 5-8 of primary school Completed secondary school Adult education Other: Other: Other: Aunt Uncle Guardian Fin Guardian Other: Completed 5th Age below 15 Completed 5-8 of primary school Adult education Other: Other: Other: Other: Aunt Uncle Guardian Other:	Name caretaker						
caretaker child Grandmother Grand father Aunt Uncle Guardian Other: (specify) Birth order of the child 6th Other: Child of Ist Other: Child of Ist Ist Other: Child of Ist Age below 15 Age above 59 Education caretaker Age above 59 Education caretaker Other: Vocational training Attended secondary school Adult education Completed secondary school Adult education Other:	Age caretaker	<20	20-35		36-50	50+	
Other: (specify) Birth order of the child Other: (specify) Ist 2nd 3d 4th 5 th Other: Child of 1 st wife 2 nd wife 3 rd wife Household size (eating from the same pot) Age 15-59 Education of caretaker None Completed 1-4 of primary school Completed 5-8 of primary school Completed secondary school Adult education Other:		Mother	Father		Sister	Brother	
Birth order of the child 6th Other: Child of Ist wife 2nd 3d 4th 5th Child of Child of Ist wife 2nd wife 3rd wife Age below 15 Age below 15 Age above 59 Education of caretaker None Completed 1-4 of primary school Completed 5-8 of primary school Completed secondary school Adult education Other:	child	Grandmother	Grand fa	ather	Aunt	Uncle	Guardian
the child 6th Other: Child of 1st wife 2nd wife 3rd wife Age below 15 (eating from the same pot) Age 15-59 Education of caretaker Vocational training Attended secondary school Adult education Other:		Other: (specify)					
Child of 1st wife 2nd wife 3rd wife Household size (eating from the same pot) Age 15-59 Education of caretaker None Completed 1-4 of primary school Completed 5-8 of primary school Completed secondary school Adult education Other:		1st	2nd		3d	4th	5 th
Household size (eating from the same pot) Age 15-59 Education of caretaker None Completed 1-4 of primary school Completed 5-8 of primary school Attended secondary school Completed secondary school Adult education Other:		6th	Other:				
(eating from the same pot) Age 15-59 Age above 59 Education caretaker Of None Completed 1-4 of primary school Completed 5-8 of primary school Vocational training Attended secondary school Completed secondary school Above secondary school Adult education Other:	Child of	1 st wife	2 nd wife		3 rd wife		
Education of caretaker None Completed 1-4 of primary school Completed 5-8 of primary school		Total:			Age below 15		l
Caretaker Vocational training Attended secondary school Completed secondary school Adult education Other:		Age 15-59			Age above 59		
Vocational training Attended secondary school Completed secondary school Above secondary school Adult education Other:		None	Comple	ted 1-4 of	primary school	Completed 5-8 of	primary school
Adult education Other:	curcunci		Vocation	nal trainir	ng	Attended secondary school	
			Completed secondary school		dary school		school
Remarks			Adult ed	ducation		Other:	
	Remarks						

Annex 3: Household Information Questionnaire

Date	Form filled by			ID number		
Name child				School		
Water source at home used for drinking water	At compound < 5 min. walking	g distance	Type of water	Rainwater Borehole ->	In compound	Out compound
	> 5 min. walking	g distance	source	Well -> Piped River	Protected	Unprotected
				Other:		T ==== 2
Toilet facility at home	Shared		Type of toilet:	Pit latrine ->	With slab	Without slab
	Not shared			Flush toilet ->	To pit latrine	To piped sewer system
				Bush		
Main source of income	Animal/Animal	nroduct sales	Crop sales	Other:	Salaried emplo	ovment
for households:	Casual labour	product sales	Trade		Remittances	yment
(mark 1 option only)	Destitute (gifts,	begging)	Other:		I	
Does your household produce it's own food	No	Yes	through the		No	Yes
			land in store	Il food from your	No 2 4 6 8 10 1	Yes 2 months
			produced fo		2 4 0 6 10 1	.Z monuis
Do you experience shortage of food	No	Yes	If yes, which months	Jan Feb Jul Aug		May Jun Nov Dec
Does your household	No	Yes: (type)	Car	Motorbike	Bicycle	
have means of transportation			Other:			
Do members of your household own mobile phones	No	Yes				
Main cooking fuel used:	Wood	Charcoal	Gas	Electricity	Diesel	Parrafine
(mark 1 option only!)	Biogas	Solar	Other:			
Main light source: (mark 1 option only!)	None Other:	Electricity	Tin lamps	Kerosine lamps		Candles
Remarks						

Annex 4. Morbidity Questionnaire

Name Child: School: (name)							
Form Filled by:	Baseline / End study						
	(circle)						
wo weeks?	Yes		No				
If yes, how many times?							
ion?							
2. Did your child suffer from diarrhea (passage of three or more loose or liquid stools per day) in the last two weeks?							
	1	2	3	4			
on?							
3. Did your child suffer from a cough in the last two weeks?							
If yes, how many times?							
on?							
4. Did your child suffer from shortness of breath in the last two weeks?							
If yes, how many times?							
on?							
he last two weeks?	Yes		No				
	1	2	3	4			
on?							
the last two weeks?	Yes		No				
	1	2	3	4			
How many days at each occasion?							
7. Did your child had to vomit in the last two weeks?							
If yes, how many times?							
on?							
body pains in the last two weeks?	Yes		No				
	Form Filled by: wo weeks? on? (passage of three or more loose or liquid stools on? on the last two weeks? on? the last two weeks? on? the last two weeks? on? the last two weeks?	Form Filled by: Baseli (circle wo weeks? Yes 1 on? (passage of three or more loose or liquid stools Yes 1 on? the last two weeks? Yes 1 on?	Form Filled by: Baseline / Er (circle) wo weeks? Yes 1 2 on? (passage of three or more loose or liquid stools Yes 1 2 on? the last two weeks? Yes 1 2 on? he last two weeks? Yes 1 2 on? the last two weeks? Yes 1 2 on?	Form Filled by: Baseline / End study (circle)			

If yes, how many times?	1	2	3	4
How many days at each occasion?				
9. Did your child suffer from a headache in the last two weeks?				
If yes, how many times?	1	2	3	4
How many days at each occasion?				
10. Did your child suffer from red and watery eyes in the last two weeks?	Yes		No	·
If yes, how many times?	1	2	3	4
How many days at each occasion?				
11. Did your child have shivers and chills during the last two weeks?	Yes		No	
If yes, how many times?	1	2	3	4
How many days at each occasion?				
12. Did your child have blood in the stool in the last two weeks?	Yes		No	
If yes, how many times?	1	2	3	4
How many days at each occasion?				
13. Did your child have a bloody urine in the last two weeks?	Yes		No	
If yes, how many times?	1	2	3	4
How many days at each occasion?				
14. Was your child hospitalized in the last two weeks?	Yes		No	
If yes, how many times?	1	2	3	4
How many days at each occasion?				
15. Was your child receiving any health related treatment (for example medicine use)?	Yes		No	
If yes, how many times?	1	2	3	4
How many days at each occasion?				·
16. (asked when the child is female &>8 yrs) Did your child start menstruating already?	Yes		No	
	since			<u> </u>

What other health	related symptoms	were present in you	ir child in the last two	o weeks?

Annex 5: Individual Dietary Diversity Questionnaire

Was yesterday a feast day or a celebration day where you ate something unusual?

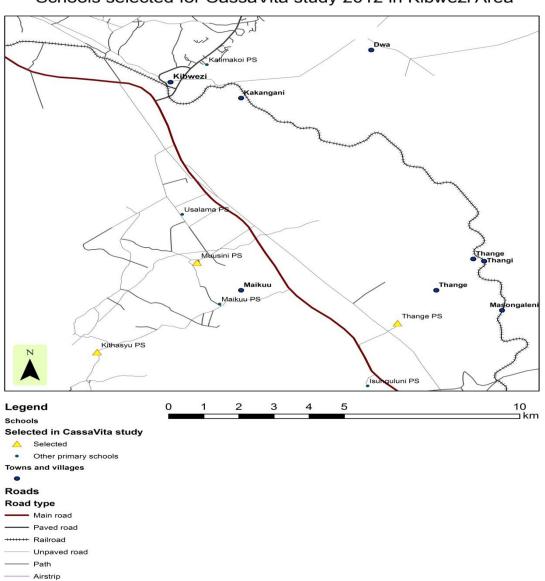
Question number	Food group	Examples	YES=1 NO=0
1	CEREALS	bread, noodles, biscuits, cookies or any other foods made from millet, sorghum, maize, rice, wheat + insert local foods e.g. ugali, nshima, porridge or pastes or other locally available grains	
2	VITAMIN A RICH VEGETABLES AND TUBERS	pumpkin, carrots, squash, or sweet potatoes that are orange inside + other locally available vitamin-A rich vegetables(eg. sweet pepper)	
3	WHITE TUBERS AND ROOTS	white potatoes, white yams, cassava, or foods made from roots.	
4	DARK GREEN LEAFY VEGETABLES	dark green/leafy vegetables, including wild ones + locally available vitamin-A rich leaves such as cassava leaves etc.	
5	OTHER VEGETABLES	other vegetables, including wild vegetables	
6	VITAMIN A RICH FRUITS	ripe mangoes, papayas + other locally available vitamin A-rich fruits	
7	OTHER FRUITS	other fruits, including wild fruits	
8	ORGAN MEAT (IRON-RICH)	liver, kidney, heart or other organ meats or blood-based foods	
9	FLESH MEATS	beef, pork, lamb, goat, rabbit, wild game, chicken, duck, or other birds	
10	EGGS		
11	FISH	fresh or dried fish or shellfish	
12	LEGUMES, NUTS AND SEEDS	beans, peas, lentils, nuts, seeds or foods made from these	
13	MILK AND MILK PRODUCTS	milk, cheese, yogurt or other milk products	
14	OILS AND FATS	oil, fats or butter added to food or used for cooking	
15	SWEETS	sugar, honey, sweetened soda or sugary foods such as chocolates, sweets or candies	
16	SPICES, CONDIMENTS, BEVERAGES	spices(black pepper, salt), condiments (soy sauce, hot sauce), coffee, tea, alcoholic beverages OR <i>local examples</i>	
Individual level only	Did you eat anything	(meal or snack) OUTSIDE of the home yesterday?	

Annex 6: Field Assistants Training Program

DAY	TIME	SUBJECT MATTER	LEARNING METHOD	LEARNING AIDS
	9.00-10.30 am	Introduction and Overview of the study O General objectives	o Lecture	Flip chartsMarker pensNote books
1	10.30-11.00 am	 Specific objectives 	Геа Break	o Pens/pencils
1	11.00-1.00 pm	Data collection techniques O Questionnaire filling (all sections), translating to Kiswahili	LectureRole playDemonstrationBrainstorming	Sample questionnaire
	1.00-2.00 pm		unch Break	C 1
	2.00-4.00 pm 4.00-5.00 pm	Data collection techniques (cont') Anthropometry Taking height Taking weight Recording measurements Ethics and conduct Professional conduct in the field Confidentiality Working hours Allowances	 Demonstration Role play Lecture Discussion 	 Seca scales Mortise tape Flip charts Marker pens Data form Flip charts Marker pens
		o Q & A		
	9.00-9.30 am	Recap of the previous day	o Discussion	Flip chartsMarker pens
2	9.30-1.30 pm	Pre-test questionnaire		o Questionnaire
	2.30- 3.30 pm	Lunch Break		
	3.30-5.00 pm	Revision of the questionnaire based on the results of pretest Conclusions and closing	o Discussion	o Filled questionnaire

Annex 7: Map of the selected schools

Schools selected for CassaVita study 2012 in Kibwezi Area



Annex 8: KNH-UoN ERC Approval



UNIVERSITY OF NAIROBI COLLEGE OF HEALTH SCIENCES P O BOX 19676 Code 00202

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KENYATTA NATIONAL HOSPITAL P O BOX 20723 Code 00202

Tel: 726300-9 Fax: 725272 Telegrams: MEDSUP, Nairobi 7th December 2011

Dr. Alice Mwangi Mboganie Applied Nutrition Programme College of Agriculture and Veterinary Sciences University of Nairobi

Dear Dr. Mwangi

RESEARCH PROPOSAL: "EFFECT OF PROVITAMIN A BIOFORTIFIED CASSAVA ON VITAMIN A STATUS OF PRIMARY SCHOOL CHILDREN IN KIBWEZI DISTRICT (CASSAVITA)" (P293/07/2011)

This is to inform you that the KNH/UON-Ethics & Research Committee has reviewed and <u>approved</u> your above revised research proposal. The approval periods are 7th December 2011 6th December 2012.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimens must also be obtained from KNH/UON-Ethics & Research Committee for each batch.

On behalf of the Committee, I wish you a fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of the data base that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely

Affirmutai PROFA N GUANTAI SECRETARY, KNH/UON-ERC

c. The Deputy Director CS, KNH

The Principal, College of Health Science, UON

The HOD, Medical Records, KNH

Co-investigators: Elise Talsma, Gloria Mbera

Annex 9: Pearson Correlation between different variable

				Household					Diarrheal		IDDS	
		RBP	Age	size	WAZ	HAZ	BAZ	ill	Days	Days	VitA	IDDS
RBP	Coef.		.219**	.026	.150*	.081	.110*	139	.624**	.643*	143**	058
	Sig.		.000	.593	.014	.098	.024	.187	.007	.018	.003	.237
Age	Coef.	.219**		.014	.147*	.054	033	062	.336	.352	063	046
	Sig.	.000		.779	.016	.267	.500	.553	.188	.238	.193	.346
Total	Coef.	.026	.014		.016	041	022	.046	243	342	008	083
household	Sig.	.593	.779		.790	.396	.657	.660	.364	.253	.877	.087
WAZ	Coef.	.150*	.147*	.016		.792**	.620**	123	096		108	100
	Sig.	.014	.016	.790		.000	.000	.355	.754		.077	.103
HAZ	Coef.	.081	.054	041	.792**		.128**	113	099	.155	.000	.000
	Sig.	.098	.267	.396	.000		.009	.282	.705	.614	.998	.995
BAZ	Coef.	.110*	033	022	.620**	.128**		074	.160	.066	087	100 [*]
	Sig.	.024	.500	.657	.000	.009		.484	.539	.830	.073	.041
Days ill	Coef.	139	062	.046	123	113	074		.132	.420	.026	093
	Sig.	.187	.553	.660	.355	.282	.484		.640	.260	.803	.373
Diarrheal	Coef.	.624**	.336	243	096	099	.160	.132			245	200
Days	Sig.	.007	.188	.364	.754	.705	.539	.640			.344	.442
Vomit Days	Coef.	.643*	.352	342		.155	.066	.420			.000	083
	Sig.	.018	.238	.253		.614	.830	.260			1.000	.788
IDDS VitA	Coef.	.143**	063	008	108	.000	087	.026	245	.000		.780**
	Sig.	.003	.193	.877	.077	.998	.073	.803	.344	1.000		.000
IDDS	Coef.	058	046	083	100	.000	100*	093	200	083	.780**	
	Sig.	.237	.346	.087	.103	.995	.041	.373	.442	.788	.000	

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

*. Correlation is significant at the 0.05 level (2-tailed Coef –Pearson Coefficient

Annex 10: Univariate analysis of the factors associated with RBP levels

Tests of Between-Subjects Effects

Dependent Variable:RBP_Ng_ml

	Type III Sum of				-	
Source	Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	210.634 ^a	43	4.898	2.575	.000	.234
Intercept	37.780	1	37.780	19.864	.000	.052
Hb_mmol_L	.237	1	.237	.124	.724	.000
CH_BirthOrder	2.346	1	2.346	1.233	.267	.003
Household_size	.023	1	.023	.012	.913	.000
Child_age_yrs	13.555	1	13.555	7.127	.008	.019
IDDS	3.594	1	3.594	1.890	.170	.005
IDDS_VitA	4.288	1	4.288	2.254	.134	.006
DaysIII	1.330	1	1.330	.699	.404	.002
ED_CT	.000	0				.000
CT_AgeG	32.899	2	16.450	8.649	.000	.046
CH_GENDER	2.459	1	2.459	1.293	.256	.004
CT_Rel	21.754	6	3.626	1.906	.079	.031
CT_EduC	4.211	4	1.053	.553	.697	.006
INC_SOURCE	38.177	8	4.772	2.509	.012	.053
LIVESTOCK	5.099	1	5.099	2.681	.102	.007
FOOD_PROD	1.232	2	.616	.324	.724	.002
MEANS_TRANSPORT	5.518	1	5.518	2.901	.089	.008
Toilet_facility	29.153	3	9.718	5.109	.002	.041
Treat_Drinking_water	2.633	2	1.317	.692	.501	.004
Q1_HasChildBeenSick	1.870	1	1.870	.983	.322	.003
ConsumeVitA	.601	1	.601	.316	.574	.001
Error	688.519	362	1.902			
Total	11708.920	406				
Corrected Total	899.153	405				

a. R Squared = .234 (Adjusted R Squared = .143)