# Effects of Initial Nutritional Status on the Responses to a School Feeding Programme among School Children Aged 6 to 13 Years in the Millennium Villages Project, Siaya, Kenya

by
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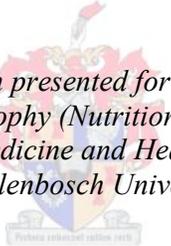
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DECLARATION

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ABSTRACT

Aim: To assess the effects of initial nutritional status on the responses to a school feeding programme (SFP) among school children in the Millennium Villages Project (MVP), Siaya District, Kenya.

Objectives: To establish baseline data on the dietary intake, prevalence of undernutrition, body composition, prevalence of anaemia, body iron stores and vitamin A status in order to assess the impact of the SFP over a 30-month period on the same outcomes. Further, the study assessed the effect of initial nutritional status on the responses to the SFP on growth, the prevalence of anaemia, body iron stores and vitamin A status.

Methods: In the MVP, a school meal additional to the children's usual daily food intake was provided consisting of locally available foods (containing whole fish *omena* and beef). The meals contributed approximately 25% of the Estimated Energy Requirement (EER), 70% of the Estimated Average Requirement (EAR) for protein, and 18% of EAR for fat. A school meal, additional to the children's usual daily food intake, was introduced to the control group 6 months into the study as a project scale up initiative. The meal provided 15% EER, 49 % EAR for protein and 10% EAR for fat. A total of 235 children participated in the study and were followed up for 30 months, with 118 in the MVP and 117 in the control group. Statistical analysis included descriptive, Pearson's chi-square test, repeated measures ANOVA and multivariate logistic regression models.

Results: The mean subject age was 7.9 years (2.0 SD) at baseline, and half (51.4%) were boys. More than half of the children (66%) had energy intakes less than the EER. At baseline, the prevalence of stunting, wasting and underweight was 16.9%, 6.0% and 3.6% respectively. Prevalence of linear growth deficit based on height-for-age z-score ≤-1 standard deviation was 48%. Anaemia was higher in the MVP group (82.2%; $P < 0.0001$) compared to controls (58.1%) while depleted body iron stores was observed in 10.7% of the children. Half of the children in the control group and 30% in the MVP group had an inadequate vitamin A status while 11% of the children had infection/inflammation. At six months after initiation of the intervention, anaemia prevalence was reduced to 41.2% among the controls and 9.3% in the intervention group ($P < 0.001$). Among the MVP group, children with initial inadequate nutritional status based on weight-for-age z-score WAZ ≤ -1 SD had a higher ($P < 0.01$) height velocity (2.3 cm/six months) by the 24th month study interval compared to those who had an initial adequate nutritional status based on weight-for-age z-score WAZ > -1 SD.

Conclusion: SPF menus were associated with potential for improved growth, gain in lean body mass and reduced anaemia prevalence when inadequate nutritional status was present at baseline.

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OPSOMMING

Doel: Om die verband tussen skoolkinders se aanvanklike voedingstatus en die respons op 'n skoolvoedingsprogram (SVP) in die Millennium Villages Project (MVP), Siaya-distrik, Kenia, te bepaal.

Doelwitte: Om basislyn data te versamel ten opsigte van dieetinname, prevalensie van ondervoeding, liggaamssamestelling, prevalensie van anemie, ysterstore en vitamine A status ten einde die impak van die SVP oor 'n 30-maande periode op genoemde uitkomste te bepaal. Verder het die studie ook die effek van aanvanklike voedingstatus op die respons tot die SVP bepaal ten opsigte van groei, die prevalensie van anemie, ysterstore en vitamine A status.

Metode: In die MVP is 'n skoolmaaltyd addisioneel tot die kinders in beide studiegroepe se daaglikse voedselinname voorsien. Die SVP se spyskaarte het bestaan uit plaaslik beskikbare voedsel (bevattende die vissoort *omena* en beesvleis) en het ongeveer 25% van die kinders se geraamde energievereistes (EER), 70% van hul proteïenvereistes (EAR) en 18% van hul vetvereistes (EAR) voorsien. 'n Skoolmaaltyd is addisioneel tot die kinders in die kontrolegroep se gewoontelike daaglikse voedselinname ingesluit 6 maande na aanvang van die studie as deel van die MVP se uitbreidingsinisiatief. Dié maaltyd het in 15% van die kinders se energievereistes (EER), 49% van hul proteïenvereistes (EAR) en 10% van hul vetvereistes (EAR) voorsien. Altesaam 235 kinders is by die studiegroep ingesluit – 118 in die MVP en 117 in kontrolegroepe – en is vir 30 maande bestudeer. Statistiese ontleding het beskrywende ontleding, Pearson se chi-kwadraattoets, ANOVA met herhaalde metings, en multivariansie logistiese regressiemodelle ingesluit.

Resultate: Die gemiddelde ouderdom van die kinders by basislyn was 7.9 jaar (2.0 SD) en die helfte (51.4%) van die respondente was seuns. Meer as die helfte van die kinders (66%) het 'n energie-inname minder as die EER getoon. By basislyn was die prevalensie van belemmerde groei, uittering en ondergewig onderskeidelik 16.9%, 6.0% en 3.6%. Die voorkoms van onvoldoende lengtegroei gebaseer op lengte-vir-ouderdom z-telling $\leq$ -1SD was 48%. Anemie was hoër in die MVP groep (82.2%; p<0.0001) vergeleke met die kontroles (58.1%), terwyl 10.7% uitgeputte ysterstore getoon het. Onvoldoende vitamine A status het voorgekom in die helfte van die kinders in die kontrolegroep en 30% van die MVP groep, en infeksie / inflammasie was teenwoordig in 11% van die kinders. Die voorkoms van anemie op ses maande na aanvang van intervensie het verbeter tot 41.2% in die kontrolegroep en 9.3% in die intervensiegroep (P < 0.001). Op 24 maande het kinders met aanvanklike onvoldoende voedingstatus (WAZ $\leq$ -1SD) in die MVP-groep groter lengtetoename (2.3

cm/6 maande) getoon as hul groepgenote met aanvanklike voldoende voedingstatus gebaseer op WAZ $\geq$ -1 SD (P < 0.01).

Gevolgtrekking: Die skoolvoedingsprogram spyskaarte het die potensiaal getoon tot 'n verbetering in groei, toename in maer liggaamsmassa en 'n verlaagde voorkoms van anemie onder kinders wie se basislynvoedingstatus onvoldoende was.

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Contributions by Principal Researcher and Fellow Researchers

The principal researcher (Peninah Masibo) developed the research protocol, planned the study and undertook all the data collection procedures with the help of study specific trained research assistants. The principal researcher also trained the research assistants and supervised their field activities. She captured all the data and conducted data analysis with the assistance of a Statistician (Prof. DG Nel), interpreted the data and drafted the dissertation. Prof. Labadarios, Prof. Herselman, Prof. Deckelbaum and Dr Ettyang provided input at all stages on the research protocol development, data collection, data analysis and writing the dissertation.

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LIST OF ACRONYMS

ACC/SCN	Administrative Committee on Coordination/Sub−Committee on Nutrition
AI	Adequate intake
ALV	African leafy vegetables
AMDR	Acceptable macronutrient distribution range
ANOVA	Analysis of variance
ASF	Animal source foods
BMI	Body mass index
CDC	Centers for Disease Control
CI	Confidence interval
CRABP	Cellular retinol-binding protein
CRP	C-reactive protein
CV	Coefficients of variation
DALYS	Disability adjusted life years
DFE	Dietary folate equivalent
DGLV	Dark green leafy vegetables
DHA	Docosahexaenoic acid
DHS	Demographic and Health Survey
DNA	Deoxyribonucleic acid
DRI	Dietary reference intake
DXA	Dual-energy X-ray absorptometry
EAR	Estimate average requirement
EDTA	Ethylenediaminetetraacetic acid
EER	Estimated energy expenditure
EPA	Eicosapentaenoic acid
ESAR	East and Southern African Region
FAO	Food Agricultural Organization
GLM	Generalised linear model
HAZ	Height-for-age z score
HB	Haemoglobin
HDL	High density lipoproteins
HPLC	High-performance liquid chromatography
ICCIDD	International Council for the Control of Iodine Deficiency Disorders
ICRAF	International Center for Research in Agroforestry
ID	Iodine deficiency
IDA	Iron deficiency anaemia
IDD	Iodine deficiency disorders
IOM	Institute of Medicine
IQ	Intelligence quotient
IU	International units
KMTC	Kenya Medical Training College
LDL	Low density lipoprotein
LM	Lower midland

MCV	Mean cell volume
MDG	Millennium Development Goals
MUAC	Mid-upper arm circumference
MVP	Millennium Villages Project
NAR	Nutrient adequacy ratio
NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio
RAR	Retinoic acid receptor
RBC	Red blood cell
RBP	Retinoid-binding proteins
RDA	Recommended dietary allowance
RDI	Recommended daily intake
RXR	Retinoid X receptors
CSC	Center for Statistical Consultation
SFP	School feeding programme
STFR	Serum transferrin receptors
UL	Upper limit
USDA	United States Department of Agriculture
VAD	Vitamin A deficiency
WCAR	West and Central African Region
WFP	World Food Program
WHO	World Health Organisation
WHZ	Weight-for-age z-score
WISC	Wechsler intelligence scale for children - revised

DEFINITION OF TERMS

Bioavailability: The fraction of an ingested nutrient that is absorbed and available for utilisation and/or storage.[1]

Catch-up growth: Acceleration of growth after a period of growth retardation.[2]

Dietary diversity: The number of individual food items or food groups consumed over a given period of time.[3]

Dietary or nutritional supplement: Any product that contains nutrients or other substances with a nutritional or physiological effect, such as vitamins, minerals, foods, botanicals, amino acids, and is intended to supplement the usual intake of these substances that may be missing or may not be consumed in sufficient quantities in a person's diet.[4]

Dietary reference intakes: Quantitative reference values for recommended intakes and tolerable upper intake levels for a range of nutrients.[5]

Food fortification: Addition of one or more essential nutrients to a food, whether or not it is normally contained in the food, for the purpose of preventing or correcting a demonstrated deficiency of one or more nutrients in the population or specific population groups.[6]

Food-based approaches: Methodologies to promote the availability and consumption of a variety of micronutrient-rich foods to improve the micronutrient status of populations.[7]

Dietary reference intakes: A system of nutrient recommendations from the Institute of Medicine (IOM) of the United States National Academic of Sciences that is intended to serve as a guide for good nutrition and provide the scientific basis for the development of food guidelines in both the United States of America and Canada. These nutrient reference values are specified on the basis of age, gender and life stage and cover more than 40 nutrient substances.[8]

Growth: An increase in the size of an organism or part of an organism, usually as a result of an increase in the number of cells.[9]

Growth failure: Growth failure is the term that describes a growth rate below the appropriate growth velocity for age.[10]

Initial nutritional status: The height-for-age, weight-for-age, BMI-for-age < -1 standard deviation z-scores from the World Health Organisation (WHO) international reference standard[11] as measured before the start of the school feeding programme.

Linear growth failure: The process of failure to reach linear growth potential as a result of suboptimal health and/or nutritional conditions.[12]

Millennium Villages Project (MVP): A project by the Earth Institute of Columbia University and the United Nations Millennium Project aimed at empowering and working with impoverished communities in rural Africa to achieve the United Nations Millennium Development Goals (MDG).[13]

Overnutrition: A chronic condition where intake of food is in excess of dietary energy requirements, resulting in overweight and/or obesity.[14]

School feeding programme: Provision of meals or snacks at school to reduce children's hunger during the school day.[15]

Undernutrition: The outcome of insufficient food intake and repeated infectious diseases, which includes being underweight for one's age (low weight-for-age), too short for one's age (low height-for-age), and too thin for one's height as measured by low weight-for-age or BMI-for-age, as well as deficient in vitamins and minerals.[16]

Z-score: The deviation of an individual's value from the median value of a reference population, divided by the standard deviation of the reference population.[11]

1   CHAPTER ONE: INTRODUCTION, LITERATURE REVIEW AND MOTIVATION FOR THE STUDY

1.1   Introduction

School-age children make up a considerable portion of the world's populace, approximated at 15% of the people in industrialised countries and 24% in developing countries.[6,17] Three out of ten people in Kenya are school-aged children,[18] making this age group an important population group. The school-age period is characterised by an increased need for nutrients in preparation for the adolescent years. This dynamic period of growth and development forms a foundation for good health as children go through physical, emotional and social changes.[19] During this period, children tend to change their eating habits, which in turn influences food choices, nutrient intake and nutritional status. Good nutrition is an essential determinant of the health, physical growth, development, educational performance and progression in life of school-age children. Feeding habits established during this life period may remain throughout life.[7] School children spend an average of seven hours each day attending school. What they eat at school contributes largely to their dietary sufficiency and this school-age period provides an opportunity to improve their dietary intake and nutrient adequacy, especially in resource-restrained settings. In addition, good nutrition is known as a cornerstone of survival, health and development in present and successive generations.[20]

Most of the world's children aged between 5 and 15 years attend schools, although many do so under difficult conditions,[21] and many stop schooling before the end of high school.[22] Health and nutrition for school-age children has received the least attention globally, as the main focus of international efforts and programmes has been on reducing infant and child mortality.[23] School-age children comprise a group at risk of nutritional deficiencies.[24] Therefore, the health and nutrition of school children is of critical importance and calls for attention to be given to this age group, as it forms the backbone of a country's future economy, since children have always been viewed as investments. This has been demonstrated further by the recognition of the health of school children as a priority in international health in the World Bank development report.[25]

1.1.1   Normal growth and development of school-age children

The middle childhood and pre-adolescent years are characterised by increased physical, cognitive and emotional growth and development in preparation for the physical and emotional changes in adolescence.[26] Growth is steady during the school-age years, with a slower growth velocity than in infancy.[27] The average height and weight gain among children between the ages of 5 and 10 years is 30 centimetres and 12 kilogrammes respectively.[28] Weight gain increases slowly, while height gain gradually slows during the period from 5 to 10 years old. The contribution of fat to total weight gain is constant for boys, and gradually increases for girls. The average weight gain per day for children

aged 7 to 10 years is 5 to 12 g/d while the average height gain is 4 to 5 mm/month.[28] Boys are heavier than girls up to the age of 8 years, following which the girls grow to be heavier due to a gain in body fat associated with pubertal changes.[29] Normal growth and development in the adolescent and pre-adolescent period are regulated by an intricate interaction of hormonal influences, tissue responsiveness and nutrition, in addition to metabolic and genetic factors.[30] The interaction of environmental and genetic factors influences the physical growth of this age group.

The main environmental factors affecting the growth of school-age children in resource-poor settings include poor food consumption patterns, inadequate sanitation facilities and poor hygiene practices, frequent illness, as well as a lack of access to proper health facilities.[31] Periodic monitoring of growth amongst the school-age population is important in order to detect any alterations in normal growth patterns.

1.1.2   Nutritional requirements of school-age children

The demands for growth and the maintenance of physical activity cause an increased need in children for nutrients.[29] Nutrient requirements are determined using the dietary reference intakes (DRI), which were developed by the Food and Nutrition Board of the National Academy of Sciences' Institute of Medicine.[32] The DRIs include the estimated average requirement (EAR), recommended daily allowances (RDA), adequate intake (AI), tolerable upper intake level (UL), acceptable macronutrient distribution range (AMDR) and estimated energy requirement (EER).[32] The proportion of a population with usual nutrient intake below the EAR for age and sex is considered to have inadequate nutrient intake and is used to estimate the prevalence of inadequate intake.[33]

Body requirements for most nutrients increase steadily for this age group (school-age children), as reserves are being prepared for the approaching growth spurt and adolescence period. The energy and protein needs of school-age children vary depending on body size, growth patterns as well as physical activity levels. The estimated energy requirement (EER) for school-age children ranges from 1,546 kcals per day for 4-year-old boys, to 2,804 kcals for 14-year-old boys. For girls, the EER ranges from 1,455 kcals 4-year-olds to 2,309 kcals for 14-year-olds.[34] These EERs are based on an active level of physical activity. The protein RDA for children 4 to 13 years old is given as 0.95 g/kg/day.[34] A diet consisting of 30% total calories from fat is able to meet the daily energy and fat requirements of school-age children.[35] Of particular importance in this age group are vitamins and minerals, which are needed to maintain growth and physical activity. Poor eating habits coupled with an inadequate intake of nutrient-dense foods exposes school-age children to the risk of

deficiencies in iron, thiamin, vitamin A and calcium.[36] The EAR for vitamin A increases with age, from 275 µg/day for children aged four to eight years to 445 µg/day for boys aged 9 to 13 years and 420 µg/day for girls of the same age.[34,37] During periods of rapid growth there is an increased demand for iron, which is required for increased haemoglobin mass and concentration, especially in boys,[38] and to accommodate the losses experienced by girls through high menstrual loss or teenage pregnancies.[39] The EAR for iron for children aged four to eight years is 4.1 mg/day. This requirement differs in boys and girls from 9 years of age.[37] Boys require 5.9 mg/day between 9 and 13 years, increasing to 7.7 mg/day between 14 and 18 years. Girls require 5.7 mg/day from 9 to 13 years, with an increase to 7.9 mg/day between 14 and 18 years.[37]

Calcium is a critical nutrient for this age group, as it is required for the formation metabolism and maintenance of bone and teeth. Calcium is also important for regulating the heartbeat and controlling blood pressure and muscle contraction. The EAR for calcium increases from 800 mg/day for children aged four to eight years, to 1,100 mg/day for children aged 9 to 18 years.[40] The sources of food that are rich in calcium, including milk and milk products as well calcium-fortified foods, may not be easily accessible by this age group.

1.1.3 Nutrient intake of school-age children

Studies conducted in middle and low-income countries where nutrient intake is likely to be affected by food unavailability at the household level report low nutrient intake by school children. In KwaZulu-Natal, South Africa, Faber and colleagues[41] reported that energy intake was below the RDA, but protein intake was adequate, although it was mainly from plant sources in school-age children. A study in the North West Province of South Africa further reported an inadequate energy intake of 8 013 kJ compared to an RDA of 10 500 kJ for children aged 11 to 14 years.[42]

A few nutrient adequacy studies have been carried out among school-age children in Kenya, although the reporting of adequacy of dietary intake was done using various methods. For example, a study in Suba District, Nyanza Province measured nutrient intake among 1,383 school children aged 5 to 17 years using the nutrient adequacy ratio (NAR),[43] while in the rural Western Province, Walingo and Musamali measured adequacy of nutrient intake using DRIs.[44] In Embu District, Eastern Province, the adequacy of nutrient intake was measured with the previous version of the recommended dietary allowances (RDA).[45] The findings of these studies show inadequate levels of energy intake by school-age children in Kenya, while protein intake is shown to be adequate or above the recommended intake. Among the children in Suba District, Nyanza Province, the NAR for energy decreased with increasing age and varied on the basis of sex.[43] Boys aged 17 years had a

lower energy NAR of 60.8% compared to their 10-year-old counterparts, who had a NAR for energy of 90.4%. Similarly, the NAR for energy intake among 17-year-old girls was 88.4%, compared to 94.9% among girls aged 11 years.[43] NAR for protein intake exceeded 100% in boys up to 13 years old, but decreased to 85.5% by the age of 17, while the NAR for protein intake remained above 100% for all girls aged 5 to 17 years, although it was highest among the younger children.[43] Mean energy intake among school children in rural Western Province was reported as 77% of the RDA, while protein was 117.6% of the RDA. In the Eastern Province study, the mean energy intake was almost equal to the RDA, while protein intake was 50 g,[44] which is equivalent to 147% of the RDA for children aged 6 to 7 years.[45] The differences in the methodology of reporting nutrient adequacy in the Kenyan studies limits the ability to compare these findings and to determine the nutrient intake adequacy among school-age children. These studies, conducted in three different provinces, show a general disparity in the intake of energy and there is a need for further studies to establish the nutrient intake among Kenyan school children, utilising DRIs for comparability with studies from other parts of the region.

The adequacy of the intake of micronutrients such as vitamin A, iron, B vitamins, calcium and magnesium by school children varies across the world. In the United States of America (USA), Suitor and Gleason[33] reported an adequate intake of iron, B vitamins, folate, calcium and magnesium in school children. In the Mexican national survey, the estimated median micronutrient intake was above 100% adequacy for iron, vitamin A, vitamin C and zinc, while the percentage adequacy for folic acid and calcium was lower, at 82% and 75% respectively.[46] The percentage adequacy for iron, phosphorus, magnesium, and vitamins A, C, E and B was found to be above 100% in the Taiwan national survey, while the percentage adequacy for calcium was 64%.[47]

Studies from the African region that have reported micronutrient intake by school children generally reveal a low intake. For instance, in South Africa, calcium intake was low in school children from KwaZulu-Natal province, with 67% meeting the RDA,[41] while the daily intake of zinc was below the EAR for 16% of children aged 7 to 8 years and 83.7% for children aged 9 to 11 years living in an informal settlement in the Vaal Region of Gauteng province.[48]

A variety of micronutrients are likely to be low in the diets of school children in Kenya, particularly iron, zinc, vitamin B_{12}, riboflavin, calcium and vitamin A.[49] Adequacy of nutrient intake measured using the nutrient adequacy ratio (NAR) in the Suba District study showed NARs of at least 100% for thiamine, vitamin A and vitamin C, while iron and niacin had low NARs for children aged 7 to 17 years, ranging from 94% to 28.8% at 17 years for iron, and 94.8% for niacin for 7-year-olds. The

lowest NAR was 66.7% for children aged 10 years.[43] In the same study, sodium, potassium and calcium intake were inadequate. Low intakes of iron, zinc, vitamin B_{12}, calcium, vitamin A and riboflavin have been observed in studies of school children in the Embu region of the Eastern Province by Neumann et al.[50] The intake of vitamin B_{12} was reported as 0.4 µg, riboflavin was 1.1 mg, vitamin A was 320 µg RE, calcium was 261 mg, and available iron was 1.49 mg, while available zinc was 1.5 mg.[50] Further reported in the same group of studies in Embu was that the mean intakes of vitamin B_{12} and A, calcium and zinc were found to be lower than the RDIs for children aged 7 to 9 years.[49] Iron, zinc, vitamin B_{12} and calcium intakes were below two thirds of the FAO/WHO recommended intakes in 67 to 100% of the children.[51] Earlier studies in the same area by the same group of researchers reported a 86.9% prevalence of inadequate intake of vitamin B_{12}, a 1.6% prevalence of inadequate riboflavin intake, a 91.2% prevalence of inadequate calcium, a 31.2% prevalence of inadequate iron intake and a 29.5% prevalence of inadequate zinc intake.[52]

1.2 Nutritional Status of School Children

Despite the promotion of health and nutrition services in primary schools, the data available on the actual nutritional status of school-age children in developing countries and countries in transition is limited.[53] Longitudinal studies on the growth of school-age children are limited, despite the importance of this age group, which comprises almost a quarter of the population in developing countries.[17] The available studies indicate that the patterns of prevalence of undernutrition in school-age children are similar to the patterns seen in preschool children, which offer more robust data.[31] School-age children are faced with a range of nutritional problems that include stunting, underweight, anaemia, iron deficiency, vitamin A deficiency and iodine deficiency.[53] Other micronutrient deficiencies reported in this population, although to a lesser extent, include folate, riboflavin, niacin, zinc, and vitamin B_6 and B_{12}. In addition, overweight and obesity are increasing problems among school-age children in countries experiencing a nutritional transition.[53]

1.2.1 Undernutrition among school-age children

Undernutrition among school-age children, as measured by stunting, underweight and wasting, is a matter of public health concern around the world.[53] Stunting is widely believed to occur cumulatively from early childhood and tends to increase in magnitude throughout the school-age years.[31] Contrary to this widely believed factor, some studies have found that the onset of stunting can occur among school children. Stoltzfuls et al.[54] reported a dramatic increase in stunting with age, from 14% in 7 year-olds to 83% by 13 years of age. In addition, a study in Uganda reported a higher likelihood of undernutrition among older children (10 to 14 years) compared to younger ones (6 to 9 years),[55] which suggests an onset of stunting in late childhood. Undernutrition among

school-age children has been reported in rural populations and in informal urban settlements in low-income countries. In Sri Lanka's dry Moneragala District, half of the school children in grades 1 to 5 were reported as moderately underweight or wasted, while one fifth were moderately stunted.[56] The prevalence of stunting among school children aged between 7 and 13 years in Baghdad was 18.7% and increased with age, from 7.7% among 7-year-olds to 22.4% among 12-year-olds.[57] The prevalence of wasting, stunting and underweight was reported as 21.2%, 47.4% and 51.7% respectively in a study among the school-age children of tea garden workers in Assam, India.[58]

Studies in African countries indicate varying levels of undernutrition among school-age children. TABLE 1.1 contains a summarised description of selected studies on the nutritional status of school children in African countries, outlining the study designs, setting, participants, key outcome variables and the main findings. These studies were selected for review based on their availability, their study population of school children in sub-Saharan African countries, and the fact that they were published in 2000 or later. One systematic review was included for a global perspective. These studies show the presence of undernutrition, as assessed by stunting, wasting and underweight as well as varying degrees of anaemia and micronutrient deficiencies, across the region. The first nationwide survey of the health of school children in Chad reported incidences of 18.7%, 16.5% and 4.6% for stunting, underweight and wasting respectively.[59] In Ethiopia, a national survey among school children aged 5 to 14 years reported 22.3% stunting and 23.1% too thin for age, based on low body mass index (BMI) for age.[60] The prevalence of stunting in school children in Zanzibar was reported as 14% among 7-year-olds and increased to 60% by 10 years of age.[54]

No national survey has been done on the health and nutritional status of school-age children in Kenya. However, a few studies (TABLE 1.1) have been conducted in different regions that have reported varying prevalence of undernutrition in this age group. A prevalence of 30.2% stunting, 14.9% underweight and 4.5% wasting was reported in a peri-urban slum in Nairobi.[61]

A study in the Embu District of Eastern Province, Kenya reported a higher prevalence of stunting among school children, at 19.4%, with a 4.6% prevalence of severe stunting and 30% underweight.[50] Another study in Makueni district in the same province reported a higher prevalence of undernutrition, with 56.4% stunting and 30% underweight.[62] In Western Kenya, the prevalence of stunting among school children has been reported as 18.5%, with 5.9% wasting and 15.8% underweight.[63] These studies point to the fact that undernutrition remains a challenge for school-age children in Kenya, although the main concern in public health and nutrition intervention programmes in the country is focused on nutrition for children under the age of five.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Beasley et al. (2002)[59]	National cross-sectional survey	1,024 school children aged 9 to 10 years in Chad	Anthropometry: • Height, weight Micronutrient status: • Haemoglobin concentration • Clinical examination of the thyroid gland Stool samples: • Intestinal worm infestation	Anthropometry: • Prevalence of stunting was 18.7%, underweight was 16.5% and wasting was 4.6% • 21.1% were anaemic (< 110 g/dl) • 23.3% had goitre Intestinal worm infestation: • Presence of intestinal infestation
Hall et al. (2008)[60]	National cross-sectional survey	7,572 school children in grade 3 and 4, Ethiopia	Anthropometry: • Height, weight Dietary intake: • Fruit, vegetables and meat consumption Micronutrient status: • Haemoglobin concentration • Serum retinol	Anthropometry: • Prevalence of stunting was 22.3%, while 23.1% of the children were underweight • Boys had a lower mean height-for-age z-score than girls Dietary intake: • 23.8% consumed fruit the day before the survey • 26.3% consumed vegetables • 38.4% consumed both fruit and vegetables • 16.5% consumed meat • Age was correlated with reported fruit, vegetable and meat intake Micronutrient status: • The prevalence of anaemia was 9.8%, while 13.8% had goitre and 0.8% had xerophthalmia • Girls were more at risk of goitre than boys • Children with xerophthalmia were more likely not to have eaten fruit and vegetables on the previous day Infections: The prevalence of hookworm infestation was 7.7%

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Chesire (2008)[61]	Cross-sectional survey	383 school children aged 6 to 12 years; peri-urban slum, Nairobi, Kenya	Anthropometry: • Height, weight School attendance	Anthropometry: • The prevalence of wasting was 4.5%, while 14.9% were underweight and 30.2% were stunted • Stunting and underweight were associated with sex, age, school attendance and household income
Wilson et al. (2010)[62]	Cross-sectional survey	272 school-age children 4 to 17 years; in a rural setting, Makueni District, Kenya	Anthropometry: • Height, weight Stool samples: • Intestinal worm infestation; *S. mansoni*	Anthropometry: • 56.4% stunting, 25.2% wasted Infections: • Presence of *S. mansoni* infection • Age, sex and *S. mansoni* infection associated with stunting
Francis (2012)[55]	Cross-sectional survey	432 primary school children aged 6 to 14 years, Central Uganda	Anthropometry: • Weight, height, MUAC Stool samples: • Intestinal worm infestation	Anthropometry: • Children 10 to 14 years of age were more likely to be stunted compared to those aged 6 to 9 years • Stunting was higher in urban slums than in rural areas • Boys were three times more likely to be underweight compared to girls • Moderate acute malnutrition was higher among children 11 to 14 years of age compared to those aged 6 to 10 years Helminthic infections: • The prevalence of helminthic infections was 16 % • Most common helminthic infection was hookworm (67%) and *Trichiuris trichiura* (19%) • Helminthic infection was higher in rural compared to urban areas (9.6% versus 6.3%) Underweight, stunting and moderate acute malnutrition were associated with helminthic infections

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Neumann et al. (2003)[50]	Randomised control trial; three school feeding interventions Meat, milk and energy groups offering an average of one fifth of the required daily energy intake Mid-morning snack for 23 months on school days	554 primary school children in grade 1 in a rural setting in Embu District, Kenya	Anthropometry: • Weight, height, head circumference, triceps and subscapular fat folds Dietary intake: Micronutrient status: • Haemoglobin concentration • Biochemical markers for vitamin B_{12}, vitamin A, iron, zinc, copper, folate and vitamin B_2	Baseline results Anthropometry: • 19.4% were stunted, with 4.6% severe stunting, and 30% were underweight • Stunting higher in boys than girls • Stunting and underweight increased with age • Leanness with triceps and subscapular fat folds < 5th percentile based on NHANES reference data was observed Dietary intake: • Low intake of animal-source proteins • Inadequate micronutrient intake Micronutrient status: • Almost half of the population had anaemia (48.9%), and 2.7% had severe anaemia • Prevalence of low serum iron was 52.4%, low serum zinc was 65.6%, while the prevalence of low plasma ferritin concentration was 6.3% • 30.5% and 37.7% of children had severe and moderate vitamin B_{12} deficiency respectively • 22% and 68.6% had moderate and severe vitamin A deficiency respectively Infections: • Infections were highly prevalent, reflected by elevated CRP (17.8%) • High presence of parasitic infestation • Prevalence of malaria (50%), with 45.4% having an enlarged spleen

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Siekman et al. (2003)[64]	Same as Neumann (2003)[50] Results include data up to 9 months	Same as Neumann (2003)[50]	Anthropometry: Weight, height, head circumference Micronutrient status: • Haemoglobin concentration • Biochemical markers for vitamin B_{12}, vitamin A, iron, zinc, copper, folate and vitamin B_2 Stool samples: • Intestinal worm infestation Infections Malaria CRP	Micronutrient status: • Significant increases in the concentration of haemoglobin, serum iron, plasma vitamin B_{12} and retinol across all groups by one year post intervention • Vitamin B_{12} plasma concentration increased significantly for the meat and milk groups compared to the reduction observed among the energy and control groups • No significant changes between groups for zinc, iron, vitamin A and copper Infections: • Higher decrease in prevalence of intestinal infestations in meat and milk groups compared to controls • CRP was significantly associated with changes in haemoglobin, copper, vitamin B_{12}, retinol, riboflavin and ferritin • Correlation between decreases in CRP and ferritin • Negative and significant correlation between change in plasma retinol and changes in CRP
Omwami et al. (2011)[64]	Same as Neumann (2003)[50]		School attendance rates	Improved school attendance by intervention groups

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Gewa et al. (2007)[65]	Same as Neumann (2003)[50]	525 children in the same study as Neumann[50]	Cognitive function assessment: • Non-verbal meaning test • Arithmetic test Digit span forward and backward test	Cognitive function: • Daily iron and zinc intake positively predicted cognitive function gains • Daily available iron and zinc intakes were positive predictors of RCPM cognitive test and the digit span forward test score gains respectively Meat group associated with significantly greater gains on Raven's Progressive Matrices than other groups.
Grillenberger et al. (2003)[66]	Same as Neumann (2003)[50]	Same as Neumann (2003)[50]	Anthropometry: • Weight, height, head circumference, triceps and subscapular fat folds	Post-intervention at 23 months Anthropometry: • Significant increase in weight in the three intervention groups compared to controls • Decrease in WHZ and stagnated HAZ • Children with a baseline HAZ below the median in the milk group gained more height (1.3 cm) than those in the meat and control groups • Meat group gained more MUAC than the milk and energy groups • Meat group gained 80% more mid-upper arm muscle area and 30% more than the energy group, with a further 40% more gain in mid-upper arm muscle area than the control group • No significant changes were observed in changes in body fat
Murphy et al. (2003)[67]	Same as Neumann (2003)[50]	Same as Neumann (2003)[50,67]	Dietary intake: • Home food intake	Post-intervention at 24 months Dietary nutrient intake: • Significantly higher increase in nutrient intake (energy, protein, riboflavin, vitamin A, iron and zinc) for the meat group compared to the other groups, except for calcium

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Best et al. (2010)[53]	Systematic review	369 papers included in the review, providing data on 76 countries in Latin America, Africa, South-East Asia, Western Pacific and Eastern Mediterranean. All research papers had data on school-age children	Anthropometry: • Weight, height Micronutrient status: • Haemoglobin concentration • Serum or plasma ferritin • Urinary iodine • Serum or plasma retinol • Serum or plasma zinc	Anthropometry: • Stunting, wasting and underweight varied across the regions • Median prevalence of stunting across regions was 18% (7% to 25% IQR) • Higher prevalence of stunting in rural populations in countries in Asia • Median prevalence of underweight was 17% (7% to 34% IQR), with highest prevalence in South-East Asia (39%) and the lowest in Latin America (8%); the prevalence of thinness based on BMI-for-age z-score was about 35% in Africa and South-East Asia, and less than 15% in the other regions Micronutrient status: • Micronutrient status varied across the regions • Median prevalence of iron deficiency was 19% and it was 12% for iron deficiency anaemia • Highest prevalence of iron deficiency was in Africa (29%) • Median prevalence of iodine deficiency was 23%, with high prevalence (> 60%) in Africa, Asia and Eastern Mediterranean countries • High prevalence of vitamin A deficiency (> 50%) among rural and urban poor African school children and among urban school children in India • Studies limited on B vitamins, vitamins C, D, E and K, as well as other minerals and trace elements

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Opara et al. (2012)[68]	Cross-sectional survey	418 children aged 2.9 to 14 years attending schools in Akwa Ibom State, Nigeria	Anthropometry: • Weight, height Stool samples: • Intestinal worm infestation	Anthropometry: • The prevalence of stunting, underweight and wasting was 42.3% vs. 29.7%, 43.2% vs. 29.6% and 10.9% vs. 6.4% for rural and urban pupils respectively Helminthic infections: • Helminthic infections were present in 67.4% of the children • Prevalence of infections was higher in rural than urban children. Hookworm infestation was the most common (55.9%) • Hookworm and *A. lumbricoides* were significantly associated with stunting, wasting and underweight
Amare et al. (2012)[69]	Cross-sectional study	100 elementary school children 10 to 12 years of age, Northwestern Ethiopia	Anthropometry: • Weight, height Micronutrient status: • Serum levels of trace elements, magnesium, zinc, selenium and copper Stool samples: • Intestinal worm infestation	Anthropometry: • 23%, 21% and 11% of the school children were stunted, underweight and wasted respectively Micronutrient status: • 80% had two or more coexisting micronutrient deficiencies • 10% had three coexisting micronutrient deficiencies • 2% had magnesium deficiency • 47% had zinc deficiency • 62% had selenium deficiency • Deficiency of selenium and zinc combined was 34% • Magnesium, copper and molybdenum deficiencies were correlated with height-for-age z-scores Infections: • Prevalence of intestinal worm infestation was 18%

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Baumgartner et al. (2012)[70]	• Randomised, placebo-controlled double-blind 2-by-2 factorial design • Supplementation provided for 8.5 months Supplement: • Iron group – 50 mg iron sulphate • Mixture of docosahexaenoic acid and eicosapentaenoic acid (DHA/EPA) supplemented groups – two oral gelatin-coated fish oil capsules containing 420 mg DHA and 80 mg EPA • Placebo group	321 school children aged 6 to 11 years in low-income rural villages, KwaZulu-Natal, South Africa	Anthropometry: • Weight, height Micronutrient status: • haemoglobin • iron status • zinc protoporphyrin, • serum ferritin • transferrin receptor • C-reactive protein Cognitive assessment: • Kaufman Assessment Battery (KABC –II) • Hand movement test • Triangles test	Baseline findings: Anthropometry: • Overall prevalence of stunting was 6.2%, mild stunting was 31.4% • Prevalence of underweight was 2.2% • Prevalence of overweight measured as BAZ between > 1 SD and < 2 SD was 21.9% • Prevalence of obesity as BAZ $\geq$ 1 SD was 21.5% Micronutrient status: • Anaemia – 20.9% • Iron deficiency (SF < 15.0 mg/L)[3] – 26.5% • Iron deficiency (Tfr < 8.3 mg/L) – 11.5% • Iron deficiency (ZnPP < 70 mmol/mol haem) – 62.9% • Iron deficiency anaemia haemoglobin (< 11.5 g haemoglobin/dL and serum ferritin < 15 mg SF/L) – 30% • Zinc deficiency (< 65 mg SZn/L) – 24.6% • CRP (> 5 mg/l) – 7.1% Results after intervention: • Anaemia prevalence decreased in intervention and control groups, from 20.6% to 1.9% in children who received iron and from 21.1% to 5.0% in children who received the placebo • Growth improved significantly more for children who received DHA/EPA treatment • All children showed a significant improvement in learning abilities • No significant intervention effect and no iron to DHA/EPA interaction on the KABC test scores • Iron supplementation improved recall

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Troesch et al. (2011)[71]	Double-blind placebo-controlled trial of a micronutrient powder containing 2.5 mg iron, 2.5 mg zinc and 60 mg amino acids	192 school children aged 5 to 11 years from Northern Cape, South Africa	Anthropometry: • Weight, height, MUAC, triceps, subscapular Micronutrient status: • Haemoglobin concentration • Iron status (serum ferritin and transferrin receptor) • Serum zinc	Anthropometry • Mean weight-for-age z-score was -1.5 in treatment group and -1.4 in controls at baseline • Height-for-age z-scores were -1.6 in treatment group and -1.4 in controls at baseline • Percentage body fat was 9.6 and 10.3 in treatment and control groups respectively at baseline. Similar at 6 months • Arm muscle area: 17 cm^2 at baseline for treatment group and 17.5 cm^2 for controls; 18.4 cm^2 in treatment group at 6 months and 19.7 cm^2 in controls at 6 months Micronutrient status: Anaemia: • Anaemia prevalence at baseline was 6.3% and 8.2% in treatment and control group respectively • Anaemia prevalence at six months was 5.3 in treatment group and 12.4% in controls • Iron deficiency at baseline was 75% and 74 % in treatment and control groups respectively and decreased to 18.9% and 48.5% in treatment and controls respectively at 6 months • Zinc deficiency was 47.4% in treatment group and 52.5% in controls at baseline, and 30.5% in treatment group and 47.4% in controls at 6 months Micronutrient powder with low doses of highly absorbable iron and zinc reduced iron and zinc deficiency and improved weight-for-age z-scores

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

TABLE 1.1: Summary of selected[a] studies on the nutritional status of school children in sub-Saharan Africa (Continued)

Reference	Study design	Participants and study setting	Outcome measure	Main findings
Friis et al. (2003)[72]	Two by-two factorial design, randomised, placebo-controlled, double-blind trial of daily multi-micronutrient supplementation and multi-helminth chemotherapy on all school days for one year Content of the micronutrient tablet: one RDA for 11- to 14-year-olds of vitamin A, vitamin C, vitamins B_1, B_2, B_6 and B_{12}, folate, niacin, iron, zinc, copper, iodine, selenium Multiple helminthic chemotherapy: albendazole (600 mg) and paraziquantel (40 mg/kg)	746 children in grades 5 and 6, Nyanza Province in Western Kenya	Anthropometry: • Weight, height Micronutrient status: • Haemoglobin concentration Stool samples: • Intestinal worm infestation Malaria infections	Baseline results Anthropometry: • Mean WAZ and HAZ were -1.11 and -0.96 respectively • Prevalence of stunting, wasting and underweight not reported Haemoglobin concentration: • Mean haemoglobin concentration was 123.7 g/l • 41.1% were anaemic • No significant differences in mean Hb between boys and girls, but prevalence of anaemia was higher in boys than girls Infections: • The prevalence of malaria parasitemia was 59% Helminthic infections: • 14% *A. lumbricoides*, 45% *T. richiura*, 55% hookworm and 71% S. *mansoni* Eight months' follow-up • Micronutrients and anthelminthics increased Hb by 3.5 g/l (95% CI; 1.7, 5.3) and 2.0 g/l (95% CI; 0.2, 3.9) respectively, irrespective of initial nutritional status and Hb status • No significant differences in the effects of the two interventions between anaemic and non-anaemic children • Stronger effect of micronutrient supplementation on Hb among the boys compared to the girls • No interactions between the interventions and HAZ and WAZ

[a] These studies were selected for review based on their availability, covering study population of school children in sub-Saharan African countries, and having been published in the year 2000 or later.

1.2.2   Consequences of undernutrition among school-age children

It is well recognised that suffering from undernutrition during the school years can constrain a child's physical and mental growth.[53] Childhood undernutrition diminishes adult intellectual ability and work capability, which result in economic difficulties for individuals and their families.[73] School-age children who suffered from early childhood malnutrition have been found to have poorer IQ levels and school achievement, and more behavioural problems, than matched controls and, to a lesser extent, siblings.[74] Undernutrition increases the risk for morbidity and mortality among young children.[74,75] In addition, undernutrition in school children results in reduced capability to study, lower test scores, slow or lack of improvement in school grades, as well as increased school absence, lateness, and school delay.[76] Stunted rural Guatemalan children had lower literacy scores, completed fewer years of school and scored lower in tests of intellectual ability than their peers who had grown normally.[77] The physical and mental developmental retardation associated with undernutrition may result in non-enrolment or late enrolment in school.[78]

Stunting, which is an indicator of chronic undernutrition, leads to reduced adult body size, causing deficits in productivity in adulthood[77] and thus perpetuating the intergenerational cycle of poverty.[79] Thinness in school-aged children can result in delayed maturation, insufficiency of muscular strength and work capacity, as well as reduced bone density later in life.[75] Shorter women with smaller pelvic sizes have a higher risk for obstetric complications and are exposed to the possibility of delivering low-birth weight infants.[76,80] In addition, smaller mothers are likely to have smaller babies, the resulting intergenerational cycle of undernutrition perpetuates a generational transmission of undernutrition and poverty. If this child is a girl, she is likely to suffer undernutrition into her child-bearing years, thus risking becoming another under-nourished mother and extending the intergenerational cycle of undernutrition.[81] The cycle of undernutrition also perpetuates the cycle of poverty, because an undernourished child has limited potential for learning and skills development at school, and thus does not achieve his or her full growth and development potential.[82] This phenomenon is common in many African households, and symbolises the close link between undernutrition and unrelenting poverty.[83] Furthermore, low birth weight is associated with a risk of developing type 2 diabetes during the school-age years[84] and later in life,[85,86] as well as the increased risk of developing cardiovascular diseases in the future.[86]

The correlation between underweight in children and increased risk of morbidity in children under the age of five years has been described in various studies. For example, in the Sudan the risk of having diarrhoea was two times higher (95% confidence interval, CI (1.64, 2.43) in children with severe wasting and 1.75 times higher (95% CI (1.56, 1.96) in those with severe underweight

compared with children with a weight-for-age and weight for height of > 1 SD.[87] In a similar study in Papua New Guinea it was found that there was a significant association between growth faltering in children younger than five years and acute lower respiratory infections.[88] In the Kenyan study, stunting among toddlers was associated with a higher incidence of acute lower respiratory tract infection.[89] Undernourished children have more severe diarrhoeal episodes, characterised by longer duration of illness, hospital admission, a higher possibility of dehydration and associated growth failure.[90] One study among Columbian school children found a relationship between stunting and an increased incidence of cough with fever during the school year.[79]

Studies have reported lowered work capacity in undernourished school children and adolescents. In Guatemala, a study by Haas et al.,[91] reported significantly lower work capacity measured as litres of oxygen consumed (VO_2 max in L/minute) among children with severe stunting, marked by height-for-age z-scores of less than -4 SD according to the WHO reference, compared to children who had higher height-for-age z-scores. In Columbia, marginally underweight adolescent boys showed reduced oxygen uptakes at maximum exertion, measured as litres of oxygen consumed (VO_2 max), when compared with boys of the same age but of normal weight.[92] A prospective study of 17-year-old Indian boys who had been followed up from five years of age reported a significant gradient of reduced work capacity with severity of stunting at a heart rate of 170 beats per minute (bpm). Boys whose height-for-age z-score was greater than -2 SD at five years had a work capacity of 632 kpm/min physical work capacity at 17 years compared to those with severe stunting less than -4 SD who had a work capacity of 357 kpm/min physical work capacity at 13 years.[93]

School-age children who suffer from severe stunting show significantly compromised reasoning and perceptual-spatial functioning, poor school grades, reduced attention and unresponsive play behaviour in comparison to their well-nourished peers.[74] Children with severe undernutrition are more listless, less lively and tend to explore their environment less, both in extent and complexity, than children who are ill with other diseases.[74] In addition, children suffering from mild to moderate undernutrition show significant deficits in intellectual and behavioural function,[94] such as slowed development in speech.[95] Stunting has a negative correlation with cognitive test scores. A follow-up study in Peru reported decreased scores on the Wechsler Intelligence Scale for Children – Revised (WISC-R) among children with persistent stunting.[96] Among rural Jamaican school children, a high height-for-age z-score was positively associated with performance in arithmetic.[97]

Stunting is associated with other long-term effects, such as metabolic alterations, which are risk factors for non-communicable diseases, including hypertension and other obesity-related

disorders.[98,99] Studies in Brazil have shown a strong connection between stunting and hypertension among adults, particularly women,[100,101] and a relationship between stunting and obesity, especially resulting from abdominal fat.[98] This association was demonstrated further in a Brazilian study involving stunted school children living in a low-income rural community, who were found to be overweight even with reported low energy intake.[102] Longitudinal studies among adolescent girls in Senegal demonstrated that stunting during early infancy (6 to 18 months of age) was a risk factor for increased accumulation of body fat, especially on the upper body – on the trunk and arms – during the adolescent years.[103] Studies utilising dual-energy X-ray absorptiometry (DXA) to analyse the changes in body composition in chronically undernourished children reported that boys had a preference to build up body fat to the disadvantage of lean body mass.[98,104] Furthermore, a study among adults of short stature found an increase in the waist-to-hip ratio of 65% in men and 29% in women for each decreased z-score of stature.[105] In addition, stunting has been associated with the risk of obesity and abdominal fatness in women (odds ratio = 1.77; 95% CI; 1.10–2.83) in a study involving a representative sample of 2 040 households in Rio de Janerio, Brazil.[106]

The question whether older children and adolescents can catch up incomplete childhood growth caused by undernutrition is debatable. Few authors have explored this question, in contrast to studies on the reversibility of undernutrition, especially stunting, in early childhood, which has been explored to a greater extent. For instance, Martorell et al.[107] reviewed the epidemiological findings on the reversibility of stunting in developing countries. The review concluded that catch-up growth can occur as a result of the prolonged pre-pubertal growth period and accelerated growth rates resulting from a change in environment or food supplementation for children younger than three years of age.[107] This argument is further demonstrated in a study involving Indian children 7 to 14 years old who were adopted by people living in Sweden. This study reported catch-up growth in height and weight for children with stunting within two years of arrival in Sweden from India.[108] The potential for catch-up growth has been demonstrated even in children who remain in the same environments. A Peruvian study reported catch-up growth in 36.8% of children stunted in infancy, showing catch-up growth occurring at four to five years.[109] Further, children in the Philippines who were stunted at age two exhibited catch-up growth at 8.5 years, while others showed catch-up growth by 12 years, without any form of intervention.[110] Predictors of recovery from growth faltering have been identified as maternal education, which influences child feeding and care practices,[111] while a higher initial height-for-age z-score assessment increases the likelihood of catch-up growth.[109]

1.2.3   Overnutrition among school-age children

There is increasing evidence for the occurrence of overweight and obesity in developing countries and countries in transition. The occurrence of overweight and obesity, referred to as overnutrition in this review, along with undernutrition is what signifies the emergence of the dual burden of malnutrition. This trend has been reported mainly in middle-income countries such as Russia and Indonesia, where between 9% and 13% of households have both underweight and overweight members.[112] Rapid nutritional transition in developing countries has seen the coexistence of child undernutrition and maternal overweight and obesity in the same households.[113,114] In China, for example, the Chinese Health and Nutrition Survey in 2002 found 8.1% of the households to have underweight and overweight members.[115] In Benin, West Africa, a study identified the dual burden of malnutrition in 16.2% of the 148 households surveyed, with maternal overweight or obesity coexisting with childhood undernutrition.[113] In the Benin study, maternal BMI was associated with the children's weight-for-height z-score, with significantly more wasted children among the mothers with low maternal BMI. An analysis of the 2008/09 Kenya Demographic and Health Survey data, covering children aged 6 to 59 months, reported the coexistence of maternal overweight or obesity in 16.6% of households.[116] Given the challenge of the double burden of malnutrition facing households in developing countries, the planning of nutrition programmes, including school feeding programmes, should be tailored to address both under- and overnutrition.

Overweight and obesity, coupled with excessive energy intake and inactive lifestyles, are a rising health challenge in many countries.[117,118] The reality of obesity in children all over the world has become a pandemic, with the growing rates of overweight children now dominating public health issues in middle- and low-income nations.[119] It was estimated in 2010 that 43 million pre-school-age children were overweight and obese, with the majority (35 million) living in developing countries.[12] There is an increasing trend of childhood overweight and obesity worldwide, with the prevalence of these conditions growing from 4.2% in 1990 to 6.7% in 2010.[12] This trend is likely to increase and is expected to reach 9.1%, or about 60 million children, by 2020.[12]

The overweight and obesity epidemic in children and teenagers has been documented in many developing countries. This phenomenon is complicating efforts to address the health and nutrition challenges of school-age children.[120] A further data analysis of the 1999 National Food Consumption Survey in South Africa, undertaken in 2007, reported the prevalence of overweight and obesity in children 12 to 108 months old at 17%, which was nearly as high as that of stunting, at 19%. In India, the prevalence of and trends in overweight and obesity showed a slight increase, from 10.6% in 1998 to 1999, to 12.6% in 2005 to 2006.[121] This same study reported a much higher

increase in the prevalence of overweight and obesity among urban women of higher socio-economic status.[121]

Studies reporting overnutrition among school-age children have reported similar trends as seen in younger children, especially in developed countries and to some extent in the developing world. One in every six school-age children is overweight, and one in four overweight children is obese.[122] The incidence of obesity has increased dramatically among school children around the world over the last several decades.[123] The National Health and Nutrition Examination Survey (NHANES) reports an increase in the prevalence of obesity among preschool children in the USA of 5% to 10.4% from 1976/1980 to 2007/2008, while the increase among children aged 6 to 11 years was from 6.5% to 19.6%.[124] In Latin America, the prevalence of obesity among school children has increased considerably.[125] This growing concern is similar to that among children from various European countries, especially in Eastern and Southern Europe.[126] Another example is the increasing prevalence of overweight and obesity observed in children between the ages of 6 and 9 years in Vietnam, from 0.5% in 1992 to 1.9% in 2002.[120]

In Bolivia, the prevalence of overweight was reported at 8.5% among children aged 5 to 10 years, with the prevalence of obesity at 0.5%. Trends in overnutrition among school children in Chile show that, from 1986 to 1998 there was a significant increase in obesity risk, from 8.3% to 19.6% in pre-pubertal females, and 5.4% to 14.6% in pubertal males.[127,128] This trend has been demonstrated further in Pakistan, where data from the 1994 Pakistan National Health Survey for children aged 5 to 14 years from urban areas was compared with data collected in 2004 among children of the same age in the Karachi area.[118] This study showed a significant increase in overweight and obesity, from 3% in 1994 to 5% in 2004 ($P < 0.001$). A cohort study in Brazil in 2010 reported overweight at 5.5% and obesity at 16.6% among children aged 6 to 11 years.[129] Among Indian pre-adolescent and adolescent children aged 9 to 15 years, the prevalence of obesity and overweight has been reported as 11.1% and 14.2% respectively.[130] In South Africa, the prevalence of overweight and obesity in primary school children aged 6 to 13 years was reported to be lower among boys (3.2% and 14%) than girls (4.9% and 17.9%).[131]

Despite the rapid increase in the prevalence of overweight and obesity among children globally, a search of the literature revealed no studies in Kenya focusing on overnutrition among school-age children. Among pre-school-age children there is growing concern of overweight and obesity in the country, with the prevalence of overweight estimated at 18%, while obesity was estimated at 3.4%

among children 36 to 59 months old.[132] This trend may be the same among school-age children, especially in urban areas.

Suffering from overnutrition during the school-age years has negative impacts on the development and health of children and can lead to unfavourable effects on the physical, mental and social aspects of child health.[133] These effects include increased risk of orthopaedic disorders,[134] high blood pressure, metabolic syndrome, non-insulin-dependent diabetes, elevated cholesterol and atherosclerosis, which are all precursors of heart disease.[135] In addition to the increased risk of developing lifestyle and diet-related non-communicable diseases, overweight and obesity during childhood can have a negative impact on psychosocial development and academic performance,[136] as well as increase the risk for morbidity and mortality later in life.[137]

Subclinical atherosclerosis in young adults is predicted largely by cardiovascular disease and diabetes risk factors acquired in childhood, such as dyslipidemia.[138] Obesity during the school-age years increases the likelihood of developing obesity in adulthood.[139] A study in the Wausau metropolitan area in the USA reported lipid abnormalities in school children enrolled in grades 2, 5, 8 and 11 (n = 586).[138] Lipid abnormalities were defined according to the American Heart Association definitions as total cholesterol ≥ 200 mg/dl, non-high density lipoprotein (HDL) cholesterol ≥ 160 mg/dl, low density lipoproteins (LDL) cholesterol ≥ 130 mg/dl, high density lipoprotein (HDL) cholesterol ≤ 35 mg/dl, triglycerides ≥ 150 mg/dl, and pattern B phenotype (average LDL particle size) < 20.5 nm.[140] The children in the Wausau school district had abnormal values for total cholesterol (18%), LDL cholesterol (27%), non-HDL cholesterol (12%), HDL cholesterol (10%), triglycerides (8%) and pattern B phenotype (5%). Dyslipidaemia, defined as the existence of at least one of these lipid abnormalities, was present in 39% of the sample, while 22% had two or more lipid disturbances.[138]

1.2.4   Body composition in school-age children

Body composition plays a central role in the health status of children and adolescents.[141] Body composition refers to the characteristic size and distribution of the component parts of total body weight.[142] Studies of body composition among school-age children from developed countries show a general trend of higher fat deposition in comparison to fat-free muscle mass. A study among American-Indian school-age children in the USA reported a high prevalence of obesity, based on BMI-for-age, with 28.6% of the children having a BMI-for-age above the 95th percentile of the NHANES reference data.[143] The same study also reported a high subcutaneous fat deposition, with 45.8% of the children having a percentage of body fat higher than the 95th percentile of the

reference population, while the triceps skinfold thickness, an index of body fatness, was notably higher in the American Indian school children than the NHANES reference data. A strong association has been reported between BMI and fat mass index among children in the USA with a BMI higher than the 50th percentile.[144] A study in Bahrain among children aged 6 to 18 years reported a higher than normal fat content compared to fat-free mass, indicating a substantial deposition of subcutaneous fat, which was attributed to a lack of physical exercise and high fat intake among the children.[145] In Japan, BMI was positively correlated with percentage body fat and waist circumference in Japanese school children.[146]

High BMI and skinfold measurements in school children in the developed world are more likely indicators of over nourishment. On the other hand, a high BMI among rural school children in developing countries is more likely to be an indicator of higher muscle mass, while low fatness can be seen as a result of undernutrition.[92] A study by Monyeki et al.[147] among rural South African school children found that BMI was highly correlated with fat-free mass, and that the relationship between physical fitness and BMI was the same as the relationship between physical fitness and fat-free mass.[147] In India, school-age children were reported to have a lower mean percentage body fat compared to mean fat-free mass. This review found only one study done in Kenya (by Neumann and colleagues[50]) that reported on body composition in school children. In this study, done in rural Kenya, school children were reported to be generally lean, with mean triceps, subscapular skinfold thickness and average arm fat area within or below the fifth percentile found for African-American children by NHANES, while the average arm muscle area was in the 10th percentile.[50] There is a need for more studies in the country on body composition in this age group, as it is important to understand the patterns of body composition, which provide a key indicator of health in relation to lifestyle diseases among children and adolescents.

1.2.5   Micronutrient deficiencies among school-age children

Widespread micronutrient malnutrition is well documented.[148] Micronutrient deficiencies are a leading determinant of morbidity and impaired quality of life worldwide, especially among children and populations in resource-poor countries.[149] Micronutrient deficiencies are a major concern for children globally,[150] and negatively impacts on growth, an increased risk for morbidity, as well as impaired mental development and learning capability in school children.[31] Among school-age children, the most commonly reported deficiencies include vitamin A deficiency (VAD), iron deficiency and anaemia (IDA) and iodine deficiency disorders (IDD). Deficiencies of zinc, vitamin B_{12}, folate and calcium are also of concern, although to a lesser extent in this age group.[151] This is especially so in low-income countries, where deficiencies of iron, zinc and vitamin A are the most

important public health problems among children.[152] It is of importance to ensure adequate micronutrient status among school-age children, as micronutrients have important functions in growth and development. Specifically, iron plays important roles in the production of red blood cells, as part of haemoglobin for oxygen transportation throughout the body, as a transportation medium for electrons within cells, as well as forming an integral part of the enzyme systems in various tissues. In addition, iron is essential for the proper functioning of the immune system, as it is required for the proliferation and maturation of cells.[153]

1.2.5.1   *Iron deficiency and anaemia*

Iron deficiency is defined as decreased total iron body stores and is characterised by a low concentration of serum iron, low serum ferritin, transferrin saturation, low haemoglobin concentration and increased concentration of transferrin receptors (sTfR).[154] An adequate supply of dietary iron is necessary to maintain body iron stores and to ensure that there is adequate iron for normal body functioning and haemoglobin synthesis. Iron deficiency is prevalent in infants, children and adolescents worldwide due to their high iron requirements during growth, low dietary iron intake and low-bioavailability diet.[155] Iron deficiency is the most significant contributor to the onset of anaemia, with a general assumption that 50% of the cases of anaemia are due to iron deficiency.[156]

Anaemia is the most common and widespread disorder worldwide and it remains a global public health problem.[156] In sub-Saharan Africa, the prevalence of anaemia is estimated at around 67.6% among pre-school-age children, 57.1 % among pregnant women and 47.5% in non-pregnant women.[156] A review on the nutrition of school children by Best reported an average IDA prevalence of 32% in South East Asia, 25% in Latin America, 24% in the Eastern Mediterranean and 31% in the Western Pacific.[53] In Africa, the prevalence of IDA is 29% according to the review by Best.[53] A study among rural Vietnamese school children on the prevalence of IDA anaemia reported a prevalence of 25.1%.[152] In India, a study on the prevalence of iron deficiency and anaemia reported a prevalence of 69% among school children, 7% of whom had severe anaemia and 41% who were moderately anaemic.[157] Various studies from across Africa have reported the presence of IDA among school children. A large study in eight countries in Africa and Asia, undertaken in 2000, reported anaemia as a public health problem in children aged between 7 and 11 years in five African countries (Ghana, Malawi, Mali, Mozambique and Tanzania), and in children aged between 12 and 14 years in four African countries (Malawi, Mali, Mozambique and Tanzania).[158]

In Nigeria, a very high prevalence of anaemia, at 82.6%, has been reported, along with 77.5% depleted body iron stores.[159] Other studies show the prevalence of anaemia in Tanzania at 77%, with 47.2% in Bukinafaso[160] and 41% in Ghana.[161] In rural Kenitra, Morocco, the prevalence of anaemia was reported as 12.2%, iron deficiency was 20.4% and iron deficiency anaemia was 7.7%.[162] In Zanzibar, a high prevalence of anaemia (63%) was reported in a large sample of school children (n = 3 605), with 41% having depleted body iron stores.[163]

The situation in Kenya is similar to that reported in other African countries. In Western Kenya, a study among school-age children reported depleted body iron stores at 14.8%.[164] According to the same study, the prevalence of anaemia was 30% among 7 to 11-year-olds and 40% among 12- to 14-year-olds, with a higher prevalence among boys than girls. Studies among school children from Nyanza Province have reported different levels of anaemia, iron deficiency and IDA. In Bondo District, Friis and others reported a higher prevalence of anaemia among girls (40.3%) aged 9 to 18 years.[72] Koukounari et al.,[165] working in the same district, reported a prevalence of anaemia of 13% among school-age children. Nyanza Province of Kenya is classified as a high malaria transmission area, with intense transmission during the rainy seasons in March to May and November to December.[166] Children living in high malaria transmission areas tend to acquire immunity against malaria in early childhood, putting them at a reduced risk of clinical attacks and malaria-related mortality.[167] However, many school-age children have asymptomatic malaria parasitaemia, which can cause anaemia.[168] The persisting problems of iron deficiency and anaemia in Kenya expose children to the consequences of the deficiency in relation to their health, growth, development and school performance.

Iron deficiency is one of the ten leading determinants of the global burden of disease, particularly in Asia and Africa.[21] The effects of iron deficiency include a reduction in the haemoglobin content of red blood corpuscles, resulting in a reduction in oxygen-carrying capacity.[169] Iron deficiency also causes impaired immune function and thus increases susceptibility to infections[169] and longer duration of infections in anaemic children in comparison to healthy children.[170] The role of iron in proper cell differentiation, cell growth and enzymatic functioning of immune cells explains the increase in severity and duration of infections in anaemic children.[171] Iron deficiency in children leads to slow physical development, impaired and poor growth, increased fatigue, poor cognitive performance and delayed psychomotor development.[172] A shortage or lack of iron early in life is thought to be a potential inhibitor of neurotransmitters, compromising brain function.[157] In addition, children with iron deficiency have an increased risk of chronic lead poisoning.[173]

The consequences of iron deficiency and anaemia in adults include reduced physical activity, fatigue and reduced work performance,[26] as well as reduced aerobic work capability.[174] Iron deficiency affects physical activity in several ways, which include impairing work performance, reducing the functioning of such as skeletal muscle,[175] and affecting oxygen consumption. Iron deficiency impairs the capacity of skeletal muscle, even in mild anaemia, which is known to reduce performance during brief but intense exercise.[176] The physical productivity loss caused by iron deficiency per year was quantified at about USD 0.32 per head, or 0.57% of the gross domestic product, in ten countries.[177] It is estimated that if iron fortification reached 50% of the population in Africa, it would avert 570 000 disability-adjusted life years (DALYs) every year.[178]

1.2.5.2   Vitamin A deficiency

Vitamin A plays a critical role in eye health, in the integrity of the epithelial cells,[179] the maintenance of the immune system and reproductive system, as well as in bone health.[180] The transduction of light into the neural signals necessary for vision requires retinal in the form of 11-*cis*-retinaldehyde.[181] Normal differentiation of the cornea and conjunctival membranes, which helps prevent the occurrence of xerophthalmia, requires the presence of the retinoic acid form of vitamin A.[182] Vitamin A is also important in maintaining the integrity of epithelial cells in the body.[183] Furthermore, the gene expression process that determines structural proteins such as skin keratins, enzymes such as alcohol dehydrogenase, extracellular matrix proteins and retinol-binding proteins is regulated through the activities of retinoic acid receptor (RAR) and retinoid X receptors (RXR), which are activated by retinoic acid in the nucleus.[184]

Retinoids determine the proper functioning of the immune system, which depends on cell differentiation and proliferation in response to immune stimuli. Retinoic acid is also important in maintaining an adequate level of circulating natural killer cells that have antiviral and anti-tumour activity.[185] Retinoic acid also plays an important role in embryonic development. Retinoic acid, as well as RAR, RXR, cellular retinol-binding protein (CRBP) and cellular retinoic acid-binding proteins (CRABP-I and CRABP-II), is present in temporally specific patterns in the embryonic regions known to be involved in the development of structures posterior to the hindbrain, such as the vertebrae and the spinal cord.[186] Retinoic acid is also involved in the development of the limbs, heart, eyes and ears.[187] Further, an association between vitamin A deficiency and pathogenesis of anaemia has been described.[188] The mechanisms of this association involve the role of vitamin A in the enhancement of growth and the differentiation of erythrocyte progenitor cells, iron mobilisation from tissues, and reducing the anaemia associated with infections.[188]

Vitamin A deficiency (VAD) is common in developing countries, especially in young children and pregnant women. It causes altered immune function and increases the risk of infections. The World Health Organisation (WHO) provides global estimates of VAD based on night blindness and serum retinol data of preschool children and pregnant women. Night blindness affects 0.9% of preschool-age children, or 5.2 million, and 7.8% of pregnant women, or 9.8 million, at risk of VAD globally. Low serum retinol concentrations (< 0.70 µmol/L) affect 33.3% of pre-school-age children, or 190 million, and 15.3% of pregnant women, or 19.1 million, in the population at risk of VAD globally.[156]

School-age children have not been considered an 'at risk' group for VAD in the past. Data from large studies on the occurrence and the effects of VAD among school-age children is limited and global estimates are not available for this age group. However, there is a growing volume of literature in specific countries investigating the prevalence on VAD in school children, which suggests that VAD is a growing public health problem in this population group.[189] In Bangladesh, for example, subclinical VAD remains a significant public health problem among school-age and adolescent children, especially among girls.[190] It is estimated that VAD may be a larger problem among school-age children than in pre-school in Bangladesh, since the country achieves vitamin A supplementation coverage of over 90% in under-fives.[31] Various studies have reported the presence of low serum retinol concentrations among school children. For example, in rural Malaysia, 27.4% of school children were reported to have low serum retinol levels (< 0.70 µmol/L).[191] In India, a study among school-age children reported a 57.1% prevalence of VAD based on low serum retinol.[192] A survey on the health and nutritional status of public school children in Bogota, Colombia reported a 13.7% prevalence of VAD, with a severe prevalence of 1.3%.[193]

The prevalence of vitamin A deficiency in sub-Saharan Africa has been described primarily among infants and pre-school children. Vitamin A deficiency is estimated to be 19% among children younger than five years of age globally, 18.1% in the West and Central African Region (WCAR), and 20.1% in the East and Southern African Region (ESAR).[194] A national survey in Ethiopia among children aged 6 to 71 months reported a national prevalence of low serum retinol status of 37.7%.[195] In two rural schools in Burkina Faso[160], VAD prevalence was 42.7%. A study in rural Botswana[196] found a 53.4% prevalence of low serum retinol concentration among school children while a prevalence of 71% was reported among children aged 5 to 10 years in a region with a low socioeconomic status in the KwaZulu-Natal province of South Africa.[70] In Tanzania, one study reported a prevalence of 21% of low serum retinol levels among school-age children.[197]

There are few studies of the vitamin A status of school children in Kenya (Table 1.1). Neumann et al.[50] reported a high prevalence (68.6%) of moderate vitamin A deficiency based on serum plasma levels between 0.35 and 0.7 μmol/l, and a 22% prevalence of severe vitamin A deficiency (< 0.35 μmol/L) among school children in a rural area of the Eastern Province.[50] The status of vitamin A deficiency among school children in the country still needs to be established. The country's coverage of vitamin A supplementation among children under five is still very low, at 30.3%.[18] There is no established policy on vitamin A supplementation for school-age children, thus deficiencies are likely to be high in this population group.

Night blindness is an early sign of vitamin A deficiency and is caused by an insufficient amount of visual purple in the retina. Night blindness leads to xerophthalmia (keratinisation of the conjunctiva), which subsequently spreads to the cornea, causing ulceration. Persistent vitamin A deficiency results in keratomalacia, which, if not treated, leads to permanent blindness. Xerophthalmia is known to be the leading cause of childhood blindness and is responsible for 350,000 cases of blindness in children worldwide annually.[198] The prevalence of severe corneal xerophthalmia is usually highest at two to three years of age, but the prevalence of mild xerophthalmia increases with age beyond five years, probably because VAD builds up over time due to a chronic shortage of the vitamin.[199]

Besides causing xerophthalmia, VAD can limit growth, weaken immunity, and increase mortality.[182] Sub-clinical VAD is associated with increased susceptibility to respiratory tract infections,[200] and the severity and case fatality of measles and diarrhoea.[201] Globally, 627,000 deaths and 21,569,000 disability-adjusted life years are attributable to vitamin A deficiency annually.[202]

1.2.5.3 Iodine deficiency

Iodine deficiency (ID) is still a health problem in regions with inadequate iodine, despite national and international efforts to increase iodine intake through salt iodisation. An estimated 2.2 billion people (about one third of the world population) who live in areas with iodine deficiency are at risk of complications arising from the deficiency.[203] The consequences of iodine deficiency are seen throughout the lifecycle, from pregnancy through adulthood. The most vulnerable are pregnant women, foetuses, neonates, children and adolescents.[204] School-age children are exposed to the risk of ID. One third of the world's school-age children have inadequate iodine status, with urinary iodine (UI) levels below 100 μg/l.[205] According to WHO estimates, 42.3% of African school-age

children have insufficient iodine intake (UI < 100 ug/l). Iodine deficiency is persistent in the Zanzibar islands of Tanzania among school children, with a total goitre rate of 21.3% in Ugunja and 32% in Pemba.[206] The coverage of iodised salt remains low in some sub-Saharan African countries, varying from 1.0% on the Pemba island of Zanzibar to less than 10% in the Gambia and 63.5% on the Unguja island of Zanzibar. Other countries have high coverage, such as 90% in Burundi, Kenya, Nigeria, Tunisia, Uganda and Zimbabwe. A national survey of urinary iodine levels among school-age children in Kenya in 1994 reported optimum[207] levels of iodine of 100 to 199 ug/l urinary iodine (UI).[208] Based on this median IU of iodine intake in school-age children, Kenya was classified as having optimum levels of iodine intake. Similarly, Neumann et al.[50] reported urinary iodine to be in the normal range, with about 10% of school children in the low iodine intake range.

1.2.6   Summary of literature on nutritional status of school children in Kenya

This review points to an inadequate energy intake among school children in Kenya with an adequate intake of protein while the micronutrient intakes are inadequate. There is also a lack of standardized methodologies in assessing the adequacy of nutrient intake in the country and this is further demonstrated by the fact that there is no national dietary intake tool such as a food frequency questionnaire that has been standardized for use in any population group in the country. There is a lack of data on national estimates for under- and over-nutrition, body composition status as well as micronutrient status among school children in the country. Regional estimates show evidence of compromised nutritional status as shown by the prevalence of stunting, underweight and wasting. This literature review further shows evidence of micronutrient deficiencies in this population as demonstrated by depleted body iron stores, presence of iron deficiency and anaemia as well as vitamin A deficiency.

1.3   Causes of Nutritional Disorders among School-age Children

Stunting is widely believed to occur through a cumulative process, mainly in early childhood. Stunting seen during the school-age years is a reflection of earlier exposure to poor nutrition from early childhood, which tends to increase through the school-age years.[83] It has been suggested that children could experience catch-up growth if their environment improves.[209] Interventions in school-age children that complement efforts in the preschool years can reduce levels of stunting and are linked to consequences for children's health and education. Limited dietary diversity, persistent household food insecurity and repeated infections are factors contributing to the deficiency of macro- and micronutrients among Kenyan school-age children. Linear growth retardation and impaired child development is caused by inadequate dietary intakes of energy, protein and other nutrients, coupled with high infection rates and living in generally impoverished environments.[35]

On the other hand, causes of underweight among school-age children include a broad range of nutrition challenges, such as prenatal undernutrition, deficiencies of macro- and micronutrients, and morbidity.[31] The physical growth of school-age children in poor resource settings is influenced by poor food consumption patterns, infections, inadequate sanitation, inadequate access to health care and improper hygiene practices.[33,35]

The nutritional transition observed around the world, characterised by increased overnutrition, is as a result of lifestyle factors. Examples of lifestyle changes that are contributing to the increasing occurrence of overnutrition is the replacement of traditional, nutrient-dense diets rich in whole grains, fibre, fruits and vegetables with diets that have higher contents of saturated fat and refined carbohydrates.[210] In addition to the change in diet, reduced physical activity contributes to the challenge of nutritional transition. Reduced physical activity is a result of increased hours of television watching per day rather than engaging in games that involve physical activity. For example, Mexican city children aged 9 to 16 years reported watching more than four hours per day of television, compared to the two hours per day spent on vigorous physical activity.[211] Irregular mealtimes, watching television for more than two hours per day and a night-time sleep duration of less than four hours were significantly associated with overweight and obesity among children aged 3 to 6 years in Japan.[212]

In a study in Bogota, Colombia, overweight or obesity among school children was positively associated with high maternal BMI, higher socioeconomic status and a snacking dietary pattern.[134] Multiple micronutrient deficiencies often occur simultaneously as a result of a poor-quality diet.[213] In developing countries, low dietary intake of animal-source foods (ASF),[213] which are important sources of iron, zinc, vitamins A and B_{12} and protein, can result in multiple micronutrient deficiencies.[214] Families with a low socioeconomic status often cannot afford ASF such as meats, fish, poultry and dairy products, or fruit and vegetables.[215] The diets of children in poor populations are characterised by limited dietary diversity, as their diets consist predominantly of starchy staples that include few or no ASF and seasonal fruit and vegetables.[216] Furthermore, school-age children may develop a more independent eating pattern[217] that often includes out-of-home food consumption without supervision, which is likely to result in the increased intake of foods of low nutritional value, such as soft drinks, candy, and salty and fatty snacks in place of micronutrient-rich foods.[218] A study in the Embu District of Eastern Province in Kenya found that foods eaten out of home by school children during the dry season, when there is less food at home, provides 13% of the daily energy intake, while it provides 19% during rainy seasons. The foods that are consumed

most out of home include hard candies and sugarcane.[65] Poor socioeconomic status, which is common in rural households and poor urban households in Kenya and other developing countries, contributes to low availability of these rich nutrient sources and aggravates the problem of micronutrient deficiencies in household members.

There are multiple causes of iron deficiency anaemia, including inadequate dietary iron intake,[173] respiratory infections, helminth infestation, malaria, diarrhoea, and vitamin A and C deficiencies.[169] Meat iron sources contain 30 to 70% of iron in the haem form, of which 15 to 35% is absorbed.[219] Haem iron usually constitutes only 5 to 15% of the dietary intake of iron.[220] The absorption of non-haem iron found in plant sources is less than 10% and forms the bulk of the ingested iron, especially in developing countries.[221] A study in Tanzania reported that school children's diets depended on cereal and vegetable-based foods, while the intake of red meat and fruit was less frequent.[222] Factors that enhance the absorption of non-haem iron include dietary intake of haem sources such as meat, and the intake of ascorbic and other organic acids, while inhibitors of absorption of non-haem iron include phytates and tannins found in plant-based foods,[223] as well as calcium[173], which reduces the absorption of iron through intraluminal interactions.[224] Calcium supplements were reported to inhibit the absorption of iron by adult women in a study to evaluate the extent to which calcium inhibits the absorption of iron.[225]

The usual diets in rural Kenya are predominantly plant based, with only 4% of energy coming from ASF.[67] The Kenyan diet, especially in rural areas, is based predominantly on starchy staples, with few or no products of animal source. Anaemia is also caused by deficiencies of other hematopoietic nutrients, such as folate, which is the second main cause of anaemia after iron deficiency and causes megaloblastic anaemia.[226] Folate deficiency weakens cell division and protein synthesis, resulting in large, hypochromic and immature red blood cells (RBCs).[227] Parasitic infections or diarrhoea can also lead to multiple micronutrient deficiencies due to the limited absorption or utilisation of nutrients.[228] The association between hookworm infection and iron deficiency anaemia among children is well established and is known to be a major risk factor for morbidity in most developing countries.[229] An estimated 1.2 billion people residing in tropical developing countries are infected with hookworm.[230] The mechanism by which infections with hookworm cause anaemia is through intestinal blood loss.[231] Adult hookworms attach to the mucosa in the upper small intestine, where they ingest tissue and blood[232] and cause blood loss of the host, as well as other losses that occur from the bleeding of the damaged mucosa. The risk of anaemia increases with an increase in intensity of hookworm infection,[229] as worm load and faecal egg count are strongly correlated with

the amount of blood loss.[233] Schistosomiasis and hookworm were reported to significantly influence the haemoglobin status of school children in a Tanzanian study.[222]

Malaria infection, which is common in sub-Saharan Africa and in Kenya, is a known risk factor for anaemia. According to a meta-analysis of malaria intervention trials among African children, both symptomatic and asymptomatic malaria contribute to anaemia.[234] Erythropoiesis and haemolysis lead to a decrease in haemoglobin (Hb) concentration, thus causing malarial anaemia.[235]

Habitual inadequate dietary intake of vitamin A is the main cause of vitamin A deficiency. This is particularly so in populations with low intakes of animal-source carotenoid foods that are converted to vitamin A. Inadequate intakes of fat, which facilitates the absorption of carotenoids, also perpetuates vitamin A deficiency.[73] In the developing world, where diets are mainly plant based, the main form of vitamin A usually consumed is the form of β-carotene.[236] The availability of these β-carotene-rich foods is seasonal and their consumption is irregular in resource-constrained, food-insecure environments. An association was found between low serum retinol concentration and children from poor households in a vitamin A status study among Colombian school children.[193] This association has also been reported among pre-school-age children and women of childbearing age in developing countries.[237] These associations suggest that poorer people may have less access to vitamin A-rich foods, since they often consume a carbohydrate-based diet and cannot afford foods from animal sources, fresh fruit and vegetables, which would provide a sufficient supply of vitamin A.[193] Maternal educational attainment is negatively correlated with the risk of VAD in children and adolescents.[238] Boys in Indonesia were found to be at a higher risk of low serum retinol concentration if their mother had no formal education.[238]

Fats, proteins, zinc and vitamin E are the most important nutrients that influence vitamin A status. The absorption of retinol and carotenoids is lower than normal in individuals with a low fat intake of less than 5 g per day.[239] Vitamin A metabolism is impaired during periods of severe lack of dietary protein as a result of the low synthesis of enzymes, retinoid binding proteins and receptors.[201] Vitamin A deficiency may also result from a reduced synthesis of enzymes, even with adequate intake. Furthermore, liver stores of vitamin A will not be released into the blood stream if there is depressed synthesis of retinoid-binding proteins (RBP).[240] The presence of vitamin E in the liver increases the absorption of vitamin A, while zinc deficiency may contribute to vitamin A deficiency by decreasing the enzymatic oxidation of retinaldehyde in the retinal.[240]

1.3.1   Summary of the Causes of Nutritional Disorders among School-age Children in Kenya

Insufficient dietary intake resulting from seasonal variations of food availability, lack of adequate food and limited dietary diversity have been identified in the literature as causes of nutritional disorders among school children in Kenya. Low socio-economic status of households especially in rural areas and low income urban areas limit households ability to afford a diversified diet thus restricting dietary intake to predominantly plant based sources that are not nutrient dense and provide low bioavailability of nutrients. In addition to insufficient dietary intake, infections such as malaria and hookworm infestation are key contributors to nutritional disorders among school-age children in Kenya

1.4   Nutritional Interventions for School-age Children

Interventions to control micronutrient deficiencies can be classified under three broad categories, namely supplementation, food-based approaches and public health interventions.

1.4.1   Supplementation

Micronutrient supplementation refers to the periodic administration of micronutrients as capsules, tablets or by injection to specific individuals or population groups. This intervention is used for the supply of specific nutrient(s) in a highly bio-available form, and is implicated in the control of known deficiencies in populations. In intervention trials, multiple micronutrient supplementation has a positive impact on growth, cognitive outcomes and nutritional status in school-aged children. A meta-analysis of studies on the effect of micronutrient supplementation on cognition in school-age children reported the likelihood of a small positive effect of multiple micronutrients on reasoning ability and a positive effect on academic performance in children aged 5 to 16 years.[241]

A high micronutrient supplementation providing 100% of the RDA for iodine, iron, vitamin A, riboflavin, vitamin B_6, vitamin B_{12} and folate for children aged 7 to 9 years given over a year established more beneficial linear growth improvements compared to a low micronutrient supplementation of 15% RDA of the same nutrients.[242] In another multiple micronutrient supplementation study, Australian school children aged 6 to 10 years supplemented with a micronutrient mix consisting of iron, folate, vitamin B_6, vitamin B_{12}, vitamin A added at one times the RDA, and zinc added at 50% the RDA, showed a significant increase in scores on tests representing verbal learning and memory.[243] A micronutrient-fortified powder containing 5 mg iron as H-reduced elemental iron, 270 µg of vitamin A as palmitate, 50 µg of iodine as potassium iodide and 5 mg of zinc as zinc sulphate, given with school lunch in Thailand, resulted in a significant improvement in visual recall performance among school-age children.[244] In Kenya, a multiple

micronutrient supplement with vitamin A, vitamin C, vitamin B_1, vitamin B_2, vitamin B_6, vitamin B_{12}, folate, niacin, iron, zinc, copper, iodine and selenium resulted in a 0.92 g/dl increase in haemoglobin levels among school children aged 11 to 14 years.[72]

Micronutrient supplementation often requires a well-organised distribution system and a high degree of consumer compliance, particularly when the supplements need to be taken frequently over an extended period of time.[245] In addition, this strategy often has little involvement of the community in the design and implementation, and this limits its sustainability over time.[246] Micronutrient supplementation programmes are characterised by poor coverage. In Kenya, for instance, where a vitamin A supplementation programme for infants and young children is part of the national nutrition policy, only 30% of children fewer than five years receive vitamin A supplementation and 5% receive iron supplementation.[18] Although it generally is agreed that micronutrient supplementation programmes can have an immediate effect on micronutrient deficiencies,[7] these programmes have to be complemented with more sustainable food-based approaches.

1.4.2   Food-based approaches

Food-based approaches to micronutrient intervention are more sustainable strategies for improving micronutrient status at population level.[7] These strategies include increased food production, dietary diversification and food fortification. Food fortification and dietary diversification have been advocated as two effective food-based approaches for combating micronutrient deficiencies.[247] Food-based approaches increase access to a variety of micronutrient-rich food, contribute to the improvement of general dietary intake, and are able to meet the needs for multiple micronutrients. Using food-based approaches for community-level interventions has multiple social, economic and health benefits, which include increased potential to improve agricultural production and sustain rural households.[245] Food-based approaches bridge the gap that is created by the seasonal availability of micronutrient-rich foods by increasing year-round access to and availability and consumption of an appropriate diet. This promotes the overall health and nutrition of individuals, increases household income and supports sustainable livelihoods at the community level. This sustainability gives food-based approaches an advantage in preventing micronutrient deficiencies, while improving household food security and the overall nutrition and wellbeing of the community.[248]

1.4.3   Food fortification

Micronutrient fortification involves the addition of one or more micronutrients to foods, mainly during industrial processing, so as to increase the intake of the specific micronutrients in order to correct or prevent a deficiency while providing a wider health benefit for a population.[245] Interventions with multiple micronutrients have led to beneficial effects on micronutrient status, improved linear growth and cognitive development in school children.[246] Abrams et al.[196] found a significant beneficial effect of the multiple micronutrient fortification of food on changes in weight, BMI, MUAC and weight-for-age z-scores, as well as changes in serum ferritin, folate, riboflavin, Hb and MCV among school children in Botswana. A reduced prevalence of vitamin A deficiency, from 21% to 11%, and a significant beneficial effect on weight, height and BMI, have been reported as a benefit of multiple micronutrient fortification among school children in Tanzania.[197] The fortification intervention group in Tanzania gained on average 0.55 kg more weight, 0.57 cm more height and 0.35 kg/m^2 BMI compared to controls.[197] Multiple micronutrient-fortified milk resulted in a beneficial effect on change in total bacteria, bifidobacteria and bacteroides at three months of an intervention, with higher amounts of bacteria in the control group, among Australian school-aged children.[249] A significantly higher score on visual recall of familiar objects has been reported among children with multiple micronutrient fortification in comparison to controls.[244] The same study reported a lower incidence of respiratory-related symptoms and diarrhoea in the intervention group in school children in Thailand.

Food fortification programmes use centrally processed foods that require a wide distribution network to reach their intended beneficiaries. This has limited the impact of fortification in resource-poor countries, because of poor distribution networks and low purchasing power, which limit physical and economic access to centrally processed foods.[246] In the rural areas of low-income countries, most foods are often grown, processed and eaten at the community level, thus making it difficult for the most vulnerable people to access micronutrient-fortified food products.

1.4.4   Dietary diversification

The main objective of dietary diversification interventions is to enhance the availability of, access to and utilisation of foods that are rich in bio-available micronutrients by poor families in developing countries.[246] Thus, this approach involves the identification of micronutrient-rich foods, changes in traditional food production practices to increase the supply of such foods, and the promotion of household access to and consumption of these foods. Community-based dietary diversification approaches are more sustainable, have economic feasibility and are culturally acceptable for improving micronutrient status compared to other methods.[250] Examples of

community dietary diversification that have improved micronutrient status are the introduction of yellow- and orange-fleshed sweet potatoes, which resulted in higher vitamin A food frequency scores for children in rural Western Kenya,[251] and increased vitamin A serum retinol concentration in young children in rural Mozambique.[252]

The community-based approach involves building relationships with the community and involving community members in the setting up, execution, monitoring and evaluation processes, with the ultimate goal being to address the key nutritional and related needs in that community.[248] The strategy includes school feeding programmes, agricultural programmes such as homestead gardening, small livestock production and plant breeding, and changing traditional household food preparation and processing methods to ensure the high bioavailability of micronutrients.[253] An example of a community-based and food-based approach and dietary diversification is school feeding programmes that are initiated by the community and that involve food production within the school or the community. This offers an opportunity to increase the production of micronutrient-rich foods within a community, thus contributing to a community's socio-economic development by increasing market demands for the micronutrient-rich foods.

1.4.5   School feeding programmes

School feeding programmes (SFP) are designed mainly to provide meals or snacks to reduce short-term hunger and to improve school children's physical, mental and psychosocial health.[133,254] School feeding programmes have positive impacts on nutritional, education and health outcomes.[255] The nutritional outcomes include improved food energy consumption, growth, body composition and micronutrient status. School feeding programmes are known to increase nutrient intake by participating children. For example, in Peru, an evaluation of a government-sponsored school breakfast programme by Jacoby et al. showed that the energy intake by children receiving school breakfast increased by 15.2%, their protein intake increased by 28%, while their dietary iron intake increased by 60%.[256]

A longitudinal observation was conducted by Arsenault et al.[257] in public schools in Bogota, Colombia to examine if a state-launched snack programme had any impact on the children's nutritional status. The mid-morning snack was designed to provide 30, 50 and 40% RDA of energy, iron and calcium respectively.[257] This study reported an increase in serum vitamin B_{12} levels and a decrease in the prevalence of inadequate vitamin B_{12} status, from 21.9% at baseline to 2.7% after three months. However, the snack was not associated with significant changes in haemoglobin, ferritin or folate status, and the investigators argued that this may have resulted from the decline in

erythrocyte folate that results from treatment with iron,[258] and that the children had relatively high iron intakes during the school year resulting from iron supplementation and school snack interventions.[257]

A randomised controlled trial in Kenya allocated 555 children to three feeding treatment groups (red meat, milk and energy) and one control group (Table 1.1).[259] The meat feeding treatment was made up of *githeri* (a mixture of maize and beans) and ground beef; the milk feeding treatment was made up of *githeri* and cow's milk; while the energy feeding treatment contained *githeri* and cooking fat. At the one-year follow-up there was a significant increase in plasma levels for vitamin B_{12} in the meat and milk groups, which resulted in a decrease in the prevalence of B_{12} deficiency. There was a significant reduction in the prevalence of severe vitamin B_{12} deficiency – by 25.4 and 20.9 percentage points in the meat group and milk group respectively. The change in prevalence of moderate B_{12} deficiency decreased from 80.7% to 64.1% in the meat group and from 71.6% to 45.1% in the milk group.[259]

The evidence for whether school feeding programmes contribute to increasing the nutrient intake of children or whether they are a substitute for food eaten at home is not conclusive. The study in Peru by Jacoby et al.[256] reported that the observed increase in dietary intake in the experimental group was not compensated for by lower dietary intake at home, but rather that the breakfast snack significantly increased overall dietary intakes compared to the control. An empirical analysis was done in India using data from the nationally mandated school meal programme on food consumption during school days and non-school days.[260] The study showed that the meal programme provided significant proportions of daily intakes of energy, proteins, carbohydrates, calcium and iron, indicating that the programme succeeded in improving the dietary intake of those essential nutrients in which the diets of Indian school children were found to be insufficient.

In Kenya, the main provider for school meals has been the Word Food Program (WFP) in partnership with Ministry of Education (Government of Kenya). These programme mainly target the most vulnerable children in the arid, semi-arid areas and those living in slum areas. The coverage for such programme targets about 1.2 million children which is approximately 16% of the children in primary schools in the country.[261] There is no national wide school feeding programme implemented in Kenya. Other initiatives to provide school meals to children in Kenya is limited to programme by specific schools and parents which is a common practice in private schools but less in the public primary schools.

The 1999-2008 World Food Programme (WFP) impact evaluation report on Kenya showed that the WFP school lunch, covering school children in arid, semi-arid and urban slum areas, provided 90 to 100% of the energy RDA for nearly 20% of school children in arid areas.[262] The same report showed that about 30% of the children in WFP school feeding programmes in arid areas were almost completely dependent on school meals to meet the RDA for protein. This WFP report suggests that food sources for children away from school in these areas are highly deficient in essential nutrients.

In addition to the nutritional outcomes of the school feeding programmes there also are educational and learning benefits from providing school-age children with a meal or snack. These include improved cognition and academic performance, increased school attendance and enrolment, and providing encouragement to students to stay in school for longer, thus decreasing dropout rates.[133,255,263] In Bangladesh, Ahmed[254] evaluated the impact of SFPs in the WFP that covered food-insecure areas. The WFP SFP raised school enrolment by 14.2% and net enrolment by 9.6%. The same study reported increased school attendance of 1.3 days a month and a reduction in the dropout rate by 7.5%. In addition, the Bangladeshi children from food-insecure areas who benefited from the WFP school feeding programme increased their test scores by 15.7% points, mainly in mathematics, in which the SFP participants scored 28.5% higher than non-participating children. Similarly, the WFP report on Kenya[262] states that school meals have a positive effect on enrolment and may help to keep children in school through the primary years.

The Kenyan randomised controlled trial discussed earlier found children who received supplementation in meat and energy performed significantly better in arithmetic tests over time compared to the milk and control groups.[264] The meat group in this study had the greatest percentage increase in total test scores (indicators of school performance), mainly in arithmetic, compared to the other feeding groups and controls. However, a previous observational study conducted in Kenya in 1998 found no differences in school attendance rates between schools participating or not participating in a school feeding programme.[265]

School feeding programmes are a broad-based intervention that has the potential to make an important contribution towards the attainment of several Millennium Development Goals (MDGs). School feeding programmes directly address MDG 1, of reducing hunger by half; MDG 2, of achieving universal primary education; and MDG 3, of achieving gender equality. School feeding programmes increase school enrolment, especially among girls, who are sent to school because of the feeding programme, even in areas where sending girls to school may not be a priority for

parents.[254] For example, although food was reported to be the original incentive for sending girls to school in an evaluation done in Pakistan, Morocco, Niger and Cameroon, the parents of the participating girls built up an interest in the education of their daughters.[266] This enhances the parents' loyalty to educating the girls even further than the duration of the school feeding programme.[266] School feeding programmes also serve as delivery points for other beneficial health programmes, like de-worming for intestinal parasites and health education programmes, wider socioeconomic benefits, such as increased household income, and contributing to breaking the intergenerational cycle of undernutrition, hunger and poverty.[262] The potential benefits of school feeding programmes found in the literature are summarised in Figure 1.1, which shows that the wider societal benefits result directly from individual benefits or directly from school feeding programmes.

There are a number of factors that have an impact on the implementation and success of school feeding programmes. Greenhalgh and colleagues[267] reviewed studies on school feeding programmes to understand their efficacy. In their review they identified a number of process factors that seem to enhance the efficacy of school feeding programmes:[267]

- well-organised schools that form part of an efficient distribution chain,
- target group with a clear nutritional deficiency, mainly of energy, which the trial aims at correcting,
- intervention developed with local teams rather than distant experts,
- piloting of a supplement in order to exclude intolerance and establish palatability and acceptability
- close supervision during implementation

School feeding programmes may not always provide positive outcomes. The success of a school feeding programme is hindered when the offered food is not consumed, or when it does not provide an adequate amount of the missing nutrient.[256] Low bioavailability of nutrients in the school meal can result in a lower than expected outcome. For instance, Grillenberger et al.[66] suggested that a milk supplement given in the Embu study in Kenya may have contributed to a decreased absorption of iron and zinc. A compensatory adjustment of appetite has been reported in some studies, where supplemented children tend to eat less at the next meal.[268] In addition, substitution may occur, in terms of which children participating in school feeding programmes are provided with less food at home. This was suggested to be the cause of lower than expected benefits from food supplementation in a study undertaken by Powell et al. in Peru[268,269] and Agarwal et al. in India.[269] A lack of proper implementation of school feeding programmes can result in less than expected

benefits. This can occur if the study deviates from the original protocol as a result of ethical issues, or because of a lack of cooperation from control groups.[267] For example, a study design that requires nutritious meals to be served to one group and not another can result in ethical difficulties preventing the successful implementation of such a design. This calls for consultation with and the involvement of communities and target groups at the design level in order to resolve any design difficulties and to incorporate the views of the recipient community.[267]

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│       School feeding programmes      │
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┌─────────────────────────────────────┐
│         Impact on individuals        │
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Nutritional benefits

- Relief of hunger – MDG 1
- Improved food consumption
- Increased nutrient intake
- Improved growth
- Improved body composition
- Decreased micro-nutrient deficiencies
- Increased bone mineral density

Educational benefits

- Increased enrolment – MDG 2
- Increased attendance
- Increased time spent at school and reduced absenteeism
- Enrolment of girls – MDG 3
- Increased school time quality
- Improved cognition
- Improved school performance

Other health benefits

- Reduced intestinal parasites
- Nutrition education programmes
- Sanitation and hygiene
- Improved behavioural outcomes

Wider socio-economic and societal benefits

- Increased household income
- Food security
- Increased productive life years
- Positive effect on disability-adjusted life years
- Enhanced disease awareness
- Higher earnings over longer time period
- Improved quality of life
- Stimulated local agricultural activities
- Increased local market value of food
- Gender equality
- Intervening in the intergenerational cycle of hunger, undernutrition and poverty

FIGURE 1.1: Potential benefits of school feeding programme

Source: Compiled by author from literature[133,254,255,260,263,267]

1.4.6   Summary of the implementation of school feeding programmes in Kenya

The main provider for school meals has been the WFP in partnership with the Ministry of Education and concentrates on children living in arid, semi-arid and slum areas. Besides the programme by WFP, there is no national school feeding programme in the country. Other school feeding programme programme are initiatives by individual schools where parents pay for the programme. The benefits of school feeding in Kenya have been demonstrated in terms of increased school participation, higher grades, higher school enrolment, increased attendance rates and increased number of attendance by girls.

1.5   Motivation for the Research

Although the benefits of school feeding are well documented, evidence of the impact of SFPs on children's growth and body composition remains inconclusive due to the mixed results reported by different studies.[254,263,270-272] Studies in developing countries investigating whether the baseline nutritional status of school children modifies the effect of providing them with a meal at school are few and far between. In 1998, a study in Jamaica, where a breakfast snack with 567 kcal and 27.1 g protein was provided for eight months reported no interaction between school feeding and baseline nutritional status based on weight.[268] The same study demonstrated a significant gain of 0.3 cm in height in the undernourished group, and a non-significant gain of 0.17 cm in the well-nourished group. A more recent study in Kenya in 2007 reported a greater rate of height gain in younger stunted children ($\leq$ 6 years) receiving a glass of milk in addition to a lunch snack of maize and beans compared to others receiving a lunch snack of maize and beans or one of maize, beans and meat.[264] The authors of the same Embu study found that children who were stunted at the beginning of the study seem to have benefited more from the intake of total energy and other nutrients than the non-stunted children.[45]

The Embu study used ground meat that was added to the usual meal of maize and beans that the children commonly ate at home in the area, and milk to increase the intake of animal-source foods. Our study sought to determine the effect of initial nutritional status on the responses to a school feeding programme in the Millennium Villages Project within a framework of community participation in the design, implementation, monitoring and evaluation of the project. Besides the studies done by Neumann and colleagues[66] in Embu, Kenya, our search did not find any other documented intervention studies in Kenya that have assessed the effect of initial nutritional status on the outcomes of a school feeding programme, especially one that includes animal-source foods.

In order to incorporate the locally available foods and the inputs of the recipient community, our study included small whole fish (*omena*) and maize meal (*ugali*), which commonly are consumed in

the area, and included participation by the community at every level from design to implementation. The implementation of SFPs in developing countries faces many and continuous challenges. Sustainability, the procurement of food in the light of food price fluctuations and environmental and agricultural changes, as well as cost are common challenges faced by SFP designers and planners. The effectiveness of school feeding programmes is dependent on the selection of modality, the effectiveness of targeting and the associated costs.[273] This study was carried out within the framework of the Millennium Development Villages (MVP), which is a community-driven initiative to achieve MDGs in a rural village in Kenya, as well as in other African villages. More details on this programme are provided in the section on the study methods.

The findings of the MVP baseline survey conducted in Bar Sauri village, Kenya in 2005 (n = 966) showed an overall prevalence of anaemia of 64%, 77% among children under the age of five years and 48% among children aged 6 to 15 years.[274] One out of every five children under the age of five years had severe anaemia, with haemoglobin of < 7 g/dl. Based on the assumption that maize comprised 35% of the total energy intake in Bar Sauri village, three quarters of the households had inadequate energy to meet the needs of the household members, while 95% of the households were energy deficient if maize consumption made up 100% of energy intake,[274] indicating the extent of chronic hunger in this population. It is against this background that this study was designed to cover the deficit in dietary intake and address the high levels of anaemia, as well as to determine the effects of initial nutritional status on the responses to the school feeding programme in terms of growth, body composition and micronutrient status.

The gap identified in knowledge from this review is the lack of sufficient information on the effect of a child's initial nutritional status on how they respond to school feeding intervention programs. This is an important issue to highlight since many studies included in this review did not separate the children with poor nutritional status at the baseline to see how they respond to the intervention. Our study sought to bridge this gap in knowledge.

1.6   Statement of the Research Question

The following research question was investigated in this study:

> "What is the effect of initial nutritional status on the responses of school children to school feeding programmes in the intervention and control groups in terms of growth, anaemia prevalence, body iron stores and vitamin A status in school children in the Millennium Villages Project?"

The challenge from a public health perspective is to develop and implement sustainable nutrition intervention programmes that will address the nutrient deficiencies among school-age children, while at the same time providing an avenue for the attainment of the MDGs. In Kenya, interventions to address the nutrition deficiencies of school-age children have focused mainly on arid and semi-arid areas, as well as urban slum areas. In the majority of rural households in Kenya, school children continue to attend school on empty stomachs, thus compromising their nutritional status and school performance, as well as other health outcomes. This challenge is even greater among children who already have compromised nutritional status. It is important to establish the response to a school feeding programme that includes animal-source foods by these children with already compromised nutritional status. This study therefore seeks to establish the effect of initial nutritional status on the responses to a school feeding programme.

1.7   Significance of the Study

The study provides information on the impact of a community-based school feeding intervention programme using locally available and accepted animal source foods to improve the growth, body composition, levels of anaemia, body iron stores and vitamin A status of school children. Baseline measurement provides further data on the nutritional status of school children in Kenya. The study also provides information on the effect of a child's initial nutritional status on his or her responses to a school feeding programme in the intervention and control group, and makes a contribution to the attainment of the MDGs in the Millennium Village Project in rural Kenya.

The findings are very useful, particularly to the Ministry of Education and the Division of Nutrition at the Ministry of Health, for designing nutrition interventions for school-age children that are targeted at meeting the needs of children who have compromised nutritional status. The community-based model provides an opportunity to direct the methods of managing and implementing school feeding programmes that are sustainable and at the local level. The community participatory approach also increases compliance with and sustainability of the programmes, offering a model for community empowerment that demonstrates how a community can initiate, own and implement successful programmes to meet their own nutritional needs. This study also offers an example of the successful efforts going towards the attainment of MDGs particularly on provision of basic education (goal 2), eradication of extreme poverty and hunger (Goal 1; target 2) and developing a global partnership for development.

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1.8   Conceptual Framework

The conceptual framework, as presented in Figure 1.2, was developed from the literature and shows that school feeding programmes have the potential to impact on the health and nutritional outcomes of school-aged children, as demonstrated in the literature review. The potential health and nutrition benefits investigated in this study include the impact on growth, body composition and anaemia, body iron stores and vitamin A status. The effect of the initial nutritional status of school children at baseline on responses to the feeding programme is also determined, as well as the effects of socio-economic and background characteristics.

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Household socio-demographics and background characteristics		Outcome measures

Household socio-demographics and background characteristics
- Household income
- Education of household head
- Mother's education
- Child's age (school age; 6 to 13 years)
- Child's sex
- Religion

Intervention
- School feeding programmes with animal-source foods (*Omena* fish and beef)

- Community participation

Duration
30 months' follow-up

Intervening variable
Initial nutritional status

Control
- School feeding programmes with maize and beans

- Community participation

Duration
30 months' follow-up

Outcome measures
Impact of school feeding in the intervention and control groups

- Growth
- Body composition
- Prevalence of anaemia
- Serum ferritin levels
- Body iron stores
- CRP levels
- Serum retinol levels
- Vitamin A deficiency

Effect of initial nutritional status on:
- Growth
- Body composition
- Prevalence of anaemia
- Serum ferritin levels
- Body iron stores
- CRP levels
- Serum retinol levels
- Vitamin A deficiency

FIGURE 1.2: Conceptual framework of the effects of initial nutritional status on responses to a school feeding programme

Source: Compiled by author

2   CHAPTER TWO: METHODOLOGY

2.1   Research Aims

The aim of this study was to assess the effects of initial nutritional status on the responses to a school feeding programme among school children in the Millennium Villages Project, Siaya District, Kenya.

2.1.1   Objectives

A) Among school children aged 6 to 13 years participating in the Millennium Villages Project in Siaya District, Kenya, and in the control group, the study was conducted to determine, at baseline:

1.   Dietary intake, through a structured food frequency questionnaire

2.   The prevalence of undernutrition, based on anthropometric measurements

3.   Body composition, on the basis of anthropometric measurements

4.   Body iron stores and the prevalence of anaemia, by serum ferritin and haemoglobin concentration

5.   Vitamin A status, by serum retinol concentration

6.   The level of inflammation, based on serum C-reactive protein (CRP)

B) To assess the impact of the school feeding programme in the form of two different lunches over a 30-month period on:

1.   Nutritional status and body composition, based on anthropometric measurements

2.   The prevalence of anaemia and body iron stores, by haemoglobin and serum ferritin concentration

3.   Vitamin A status, by serum retinol concentration

C) To assess the effect of initial nutritional status on the responses to two school feeding programmes on growth, the prevalence of anaemia, body iron stores and vitamin A status among school children.

2.1.2   Null Hypotheses

H_0:   There is no difference in the impact of two different school lunches on the anthropometric status, body composition, iron status and vitamin A status and prevalence of anaemia in school children.

H_0:   Initial nutritional status has no significant effect on the responses of school children in the Millennium Villages Project to two school feeding programmes in terms of growth, anaemia prevalence, body iron stores and vitamin A status.

2.2   Description of the Study Area

The study was carried out in the Sauri Millennium Villages Project (MVP) in Kenya. The Sauri MVP is located in Bar Sauri sublocation in Sauri location, in the Yala Division of Siaya District in Nyanza Province. Figure 2.1 and the map in Figure 2.2 show the administrative units in the districts and the location of Sauri village within the district. The district covers an area of approximately 1,520 square kilometres and lies between latitude 0°26' to 0°18' North and longitude 33°58' East and 34°33' West[275] on the Kenya highlands at 1,400 to 1,500 meters above sea level. It is located about 350 kilometres from the Kenyan capital, Nairobi, and lies to the West of the Rift Valley and 30 kilometres North of Lake Victoria. The general topography is characterised by transient streams, rivers and wetlands that meander through rounded hills.[276] The study area is mostly rural, with subsistence farming as the primary occupation. The short rains usually occur from August to December, with an average of 1,200 mm, while the long rains usually occur from March to June, averaging about 1,900 mm. Ecologically, the District spreads across the agro-ecological zones Lower Midland (LM_1 to LM_3), with a small area under Upper Midland (UM) (Figure 2.2).

The total population of Siaya District in 2009 was 842,302.[277] There were 381 primary schools in the district, with a total enrolment rate of 97.4%. The average number of years of school attendance by boys was 4.5 years in 2009, while it was 3.8 years for girls. The total population of primary school-going age (6 to 13 years) in 2009 was 120,718. The district has 3 hospitals, 15 health centres and 19 dispensaries. Siaya district is considered a malaria holoendemic area of *Plasmodium falciparum* transmission.[278] The average distance to a health facility is 6 kilometres. Yala Division is one of the 7 administrative divisions of Siaya District and covers 209.8 square kilometres, with a population density of 410 persons per square kilometre. Sauri location is one of the 4 locations of Yala Division, and is further subdivided into 19 sub-locations. Sauri sub-location had a total of 967 households in 2005, with an average of 5.7 persons in each household at the MVP baseline survey.[274] The population density of Sauri is high, with close on 700 people per square kilometre, and agriculture is the primary livelihood source in the area.[274] According to the MVP Sauri baseline survey, the net primary school enrolment in Sauri was 84.6%, the net secondary school enrolment was 19.2% and the net tertiary level enrolment was 4%. In 2006, 60% to 70% of the population in Yala Division was living below the Kenyan poverty line of $1 a day.[274] The control group was drawn from the same division but in schools outside of the MVP area, at the start of the study. These schools were located in the Central and East Gem locations (Figure 2.1).

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                        │   Nyanza Province   │
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                                  │
                        ┌─────────────────────┐
                        │    Siaya District   │
                        └─────────────────────┘
                                  │
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                        │      Divisions      │
                        └─────────────────────┘
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| Ukwala Division | Boro Division | Karemo Division | **Yala Division** | Uranga Division | Ugunja Division | Wagai Division |

| **Bar Sauri Location (MVP)** | | Central Gem Location | East Gem Location |

| **Bar Sauri Primary** | **Nyamninia Primary** | **Bar Turo Primary** | Muhanda Primary* | Migosi Primary* | Kanyuto Primary* |
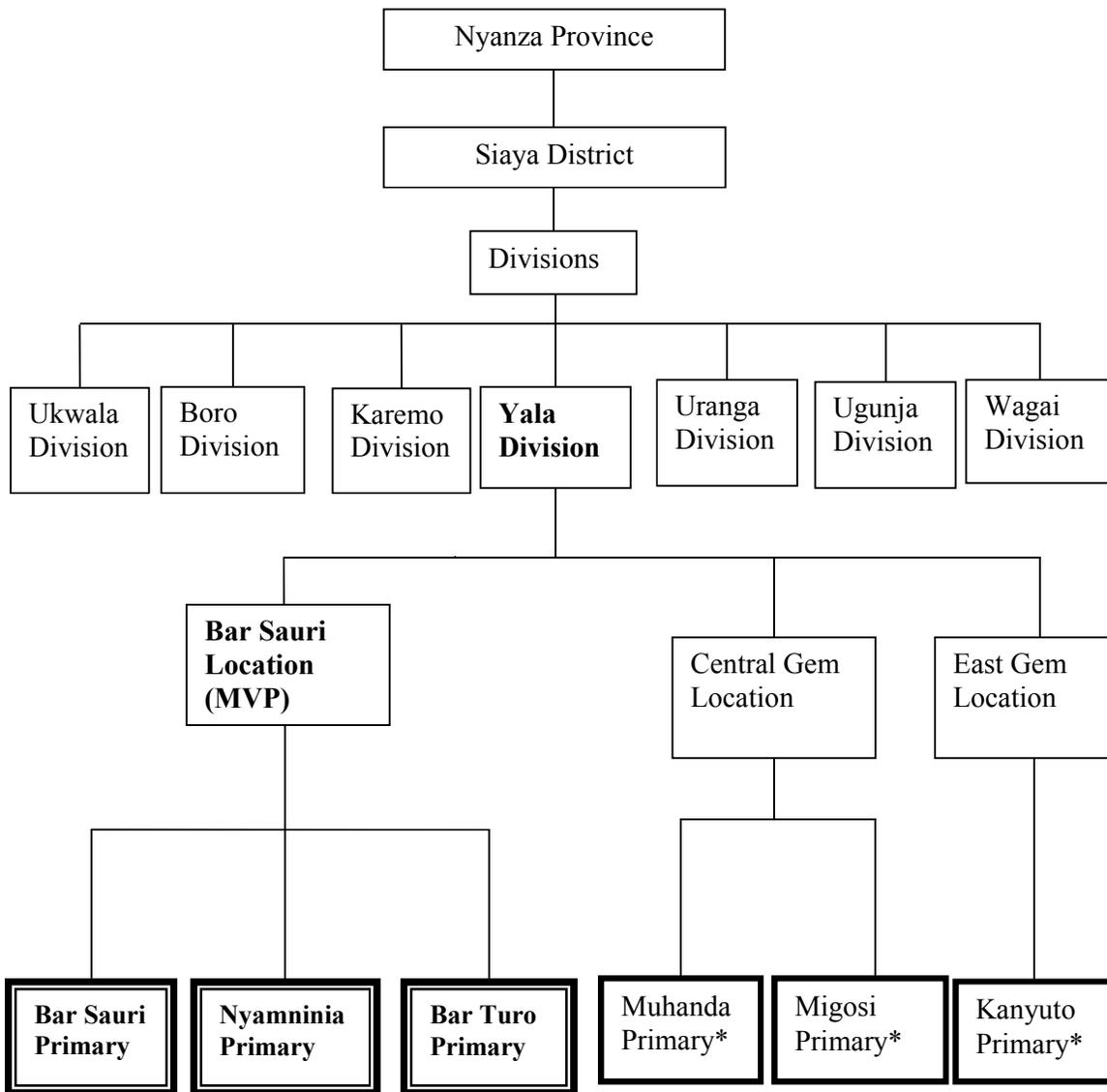
The intervention schools are indicated in bold, while the control schools are indicated with an asteriks (*)

FIGURE 2.1: Description of the location of the study population within the administrative units of Siaya District

Location of
Siaya District in
Kenya

Location of Yala Division in Siaya District, showing agro-ecological zones

a= Intervention schools b= Control schools

Location of the primary schools included in the study in Yala Division
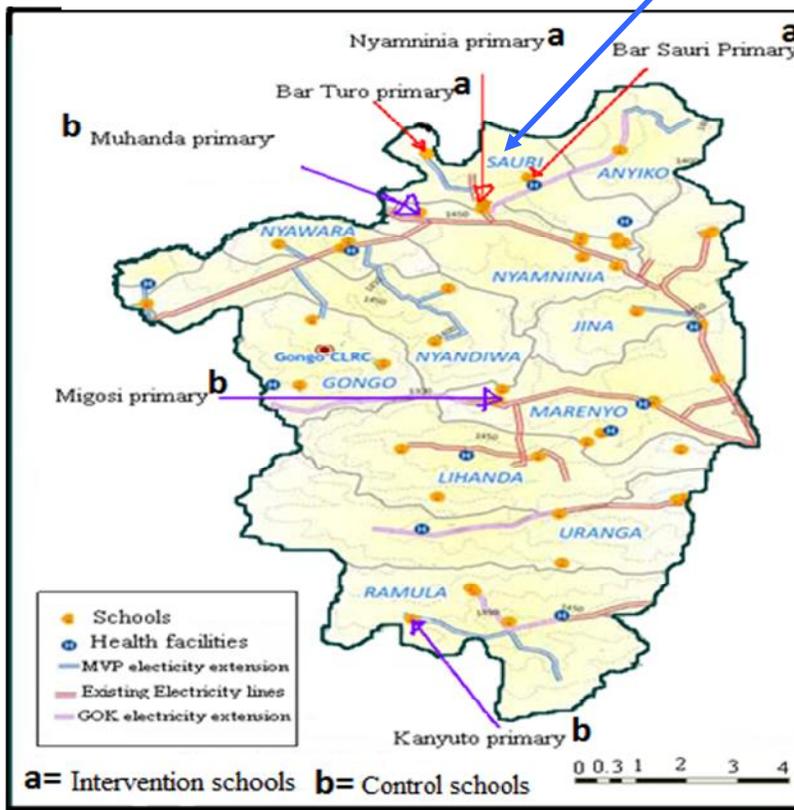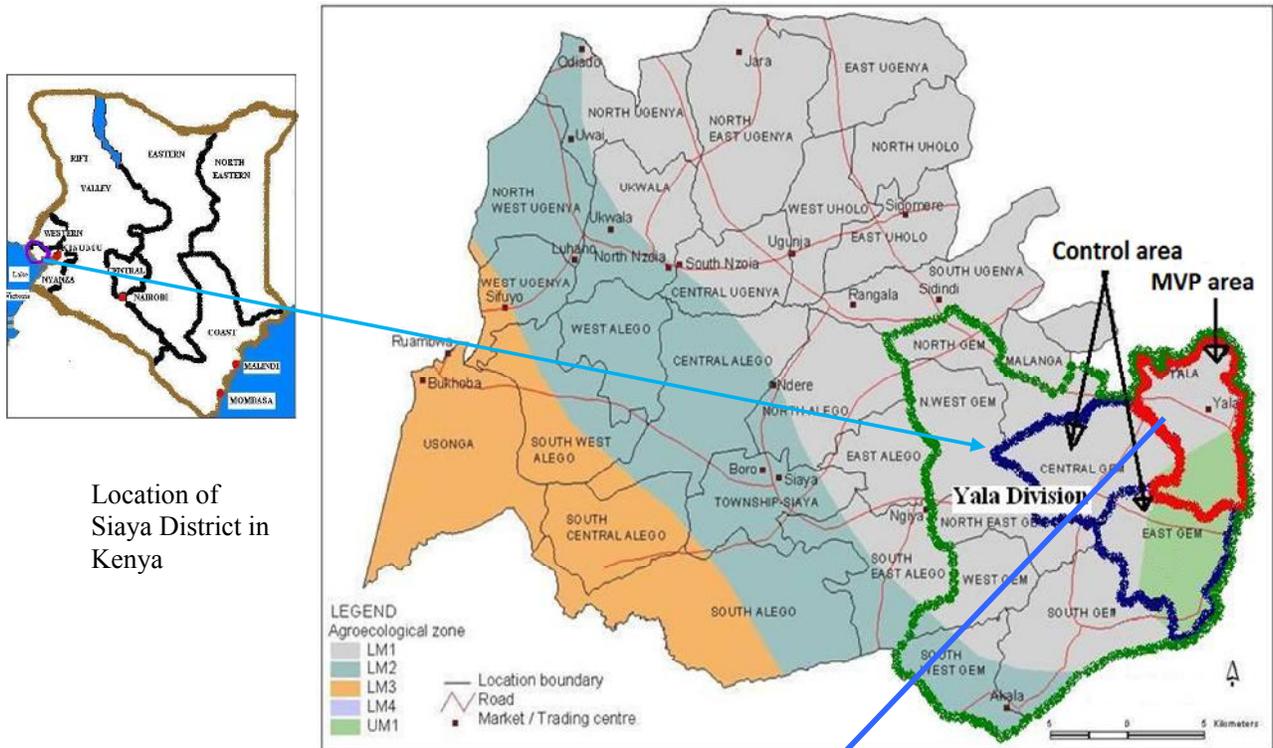
FIGURE 2.2: Map of the Millennium Villages Project

2.2.1 Millennium Villages Projects

The MVPs are an initiative of the Earth Institute at Columbia University and the United Nations (UN) Millennium Project.[279] The MVP aimed to empower and work with impoverished communities in rural Africa to achieve the United Nations Millennium Development Goals (UN MDGs) within 10 years after the establishment of the first MVP in 2004. The first MVP, which is the site of this study, was started in Kenya in July 2004, specifically in Bar Sauri village, Yala Division, Siaya District, Nyanza Province. The MVP in Bar Sauri was scaled up in January 2007 to other villages in the Siaya District. By January 2009, there were 12 MVPs in 10 countries of Africa: Kenya, Ethiopia, Ghana, Malawi, Mali, Nigeria, Senegal, Rwanda, Tanzania and Uganda, as illustrated in Figure 2.3. These villages were carefully selected to represent each of the 12 principal agro-ecological zones that reflect the range of farming, water and disease challenges on the African continent.[280,281]

The MVPs are community-driven projects that are guided by the recommendations of the UN Millennium Project on the types of interventions in agriculture, nutrition, health, education, energy, water, communications and the environment.[276] A key feature of the MVP is community empowerment and local capacity development to enable villages to pull themselves out of poverty. In Bar Sauri, the MVP worked with the local community to create a set of committees that included an executive committee, and committees on agriculture, environment, health, water, energy, roads and communication, business, and education. Each village in the sub-location elected three members to each committee, which, in turn, elected a chairperson. The committees were then charged with designing community action plans in their area of specialisation. The committees most relevant to the interests of this research were the executive committee, education committee, and roads and communication committee.
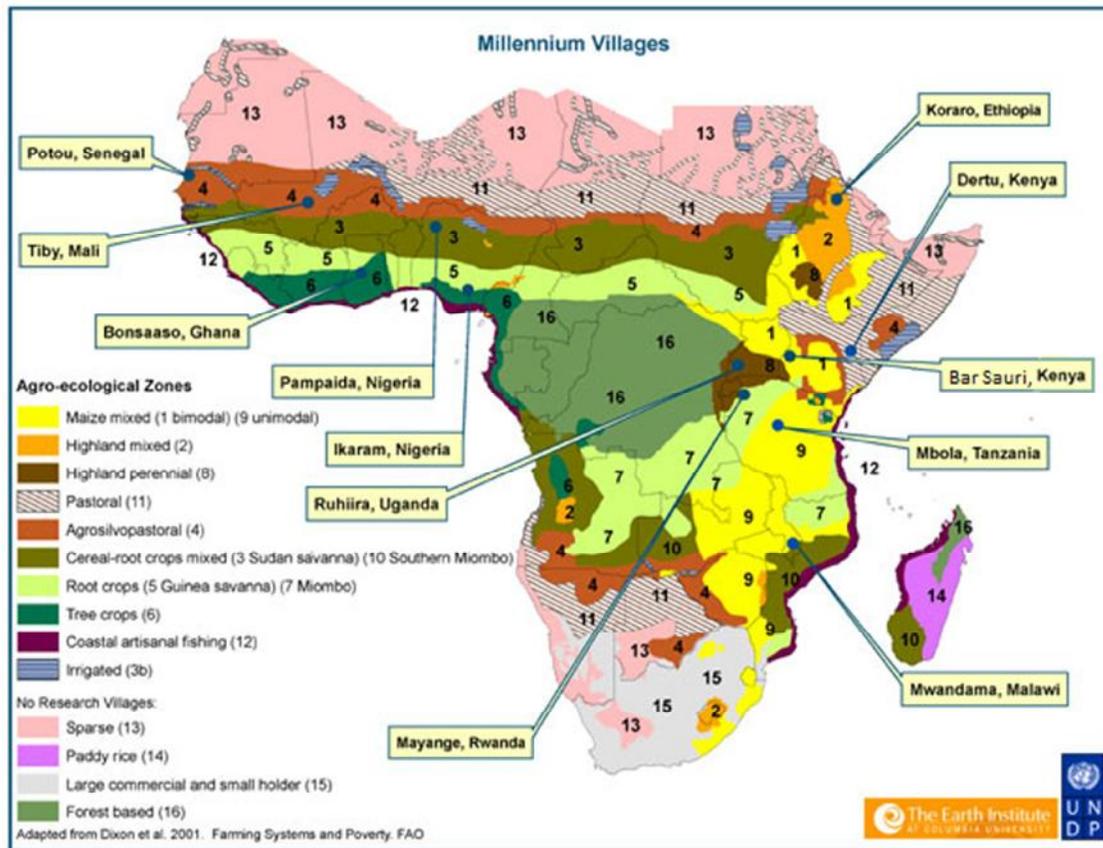
FIGURE 2.3: Map of the distribution of the Millennium Villages Projects in Africa and the agro-ecological zones.

Source: Columbia University[13]

2.3 Study Design

This was a longitudinal study designed to follow up school children for a period of 30 months. Two groups of school children, aged between 6 and 13 years, were recruited at the baseline (June 2006) and followed up for a period of 30 months until January 2009. The intervention group of children attended one of three schools, either Bar Sauri, Bar Turo or Nyamninia Primary Schools in the Bar Sauri sub-location in the MVP (Figure 2.1 and 2.2). The three schools in the control group were the Muhanda, Migosi (Central Gem), and Kanyuto (East Gem) Primary Schools in Yala Division.

2.4 Study Population

The study population was school children aged 6 to 13 years who were in nursery school or class 1, class 2 and class 3 in primary school. The three schools in the MVP had a total population of 1,598 children, while the schools in the control group had a total population of 1,650 children.[282] Of the 1,598 children in the intervention group, 627 were within the desirable age of 6 to 13 years and in school classes between nursery school and primary school class 3, while the number of children

who fell within the required age range in the control group amounted to 584 (Figure 2.4). The control schools were within a 15 kilometre radius of the intervention schools and fell within the same division, which means that the households were likely to have similar socio-economic and demographic characteristics.

2.4.1   Selection criteria

Inclusion criteria

- Children 6 to 13 years old attending primary schools in the study area.
- Children who assented to participate in the study and whose parents consented.

Exclusion criteria

- Children with known mental retardation (confirmed by parent and class teacher) were excluded from the study. However, the children were still included in the feeding programme.

2.5   Determination of Sample Size

The sample size was determined using the comparison of two Bernoulli proportions[283] with an alpha of 0.05 and beta of 0.10. The proportion p1 and p2 was 0.5 and 0.19 respectively. P1 was taken as the estimated probability of a success in a Boernoulli trial[282] while p2 was the proportion of school children with stunting in rural Kenya.[50] The required sample size was 118 subjects in each cohort, thus a total of 236 children. The model for the determination of the full sample size was provided by the Centre for Statistical Consultation at Stellenbosch University (Appendix 1).

2.5.1   Sampling method

All three schools participating in the MVP were included in the intervention group. The three control schools were selected to match the MVP schools in terms of pupil numbers and being from the same division. They were within a radius of 15 kilometres from the MVP schools for ease of accessibility. In order to determine the number of children from each school to be included in the sample per class, proportionate allocation using a sampling frame of n/N was used where n is the sample size and N the population sample. This was calculated using the proportionate sample size calculator provided by the Centre for Statistical Consultation at Stellenbosch University (Appendix 1). In this method, the population is usually divided into sub-populations (strata) and random samples taken of each stratum. Using this method, the sample selected for inclusion in the study was 57 children in nursery school (28 and 29 for the MVP and the control respectively), 77 children in class 1 (39 and 38 in the MVP and the control respectively), 51 in class 2 (26 and 25 in the MVP

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and the control respectively), and 51 in class 3 (25 and 26 in the MVP and the control respectively; see Figure 2.4).

To select the children in the individual schools, and in the appropriate classes, multi-stage sampling was used. First, the children were stratified by class and a list of all the pupils in nursery school, as well as in class 1, 2 and 3, was obtained from each school and used as the sampling frame. The lists of names were typed into SPSS version 12 software[284] and then counterchecked with the schools to ensure that these children were all available at the school. Children found to have been transferred to other schools but who still were on the teacher's list were removed. A random sample was then drawn to obtain the specific number of children from each class. The selected children were taken as the sample and identification numbers allocated to them. These identity numbers were used throughout the data management to maintain subject confidentiality.

Sub-sampling

A random subsample of 103 children was selected from the combined intervention and control groups using a similar procedure as described above to select a representative sample of the children for the analysis of serum ferritin and retinol.

2.6   Sample Recruitment

The study team, consisting of the principal investigator (PI), PK Masibo, and two field workers, visited each of the schools to identify the children who had been sampled. The parents of these children were then invited to the school and the study procedures were explained to them. The children were also given details of the study and asked for their assent to participate. All the parents of the sampled children gave written consent and all the children assented to the study. All the other children in the schools participated in the school feeding programme, even if they were not sampled for the study.

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                        ┌─────────────────────┐
                        │   Siaya District    │
                        └─────────────────────┘
                                  │
                        ┌─────────────────────┐
                        │   Yala Division     │
                        └─────────────────────┘
                    ┌─────────────┴─────────────┐
          ┌───────────────┐           ┌───────────────────┐
          │      MVP      │           │  Non-MVP controls │
          └───────────────┘           └───────────────────┘
                  │                             │
```

MVP	Non-MVP controls
Bar Sauri, Nyamninia and Bar Turo primary schools Total population = 1 598 children	Muhanda, Migosi and Kanyuto primary schools Total population = 1 650 children
Study population (6 to 13 years) N = 627 Nursery school - 130 Class 1 - 192 Class 2 - 151 Class 3 - 154	Study population (6 to 13 years) N = 584 Nursery school - 116 Class 1 - 150 Class 2 - 137 Class 3 - 181
Proportionate random sample size (n = 118) Nursery - 28 Class 1 - 39 Class 2 - 26 Class 3 - 25	**Proportionate random sample size (n = 118)** Nursery - 29 Class 1 - 38 Class 2 - 25 Class 3 - 26

FIGURE 2.4: Description of the sampling strategy for the groups to be studied

2.6.1   Allocation to study cohorts

The children included in cohort 1 were all those attending the three primary schools in the MVP, and those allocated to cohort 2 were those attending the sampled primary schools in the control area, also referred to as the control in this study. At baseline (June 2006), the primary schools in the MVP started participating in a school feeding programme and continued to do so for the next 30 months of the follow-up period. The schools in the control areas were not in any school feeding programme before inclusion in the study. In the 6[th] month of the study (January 2007), the project implementers (Earth Institute, Columbia University) scaled up the Millennium Villages concept to cover other areas of Yala Division, which included the control areas. However, the scale-up did not include introducing the diversified MVP school feeding programme (described in section 2.7) in the control areas. Instead, the Earth Institute introduced a basic meal of maize and beans as a school lunch for the control group, among other scale-up aspects such as the upgrading of the roads, the construction of health facilities and agricultural interventions. Therefore, the control group started

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having a lunch of maize and beans (*githeri*) in January 2007, in contrast to the MVP group, which had started participating in the school feeding programme in June 2006. However, all the data was collected for all the groups at baseline in June 2006 (Figure 2.5).

2.7   Description of the Intervention

The school feeding intervention was designed using locally available foods and with the participation of the local community, school children and organised village committes.

2.7.1   Identifying locally available foods

A survey was conducted in two local open-air markets in the study area, namely Yala and Luanda markets, which operated on two days of the week – Mondays and Thursdays for Yala and Tuesdays and Fridays for Luanda. The survey was done to identify locally available foods (see the Market survey tool in Appendix 2) in May 2006 and, and compile a list of all the foods available in the market per food groups (TABLE 2.1).

A meeting was held between the PI (PK Masibo) and the members of the local education committee formed by the MVP implementation office to brainstorm on the locally available foods that could be included in the school feeding programme. All the foods identified during this meeting were compiled and added to the list from the markets. The PI then counterchecked to make sure all the listed foods had been included. Names of the foods listed in the local languages were translated into English and the foods were grouped into seven food groups, namely starches, legumes and grains, vegetables, fruit, fats and oils, sugars, seeds and nuts (TABLE 2.1).

TABLE 2.1: Locally available foods in the study area: results of market survey

Food group	Types of food locally available
Starches	Maize, maize flour, wheat, wheat flour, rice, green bananas, Irish potatoes, bread, pumpkin, butternuts
Legumes and grains	Cowpeas, bean varieties, pea varieties, green grams (*dengu*), millet, sorghum and lentils
Meat	Cow, chicken, fish varieties, *omena* (small 2-inch dried whole fish), tilapia (*ngege*), Nile perch (*mbuta*), eggs, liver, offal, goat, sheep, lamb, birds, ducks and doves
Vegetables	Kale (*sukuma wiki*), cabbage, cowpeas and bean leaves, indigenous varieties (*omboga, osuga, ododo, kandhira, nyatigatiga*), mushroom, carrots, tomatoes, garlic, pepper, onions and coriander leaves (*dhania*)
Fruit	Avocado, guava, mango, banana, orange, lemon, pawpaw, pineapple, watermelon, tangerines, plumps and pears
Fats and oils	Hydrogenated vegetable fats, liquid vegetable oils, milk fat, ghee and fat from animal meat
Sugars	Refined sugar, sugar cane, honey, *sukari nguru* (unrefined sugar)
Seeds and nuts	Sesame seeds, ground nuts
Roots and tubers	Cassava, sweet potatoes, arrowroot
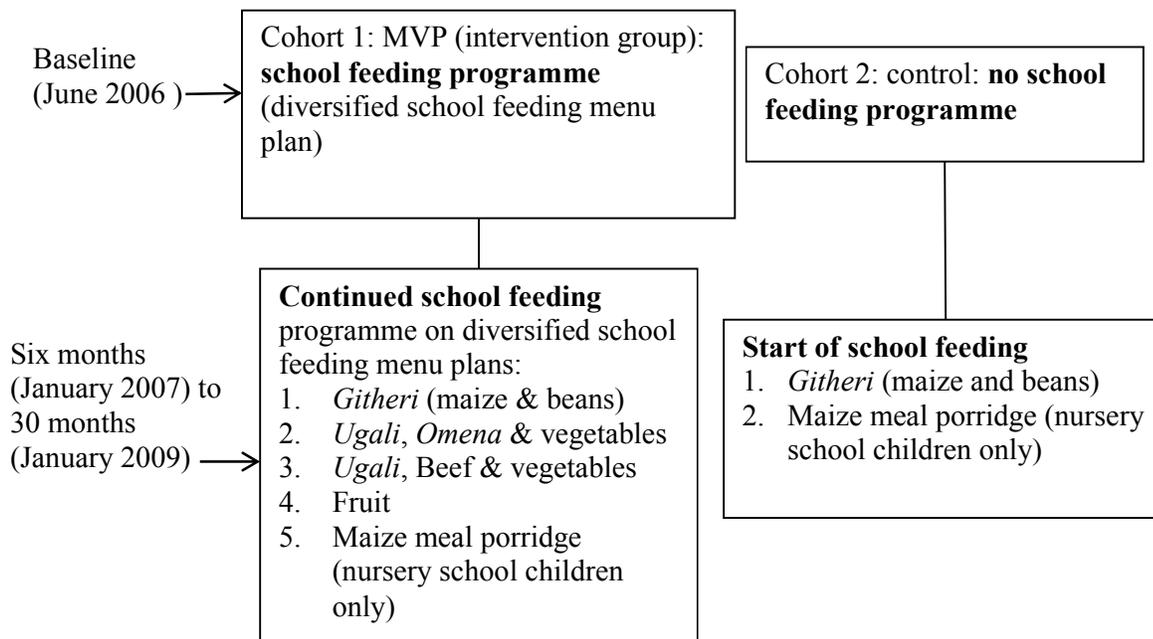
FIGURE 2.5: Allocation of study groups

2.7.2   Nutrient content of the school feeding programme

The nutrient content of the three menus that were designed was calculated on the basis of Kenyan food composition tables,[285] the food composition tables for Africa,[286] and a search of the USDA nutrient database[287] for nutrients like zinc that are not available in the Kenyan food composition tables. This calculation of nutrients was for each of the ingredients (cooked) in the recipes. A serving of 300 g of *githeri* provided 387 kcals (1 620 kJ) of energy, 17 g of protein and 4.6 g total fat (TABLE 2.2). An average serving of 323 g of *Ugali, omena* and cabbage provided an average of 467 kcals (1,954 kJ) of energy, 17.1 g of protein and 4.6 g of fat. *Ugali* and *omena* provided an average of 64.2 µg of Vitamin A and 16.3% of EAR for folate. An average serving of 312 g of the *ugali*, beef and kale provided 467 kcal (1,955 kJ) of energy, 12 g of protein and 9 g of fat (TABLE 2.3). The average nutrient content presented in TABLE 2.3 includes the contribution of maize meal porridge taken by the nursery school children and the fruits taken by children in the MVP. A complete nutrient content analysis is presented in Appendix 3.

2.7.3   Determination of portion size

In order to determine portion sizes for the food, school children in the participating schools were grouped into three categories according to their class level in school and their age distribution. The categories were: nursery class (age 4 to 6 years); class 1 to 3 (age 7 to 10 years) and class 4 to 8 (11 to 15 years). Although the study only covered children up to class 3, the portion sizes were determined for all the classes because the children were followed up for 30 months during the study and the older children were 15 years old by the end of follow-up period. The average serving for the *githeri* meal was 300 g per child, *ugali* and *omena* was 323 g, while the average serving for the *ugali* and beef was 312 g (TABLE 2.2). When children moved to a new class in a new year, the amount served was adjusted to correspond with their growth.

TABLE 2.2: Macronutrient content of the school feeding programme

Menu type	Age group	Serving size (g)	Energy (kcals) (kJ)	Percent EER	Carbohydrates (g)	Percent EAR	Proteins (g)	Percent EAR	Fat (g)	Percent AMDR
Githeri (maize and beans mixture)[2]	4 – 6 yrs[1]	200	372 (1559.2)	20	84.6	41	14.1	65.3	5.5	14.8
	7 – 10 yrs	300	387.4 (1619.9)	20.4	72.4	31	17.1	90.0	4.6	10.7
	11 – 15 yrs	400	510.4 (2136.4)	22.3	96.5	74.2	22.8	67.1	5.4	9.5
	Average serving	300	387.4 (1619.9)	20.9	72.4	55.7	17.1	74.1	4.6	10.7
Ugali, omena and cabbage	4 – 6 yrs[1]	230	443.8 (1858.1)	23.3	65.8	50.6	13.0	68.4	9.0	22.0
	7 – 10 yrs	340	474 (1984.1)	25	88.6	68.2	16.5	86.8	9.9	18.8
	11 – 15 yrs	400	557.6 (2334.6)	25.3	103.8	79.8	20.2	59.4	11.7	18.9
	Average serving	323	466.8 (1954.4)	24.5	86.1	66.2	16.6	71.6	10.2	19.7
Ugali, beef and kale	4 – 6 yrs[1]	220	439.8 (1841.4)	23.0	61.9	47.6	9.6	50.5	8	19.7
	7 – 10 yrs	330	479.5 (2007.6)	25.3	83.5	64.2	12.8	67.4	9.1	17.1
	11 – 15 yrs	385	556.4 (2329.5)	24.4	97.4	74.9	14.9	43.8	10.3	16.7
	Average serving	312	466.9 (1954.8)	24.2	80.9	62.2	12.4	53.9	9.1	17.5

EER = Average estimated energy requirement for active physical activity level
EAR = Estimated average requirement rate
AMDR = Acceptable macronutrient distribution range
[1] Macronutrient contents from the maize meal porridge is included
[2] Macronutrient contents from fruits (mango, banana, orange) is included

TABLE 2.3: Micronutrient content of the school feeding menus

Menu type	Age group	Serving size (g)	Iron (mg)	Available Iron	Percent EAR	Calcium (mg)	Calcium AI	Zinc (mg)	Percent EAR	Vitamin A (µg)	Percent EAR	Folate (µg)	Percent EAR
Githeri (maize and beans mixture)[2]	4 – 6 yrs[1]	200	4.7	0.2	5.7	84.3	10.5	1.2	30.0	0.1	0.0	132.2	82.6
	7 – 10 yrs	300	6.2	0.3	5.3	45.2	4.3	1.7	30.9	0.1	0.0	195.2	95.2
	11 – 15 yrs	400	8.3	0.4	7.0	60.2	4.6	3.4	48.6	0.1	0.0	276.2	110.5
	Average serving	300	6.2	0.3	6.0	45.2	6.5	1.7	36.5	0.1	0.0	195.2	96.1
Ugali, omena and cabbage	4 – 6 yrs	230	5.5	0.3	6.7	97.7	12.2	2.4	60.0	51.6	18.8	24.8	15.5
	7 – 10 yrs	340	5.6	0.3	4.7	61.2	5.8	3.1	56.4	65.1	18.1	34.7	16.9
	11 - 15 yrs	400	8.5	0.4	7.2	72.9	5.6	3.6	51.4	76	17.1	40.6	16.2
	Average serving	323	6.5	0.3	5.5	77.3	7.9	3.0	55.9	64.2	18.0	33.4	16.2
Ugali, beef and kale	4 – 6 yrs	220	5.7	0.3	7.0	85.0	10.6	2.1	52.5	52.4	24.1	24.2	15.1
	7 – 10 yrs	330	7.7	0.4	6.5	44.4	4.2	3.1	56.4	66.3	21.1	44.4	21.7
	11 – 15 yrs	385	9	0.5	7.6	51.8	4.0	3.6	51.4	76.2	17.1	51.8	20.7
	Average serving	312	7.5	0.4	6.4	60.4	6.3	2.9	53.4	72.8	20.8	40.1	19.2

EER = Average estimated energy requirement for active physical activity level

AI = Adequate intake

EAR = Estimated average requirement

AMDR = Acceptable macronutrient distribution range

[1] Micronutrient contents from the maize meal porridge is included

[2] Micronutrient contents from fruits (mango, banana, orange) is included

2.8   Structure of School Feeding Programme

The school feeding meal plan was structured around the three school terms as per the education system in Kenya. The first school term starts early January and ends in the first week of April, followed by a three-week holiday during the month of April. The second school term starts in the first week of May and lasts until the last week of July, followed by a one-month school holiday in August. The third school term starts in the first week of September up until the last week of November, followed by a six-week holiday in from December to early January.

The school feeding programme was implemented in the MVP as follows: the first menu (*githeri*) was eaten on three days of the week during all three school terms described above. One fruit (orange, mango or ripe banana) was served on the same days as the first menu. The three types of fruit, orange, mango or ripe banana were eaten on the *githeri* days depending on which fruits were in season and readily available. The nutrient analysis for the fruits were averaged and included in the final nutrient analysis of the menus (Appendix 3). The second menu (*ugali,* beef and kale) was taken on two days of the week during the first and third school terms. The third menu (*ugali, omena* and cabbage) was available during the second term in May, when fishing of the *omena* species is allowed on Lake Victoria. This menu was provided for two days of the week (TABLE 2.4). Figure 2.6 contains pictures of the school feeding menus taken in the field. Children in the control group did not receive any school lunch at baseline. After 6 months the Millennium Village Project was scaled up and menu 1 (*githeri*) was introduced in the control area. No fruit were given to the children in this group during the study period, and they did not receive menus 2 or 3 because they were the control group for the study and the overall upgrading of the Millennium Villages Project to other clusters nearing Bar-Sauri village did not include a research component. However, the control children received *githeri* (maize and beans) for lunch and those in pre-school received maize meal porridge, just like the MVP children. School feeding programmes were not continued during the school holidays.

TABLE 2.4: School feeding menus and the consumption schedule in the MVP

School term	Menu type	Days of the week eaten
First term (January to April)	Menu 1 (*Githeri* and orange/ banana)	Mondays, Wednesdays and Fridays
	Menu 2 (*Ugali,* beef and kale)	Tuesdays and Thursdays
Second term (May to July)	Menu 1 (*Githeri* and mango)	Mondays, Wednesdays and Fridays
	Menu 3 (*Ugali*, *omena* and cabbage)	Tuesdays and Thursdays
Third term (September to November)	Menu 1 (*Githeri* and ripe banana)	Mondays, Wednesdays and Fridays
	Menu 2 (*Ugali,* beef and kale)	Tuesdays and Thursdays

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FIGURE 2.6: Pictures of the school feeding menus taken in the field

Numbers 1 and 2 are *ugali* and *omena*, 3 shows the preparation of vegetables and the school kitchen's commercial cooking equipment, 4 shows *githeri* while 5 shows the fruit varieties.

2.9   Implementation of School Feeding Programme

The school feeding program was implemented through the Millennium Villages Project education committee as described in the sections that follow.

2.9.1   Organisational structure of villages

The MVP had eight village committees formed in line with the MDGs to implement the MVP's objectives. The village committees coordinated the work of the MVP. In particular, the education committee, with representatives of the school board, teachers and parents was in charge of all education programmes in the village, including the school feeding programme. The other duties of this committee were to undertake development programmes in the schools, such as the construction of kitchens and the allocation of bursary funds to pupils. A school feeding subcommittee was created at the beginning of this study and was made up of three members from the education committee, school representatives and the MVP implementing office representative, together with the PI. The main role of this subcommittee was to implement the school feeding programme in the study area. This committee was in charge of food procurement, the distribution of foodstuffs to the schools, and monitoring of food stocks in the schools.

The principal investigator (PI) and the research assistants were in regular consultation with the project implementation office, the education committee and the school feeding subcommittee on the implementation of the school feeding programme. The other village committees included agriculture, roads, energy, health, environment, water and business (Figure 2.7). These committees were also helpful in the implementation of the study, because they were members of the executive committee that approved decisions about the school feeding programme. The schools in the control group were organised in a similar manner when the MVP project was extended to cover the control area in January 2007, although this group did not have a research component and therefore the school feeding was limited to a basic menu of maize and beans (*githeri*).
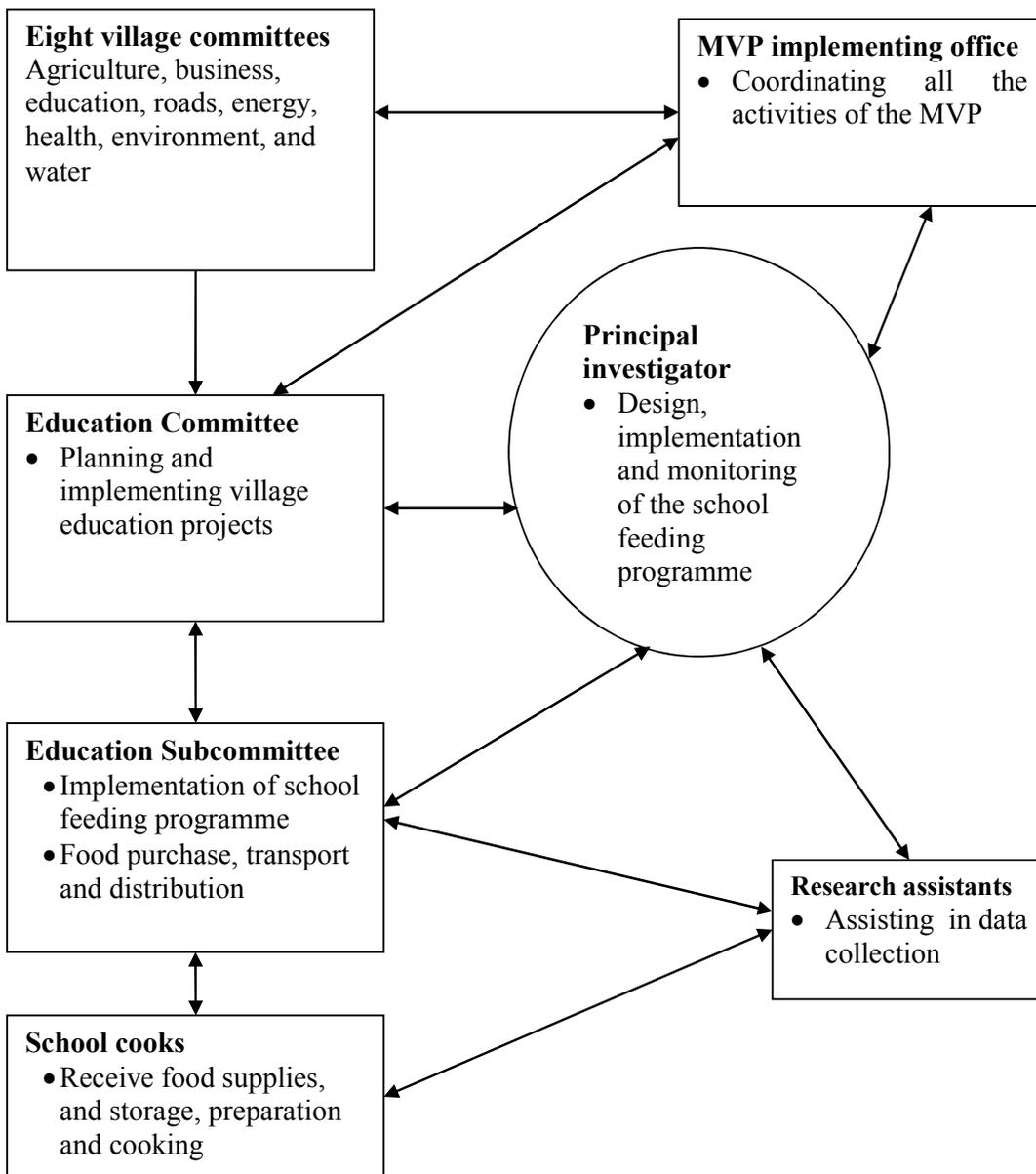
FIGURE 2.7: Village organisational structure and roles of committees

2.9.2   Role of school feeding implementation team

The school meals programme was funded partially by the Earth Institute's village project and by the community. It cost US$32 per child per year to provide the meals. The project contributed 60% while the community contributed 40%. Each parent in the school contributed 10% of their yield of maize and beans per harvest towards the feeding programme. TABLE 2.5 provides a summary of the contributions and the roles of the community and the MVP. The school feeding subcommittee was responsible for purchasing, transporting and distributing the raw foods to each of the schools. The teacher in charge of the school feeding programme in each school received the raw food deliveries and ensured proper storage of the food items, as well as supervised the preparation,

cooking and distribution of meals to the children. The cooks received the food items from the teacher in charge of the school feeding programme, and prepared and served the meals to all the children.

The PI and the two research assistants visited all the schools included in the study before the start of the study and held meetings with the head teachers and subsequently with the school management committees to brief them on the study design and the implementation of the programme.

The PI and the research assistants held a nutrition awareness meeting at Bar-Sauri Primary School, where the menus were presented to the pupils and their opinions were sought on the suggested menus and the suggested meal preparation methods. The children were asked to vote by raising their hands if they preferred a certain menu item when the item was mentioned. More children raised their hands for *githeri*, followed by *ugali* with beef and then *ugali* with *omena*.

TABLE 2.5: Role of the MVP and the community

MV Project office	Community/school
• Provision of subsidised crop seeds and fertilisers to communities	• Grow food and bring back 10% of the harvest for school meals
• Construction of improved cook stoves and pans	• Provision of firewood and labour for cooking
• Construction of water tanks	• Fetching additional water
• Provision of vegetable seeds	• Provision of green leafy vegetables
• Contribution to kitchen construction	• Contribution to kitchen construction
• Monitoring	• Implementation and monitoring

Source: Siriri et al.[288]

2.10   Monitoring the School Feeding Programme

During meal times, food distribution in the schools was done in the order of classes to ensure that the children were served with the appropriate portion sizes for their age. The children queued per class to receive their food and sat together in their classroom groups. Those who had been sampled for this study were observed by the teacher in charge of the school feeding programme to ensure that they finished all the served food. At the start of the study the school feeding subcommittee, school cooks and research assistants were trained by the investigator in the preparation methods and portion size allocation for the children.

Each of the two research assistants working with the investigator visited three schools once a week to observe the food preparation, service and consumption. They observed whether the food

preparation methods were being followed as per the cooks' training, whether the children were served in order of their classes and whether they finished the portions served to them. The teacher in charge of the school feeding programme supervised the day-to-day preparation and serving of the food to the children.

2.11   Logistics of Data Collection

The PI was in charge of all data collection activities.

2.11.1 The role of the principal investigator

The PI, a public health nutritionist, was in charge of the overall implementation of the research activities. She supervised, directed and coordinated all the activities, and also carried out the following:

- Developed the study protocol and all the data collection tools
- Developed the menus of the school feeding intervention
- Supervised the implementation of the research activities and the data collection procedures
- Trained the research support team (research assistants, school cooks, teachers in charge of school feeding and the school feeding subcommittee). Areas covered in the training are listed in the next section, on the training of the support team
- Administered the socio-demographic and dietary intake questionnaires together with the research assistants
- Obtained the anthropometric measurements together with the research assistants
- Checked the data for completeness
- Observed all the procedures of the blood sampling to make sure that the recommended procedures were followed
- Ensured safe transportation of the blood samples to the laboratories
- Data entry, data processing and cleaning
- Data analysis

2.11.2 Training of support team

The PI trained the following groups of people who were part of the support team: school cooks, school feeding subcommittee, teachers in charge of the school feeding programme, education committee, and research assistants for data collection. The PI facilitated a one-day training session at baseline, and then another session two weeks later to review the procedures. The group was trained in the following areas:

- Purpose of the study.

- Menu plans – a detailed description of menus 1, 2 and 3 was provided and demonstrations were held on the recipe and cooking methods. This training was held at Bar-Sauri Primary School, where the PI, together with the trainee team, prepared each of the three menus. An agreed upon preparation procedure was followed for each of the menus.

- Determination of portion size – a kitchen scale (Salter: Salter House Wares Ltd, England, Model 051) was used by the investigator to measure each of the portion sizes described in Table 2.2 and served in different plates. This was to demonstrate the amounts to be served per child depending on age and school grade. Each of the schools was provided with a common kitchen serving spoon to be used in food distribution, and it was agreed during the training on how food would be served per child based on the portion sizes.

- The responsibilities of each member of the support team were explained during the training, as below:

  - Six school cooks (two from each school) – preparation of school feeding menu and food distribution to the children. The cooks were also to keep an inventory record of the movement of food items in the stores.

  - Four school feeding subcommittee members – purchase of foodstuffs from the local market, distribution of the foodstuffs to the schools, monitoring of food stocks, monitoring of the implementation process, and reporting to the PI, the education committee and the MVP project implementation office on the progress of school feeding programme.

  - Three teachers in charge of the school feeding programme – each school provided one teacher to be in charge of the feeding programme in the school. Their role was to monitor the food stocks together with the school feeding subcommittee, issue food items to the cooks, monitor the distribution of cooked food to the children during meal times, and observe the children during meal times for portion size clearance.

  - Three members of the education committee – they were in charge of the education sector in the MVP and coordinated the building of school kitchens, the acquisition of cooking fuel, monitored the contribution of maize and beans by the parents and linked the school feeding programme to the executive committee of the village, as illustrated in Figure 2.7.

  - Two research assistants with high school education and a good command of English, Kiswahili and the local language (*Dholuo*) were recruited for the study. They had worked with the MVP in a census survey and had experience in field survey procedures. They were from Yala Division and knew the villages well. The research assistants worked in the field together with the PI in the following data collection procedures:
    - Study design and sampling

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- Self-introduction to the children, teachers and parents

- Filling in the consent/assent documents

- Interview techniques and the booking of appointments

- Date of birth identification methods, as explained in the data collection section

- Dietary intake methods (food frequency questionnaires)

- Labelling of blood and serum samples during blood collection

- Anthropometric measurements; this involved an explanation of the procedures and a demonstration of the measurements and recording techniques. A standardisation exercise was carried out at the Bar-Sauri Health Clinic. One research assistant measured 10 children who had been measured by the PI without the assistants knowing the results of the measurement. The second research assistant measured the same children and recorded the measurements. The PI observed the measuring techniques of the research assistants in terms of positioning of the equipment, the child's clothes, the child's attitude, the child's position, reading time, reading angle and the recorded reading. The PI discussed the outcome of this exercise with each research assistant and compared the reading with that of the PI. Where there were disparities with the PI's measurement, the research assistant was asked to identify reasons that may have caused the disparity in their own measurements and then these reasons were discussed with the PI. The process was repeated until the two research assistants had a difference of less than one unit measurement from the PI for all the anthropometry measurements. The PI is well trained in anthropometry and had conducted independent field surveys prior to this study.

2.11.3 The role of research assistants

The research assistants worked together with the PI in the following data collection procedures:

- Obtaining of consent from participants and the parents

- Administering the food frequency questionnaires

- Taking anthropometric measurements

- Observing the food preparation procedures and serving, and the consumption of the school lunch by the children

- Labelling the tubes for blood sampling

Training in interviewing techniques and the collection of dietary intake data was conducted at the MVP Bar-Sauri Health Clinic on adults who attended the clinic for treatment. The PI observed the practical interviews and recorded the responses. This data was compared with the responses recorded by the research assistants and a discussion was held between the PI and each of the

research assistants on the corrections needed to improve the interview techniques. This was repeated until the research assistants were competent in interviewing skills.

2.12   Data Collection Methods

Data was collected for a period of 30 months and involved various types of data namely; socio-demographic, food intake, anthropometry and biochemical measurements

2.12.1 Types of data collected

Household socio-demographic and food frequency questionnaire data was collected during interviews with the parents/guardians of the children at their households at baseline. The research assistants traced the physical location of the home of each of the sampled subjects, following directions given by the children, parents and teachers. Anthropometric measurements and blood sampling were done at the schools during school breaks. Blood samples were collected three times, at baseline, at 6 months and at 24 months. Due to financial constraints, blood sampling was not conducted in the 30th month of the study.

TABLE 2.6 summarises the frequency and types of data collected.

2.12.2 Socio-demographic data

The socio-demographic data collected included household income, household headship, employment of the household head, mother's marital status, mother's education as well as mother's employment (Appendix 4). The age of the child was recorded as "date of birth". The date of birth was recorded from the "road to health" card provided by the mother/caregiver/household head where available. Where the card was not available, the mother/caregiver was asked to recall the date of birth of the child. In cases where neither of the above was applicable, the date of birth was estimated using a calendar of events and probing questions. The calendar of events adopted for this study included the long rains from mid-March to April, the cold months of June and July, the dry season in August and September, and the short rains in October and November. The school calendar (described in section 2.7 above) was also used as a calendar of events.

TABLE 2.6: Measurements at baseline and throughout the study

Type of data	Frequency	Interval
Household socio-demographic status	1	Baseline
Dietary intake (FFQ)	1	Baseline
Anthropometry measurements		
Height	6	Baseline, 6, 12, 18, 24 and 30 months
Weight	6	Baseline, 6, 12, 18, 24 and 30 months
Arm circumference	6	Baseline, 6, 12, 18, 24 and 30 months
Triceps	6	Baseline, 6, 12, 18, 24 and 30 months
Subscapular		Baseline, 6, 12, 18, 24 and 30 months
Blood sampling		
Haemoglobin	3	Baseline, 6 and 24 months
CRP	3	Baseline, 6 and 24 months
Serum ferritin	3	Baseline, 6 and 24 months
Serum retinol	3	Baseline, 6 and 24 months

2.12.3 Anthropometry

Measurements of weight, height, mid-upper arm circumference, triceps and subscapular skinfold thickness were obtained using standardised techniques.[289] Measurements were taken by the research assistants and the PI at the school during school breaks before the children took lunch. The sampled children were asked to gather in a separate room, where measurements were taken for each individual. All the anthropometry measurements were taken in triplicate and an average was obtained. The PI checked all the anthropometric measurements at the end of each measurement week for completeness. The PI checked the equipment at the start of each week during the data collection period.

Height

Height was taken with a portable stadiometer (Stanley 04-116, Stanley-Mabo Ltd., Poissy, France), which is an L-shaped device to which a loaded coiled tape measure is attached. The portable stadiometer was mounted vertically on a classroom wall with firm cellotape, as shown in the photographs in Figure 2.8. The subject was asked to remove his or her shoes and to stand erect directly below the point of attachment of the tape, looking ahead with feet together, knees straight and heels, buttocks and shoulder blades in contact with the vertical surface of the wall. The movable head bar of the microtoise was lowered to touch the crown of the head. The height measurement

was done by two people. One of the measurers read the height measurement out loud and the second measurer recorded it to the nearest 0.1 cm.

Weight

Weight was taken with a Tanita portable digital bathroom scale (model 1623, Tanita Corporation of America, Inc.) to the nearest 0.1 kilogrammes. Weight was taken with minimum clothing and without shoes, with the child standing on the scale in an upright position.

Mid-upper arm circumference (MUAC)

MUAC was taken with an insertion tape (Similac advantage insertion tape), with the subject standing erect and sideways to the measurer with the head straight and looking ahead. The midpoint of the upper arm was identified between the acromion process on the shoulder blade and the tip of olecranon process of the ulna. The mid-point was determined with a non-stretch fibreglass tape measure. The circumference was measured on an extended left arm hanging loosely with the palm facing inwards, and recorded to the nearest millimetre.

Measurement of skinfold thickness

Subjects were asked to remove their upper clothing for the purpose of measuring the skinfold thickness. Measurements were taken with a Lange (Beta Technology) skinfold callipers to the nearest 0.5 mm. The triceps skinfold measurement was taken at the mid-point of the upper arm, with the arm hanging freely and the palm facing inwards. The mid-point of the upper arm was identified between the acromion process on the shoulder blade and the tip of olecranon process of the ulna. The mid-point was determined with a non-stretch fibreglass tape measure. The subscapular skinfold measurement was taken at the back of the lower angle of the scapula (bottom point of shoulder blade).[289] This was identified by asking the subject to reach behind his or her back with the right arm. The pictures in Figure 2.8 are field photos taken during data collection of height, weight, MUAC, triceps and subscapular skinfold measurements.
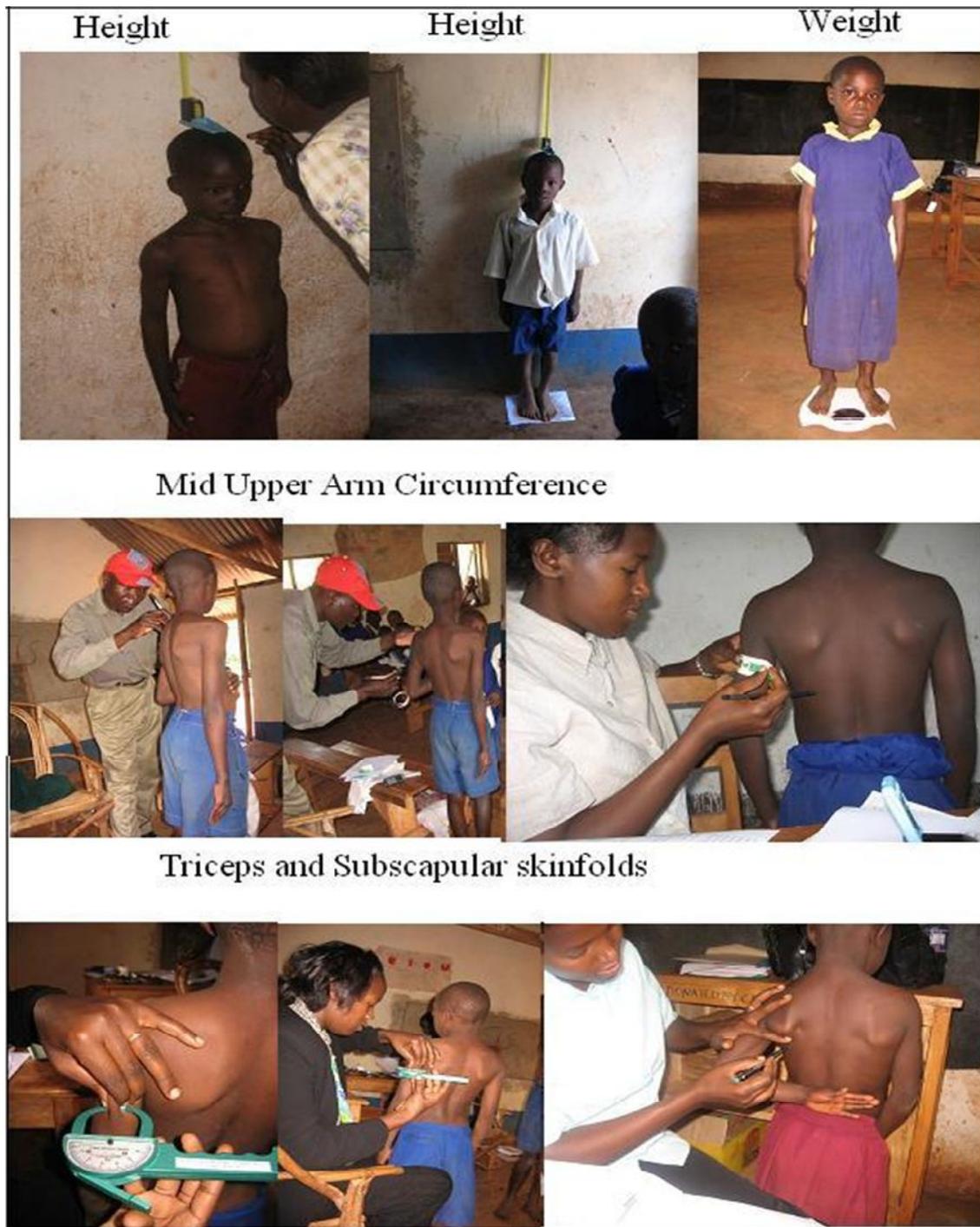
FIGURE 2.8: Photos taken in the field during the collection of anthropometry data

Pictures taken during data collection showing the anthropometric measurement procedures

2.12.4 Dietary data

Food intake data was collected with a quantitative food frequency questionnaire at baseline. A list of 106 foods consumed and available locally in the households was compiled, based on the market survey and the village committee meetings described earlier. Food items were listed in nine categories, as starches, legumes, meat, vegetables, nuts and seeds, fruit, beverages, fats and oils, and

sugars. Each food item was coded. A quantitative food frequency questionnaire (QFFQ) was designed, based on the South African National Food Consumption Survey,[290] and it was modified and validated to fit the purpose of this study. No such tool was available in Kenya at the time of the study. The respondent in the dietary survey was the mother or immediate caregiver of the child in the absence of the mother.

Quantitative food frequency questionnaire

The QFFQ was used to obtain information on the usual eating patterns and intake over the previous six months before the start of the study (Appendix 4). The modifications to the QFFQ included local food lists and incorporated appropriate portion sizes in order to estimate the individual's nutrient intake.[291]

The foods in the QFFQ were arranged according to the nine groups, starting with the commonly consumed foods at the top of the list. The QFFQ was designed to collect data on the type of food eaten, the quantity in household measures, the amount usually eaten and the frequency of intake. The frequency of intake was recorded either per day, per week, per month or never. The interviewer circled the code for each food item consumed and filled in the quantity consumed in household measures, and the frequency of consumption.

The modified food frequency questionnaire was piloted among children in Yala Primary School, which was not part of the study sample but fell within the same division of the study area. The pilot was done to verify if there were any food items that had been left out of the food list, as well as to test the interview skills of the interviewers. No further validity tests were carried out on the QFFQ, as content validity had been verified in the South African model of the QFFQ from which the one for this study was developed. Any new food items that emerged were added to the dietary data collection tools from the pre-test. The new food items included locally available varieties of vegetables, such as nightshade (*osuga*), spider-plant, (*mitoo*) and *crotaralia* species (*murenda*). The findings of the pilot were also used to decide on the household measures and the portion sizes used for the study.

The data research team was equipped with commonly used household measures obtained from the local market, which consisted of two cup sizes, a serving spoon, teaspoons and tablespoons, a plate and a bowl. They also used dry food items (maize, beans and rice) and modelling clay (porcelain) to estimate food intake. The volume of drinks was estimated by asking the respondent to pour an amount of water equivalent to the drink the child had taken into the cups the research team was

carrying. This was then recorded as the amount of drink consumed. Fruit intake was recorded as counts. QFFQ data was collected only at baseline to assess dietary intake before the implementation of the intervention.

Converting the household measures to weight

All the foods used for the quantitative food frequency questionnaire were bought from the local markets and cooked by the research team on a specific day. The cooked foods were served in commonly used household measures and weighed (Salter: Salter House Wares Ltd, England: Model 051).

2.12.5 Biochemical data

Blood sampling

Blood sampling was done three times during the study period. Non-fasting samples were drawn at baseline, at 6 months and at 27 months. Five millilitres of blood was drawn by a trained laboratory technologist with diploma training in Laboratory Technology from the Kenya Medical Training College (KMTC). The process was observed by the PI to make sure that the blood collection protocol was followed throughout the blood sampling and handling.

The sample was drawn from the antecubital fossa and transferred into vacutainers, one with ethylenediaminetetraacetic acid (EDTA) as anticoagulant and antioxidant and the second with no additives. Both tubes had already been labelled with the subject's identity number, time of sample collection and date. Samples were immediately wrapped in aluminium foil to protect them from light and stored in a cooler box with ice packs until transportation to the Centers for Disease Control (CDC) laboratory in Kisumu. The CDC laboratory was about 45 kilometres from the study area and it was ensured that the samples reached the laboratory within three to four hours after collection. Field pictures taken during blood sampling, transportation and laboratory procedures for full haemogram and for centrifuge are shown in Figure. 2.9.

FIGURE 2.9: Blood sampling field pictures

Pictures taken during data collection showing the drawing of blood (1, 2 and 3), the labelling of the tubes (4) and laboratory work (5 to 8)

Laboratory procedures and processing of blood sample

Upon arrival at the laboratory, a full blood cell count was done with a Coulter AcT 10 (Beckman Coulter Inc.). The second blood sample aliquot in the plain vacutainers was centrifuged for the

separation of serum at 3 000 RPM for 15 minutes (Eppendorf Centrifuge 5415D) and serum stored in cravial boxes (Figure 2.9, photo 8) at -70°C until analysed for retinol, ferritin and C-reactive protein (CRP). Storage tracking sheets (Appendix 5) containing the subjects' identification numbers for the study were prepared for ease in tracing samples.

Randomly selected sub samples were transported by courier in dry ice to the micronutrient laboratory at the Division of Human Nutrition at Stellenbosch University, Cape Town, South Africa, where the serum ferritin, CRP and retinol analyses were done. A serum sample transportation permit was obtained from the Ministry of Medical Services, Kenya (Appendix 6).

Serum ferritin and C-reactive protein (CRP) concentrations were analysed by nephelometry (BN ProSpec, Siemens Healthcare Diagnostics Inc, Newark, DE 19714 USA). The serum ferritin analysis was based on the principle that polystyrene particles coated with monoclonal antibodies specific to human ferritin are aggregated when mixed with a sample containing ferritin. These aggregates scatter a beam of light passed through the sample. The intensity of the scattered light is proportional to the concentration of the relevant protein in the sample. The result is evaluated by comparison with a standard of known concentration. The coefficients of variation (CV) of intra-assay precision ranged from 1.0 to 4.6%, while the CV of inter-assay precision ranged from 1.2 to 3.1% and the total CV ranged from 1.6 to 5.1%. The analytical sensitivity of the assay is determined by the lower limit of the reference curve and therefore depends on the concentration of the protein in the protein standard.

The CRP analysis was based on the principle that polystyrene particles coated with monoclonal antibodies specific to human CRP are aggregated when mixed with sample containing CRP. These aggregates scatter a beam of light passed through the sample. The result is evaluated by comparison with a standard of known concentration. A coefficient of variance (CV) of 7.6% was observed from ten replicates of a sample containing 0.41 mg/ml of CRP. The analytical sensitivity of the assay is determined by the lower limit of the reference curve and therefore depends upon the concentration of the protein in the N rheumatology standard SL. A typical limit of detection for CRP is 0.175 mg/ml for measurements performed using a sample dilution of 1:20.

Serum retinol was determined with high-performance liquid chromatography (HPLC).[292] This determination is based on the principle that serum or plasma samples are first deproteinised by precipitation, following which the fat-soluble components are extracted with hexane. After evaporation of the hexane, the residue is dissolved in methanol and aliquots are used for

quantitative determination by HPLC. A coefficient of variance of 1.4% and 4.2% was observed in nine replicates of a sample containing 0.5 µmol/L and 2.41 µmol/L of retinol respectively. The quality control parameters was a limit of detection of 0.22 µmol/L. Reports of the sample analysis are provided in Appendix 7.

2.13   Data Quality Control

Measures were taken to ensure that the data quality was controlled at every step of the research project, starting with training the research assistants, standardising the methods, entering the data, cleaning, and management analysis and reporting.

Training and standardisation

The training of research assistants and the standardisation of the methods was done a week before the actual data collection in each data collection interval.

Anthropometric measurements

Anthropometric equipment was checked and standardised as follows: the scale batteries (alkaline cells) were changed before the start of every data collection session in each term. The bathroom scale was calibrated. The bathroom scale was calibrated using a 2 kilogram commercially packaged of sugar. This package from a specific manufacturer is widely available in retail shops outlets across the country thus the choice. A packet was purchased at the start of each school term for the calibration purpose. The digital scale was set on calibration mode, and the measurement adjusted to the weight of the sugar. The stadiometer for height and the insertion tapes for the MUAC measurements were replaced with new ones once during the study period. The Lange skinfold callipers were sent to the suppliers for servicing once during the study (the service document is provided in Appendix 8). The PI was present during data collection and observed that all the anthropometric procedures were followed. The anthropometry data was checked by the PI at the end of each week. If data was missing, the research team went back to the schools, traced the subjects and repeated the measurements on the first school day of the following week.

Dietary intake

The collection procedures for dietary intake data were standardised by demonstrations of the kitchen equipment used in the field, interview techniques and the reporting of dietary intake.

Data entry

- Data entry was done by the PI (anthropometric data) and two trained data entry clerks at the MVP project offices in Kisumu (dietary, blood and socioeconomic data).
- Entered data was cross checked by the PI.

Blood sampling and analysis

The following quality assurance precautions were taken:

- Protection of samples from light using aluminium foil.

- Storage in cooler box with ice packs for 3 to 4 hours between collection and transportation to the laboratory.

- After centrifuging, tubes were inspected carefully in order to recognise possible haemolysis. Any haemolytic samples were discarded and the blood collection was repeated after explaining the situation to the child and the parent.

- A standard sample was run in the CDC laboratory on the Coulter AcT 10 machine before the study samples were analysed. This was to ensure that the machine was in good working condition before the field samples were analysed for full haemograms.

- Serum samples were stored at -70°C until transportation to the micronutrient analysis laboratory.

- Samples were transported with dry ice packs from the CDC laboratories in Kisumu, Kenya to the micronutrient laboratory at Stellenbosch University, South Africa. The sample shipment documents are included in Appendix 9.

- Samples were frozen in the micronutrient laboratory at -20°C for two months during the analysis period.

- Minimum lighting was allowed in the laboratory during the sample analysis to preserve vitamin A.

2.14   Data Analysis

Data analysis was conducted for all the various types of data with the support and assistance of the Centre for Statistical Consultation, Stellenbosch University.

2.14.1 Nutrient intake

The nutrient content per 100 g of each of the listed foods was compiled from the Kenyan food composition tables,[285] the FAO food composition tables for Africa[286] and the USDA food search.[287] This nutrient content list was used by the PI to create a food composition database to determine nutrient intake. There was no Kenyan food composition database at the time of this study. The nutrients included for analysis were total energy, carbohydrates, total fats, total proteins, vitamin A, vitamin C, folate, zinc, calcium, selenium and iron.

The usual nutrient intake was calculated with the formula:

Daily intake = (amount consumed x frequency per day x number of days per week x number of weeks per month)/30.[290] The adequacy of the nutrient intake was calculated on the basis of the dietary reference intakes (DRIs) developed by the Food and Nutrition Board of the National Academies' Institute of Medicine (IOM).[32] Energy intake was estimated based on the Estimated Energy Requirements (TABLE 2.7). The estimated average requirement (EAR) was used to assess nutrient adequacy for those nutrients with an established EAR (protein, carbohydrates, vitamin A, vitamin C, folic acid, zinc, iron and selenium). Calcium intake was assessed with the adequate intake (AI) because the EAR for calcium is not available, while total fat was assessed using the acceptable macronutrient distribution range (AMDR;TABLE 2.8).

TABLE 2.7: Estimated energy requirement

	Males		Females	
Age (years)	kcals/day	kJ/day	kcals/day	kJ/day
6	1 742	7289	1642	6870
7	1 840	7699	1719	7192
8	1 931	8079	1810	7573
9	2 043	8548	1890	7908
10	2 149	8991	1972	8251
11	2 279	9535	2071	8665
12	2 428	10159	2183	9134
13	2 618	10954	2281	9544

Source: Institute of Medicine[37]

TABLE 2.8: Dietary reference intake for nutrients

	Carbohydrates (digestible (g/day)[1]	Total protein (g /kg/ day)[1]	Total fat (g)[2]	Vitamin A (µg/ day)[1]	Folic acid (µg DFE/ day)[1]	Vitamin C (mg/ day)[1]	Zinc (mg/ day)[1]	Iron (mg/ day)[1]	Calcium (mg/ day)[3]	Selenium (mg/ day)[1]
Males										
6 to 8 years	100	0.76	25-35	275	160	22	4	4.1	800	23
9 to 13 years	100	0.76	25-35	445	250	39	7	5.9	1300	35
Females										
6 to 8 years	100	0.76	25-35	275	160	22	4	4.1	800	23
9 to 13 years	100	0.76	25-35	420	250	39	7	5.7	1300	35

Source: Institute of Medicine[37]; DFE: dietary folate equivalent; [1] Estimated average requirement; [2] Acceptable macronutrient distribution range; [3] Adequate intake

The probability approach method described by Gibson[289] was used to estimate the prevalence of inadequate nutrient intake. The approach combines the distribution of requirement and individual usual nutrient intakes for the group to estimate the proportion of individuals at risk of inadequate

intakes. This approach predicts the number of individuals within a group with nutrient intakes below their requirements, thus providing an estimate of the population at risk.

The cut-point probability was used to estimate the relative probability that the nutrient did not meet the individual's actual requirements. This was done by classifying each nutrient intake into six classes defined by the EAR and the associated standard deviation limits as provided by Gibson.[289] TABLE 2.9 shows the six classes of nutrient classification and the standard deviation limits. The number of individuals with intakes of a given nutrient within each class was then determined and this number was multiplied by the appropriate probability for each class to give the number of individuals who were likely to have intakes below their own requirements. The sum of these numbers gives the number of individuals in the population group who are at risk of inadequate intakes for each particular nutrient. This was then expressed as a percentage of the total population group to give a probability estimate for the population group as a whole.

TABLE 2.9: Six classes of observed intakes expressed as proportions of the Estimated Average Requirement[1].

	Class 1	Class 2	Class 3	Class 4	Class 5	Class 6
A. Individual's intake in terms of the distribution of requirements	<-2SD[2]	-2SD to -1SD	-1SD to mean	Mean to +1SD	+1SD to +2SD	>+2SD
B. Probability that individual intake doesn't meet requirement	1.0	0.93	0.69	0.31	0.07	0.0

Source: Beaton, American Journal of Clinical Nutrition, 1985 page 155-164; in Gibson[289]

[1]Assumptions of the model is that the distribution requirement for most nutrients is symmetrical and with a coefficient of variation at 10% or 15%.

[2]SD: Standard Deviation

2.14.2 Anthropometry

Anthropometric data were analysed with the WHO Anthroplus,[293] which calculates height-for-age, weight-for-age and body mass index (BMI)-for-age z-scores for children aged 5 to 19 years, based on the WHO 2007 reference standards. This reference contains standards of height-for-age z-score for children up to 120 cm tall, and weight-for-age z-scores for children aged up to 10 years.[294] The WHO classification of malnutrition was used to determine the prevalence of stunting, underweight and wasting.[295]

A weight-for-age, height-for-age and BMI-for-age z-score less than -2 standard deviation was classified as underweight, stunted or wasted respectively. Further classification was done to identify severe, mild and moderate undernutrition, based on the WHO cut-off points (see TABLE 2.10). All

children with mild, moderate and severe undernutrition were considered to have inadequate nutritional status. A height-for-age z-score ≤ -1 SD was used as cut-off for linear growth deficit. Overweight was determined as +1 SD BMI-for-age z-score ≤ +2 SD, while obesity was determined as > +2 SD BMI-for-age z-score. This standard deviation in the WHO reference is equivalent to the 85[th] percentile and coincides at 19 years with the adult BMI of 25 kg/m^2, the cut-off for overweight in adults.[296] Children with mild, moderate and severe undernutrition were considered as having inadequate nutritional status.

The WHO classification of the public health significance of stunting for children below five years of age was used to evaluate the public health significance of stunting among the school children, since there were no corresponding definitions for older children (TABLE 2.11). Growth velocity was calculated as $(x1–x2)/(t1–t2)$,[297] where x = anthropometric variable and t = time of measurement. The calculated growth velocities were used to assess and compare child growth in the intervention and control groups over the period of study.

TABLE 2.10: WHO classification of malnutrition based on z-scores

Nutritional status	Z-score values
Normal	> -1
Mild	< -1 to -2 z-score
Moderate malnutrition	< -2 to -3 z-score
Severe malnutrition	< -3 z-score

Source: World Health Organisation[75]

TABLE 2.11: Classification for assessing severity of malnutrition by prevalence ranges for children under five years old

Indicator	Low	Medium	High	Very high
Stunting	< 20	20-29	30-39	≥ 40
Underweight	< 10	10-19	20-29	≥ 30
Wasting	< 50	5-9	10-14	≥ 15

Source: World Health Organisation[75]

2.14.3 Body composition

The gender-specific equations of Deurenberg et al.,[298] which are validated for use in African children, were used to estimate body composition from skinfold thickness measurements.

Percentage of body fat was estimated from the Slaughter equation as:

Boys: 1.21 (triceps + subscapular) – 0.008 (triceps + subscapular)^2 – 3.2

Girls: 1.33 (triceps + subscapular) – 0.013 (triceps + subscapular)^2 – 2.5

Fat mass in kilograms was calculated as:

Boys: Weight * {1.21 (triceps + subscapular)} – 0.008 (triceps + subscapular)^2 – 3.2/100

Girls: Weight * {1.33 (triceps + subscapular)} – 0.013 (triceps + subscapular)^2 – 2.5/100

Fat-free mass in kilograms was calculated as: weight – fat mass; mid-upper arm muscle area was calculated as suggested by Gibson[289] $[C-(\pi \times TSK)]^2/4\ \pi$; where C = mid-upper arm circumference and TSK = triceps skinfold thickness.

2.14.4 Biochemical analysis

Classification of anaemia and iron status

Haemoglobin concentration (g/dl) was used to assess the prevalence of anaemia based on WHO criteria[299] (TABLE 2.12) and adjusted for altitude. According to the WHO criteria, an Hb concentration of less than 11.5 g/dl is classified as anaemia among children aged 5 to 11 years, while the cut-off is less than 12 g/dl among children aged 12 to 14 years. The cut-offs were adjusted for altitude by subtracting 0.5 g/dl for 1,500 m above sea level.[299] Children with severe anaemia (< 7.5 g/dl) were referred to the Bar-Sauri health facility for further investigation of malaria and appropriate treatment.

Serum ferritin concentration was used to estimate the extent of depleted body iron stores at a cut-off for serum ferritin of less than 15 ug/l for children aged five and older (TABLE 2.13).[299] A mean cell volume less than 82 femtolitres (fl) with a low haemoglobin was used to diagnose microcytic anaemia.[299] A CRP level ≥ 10 mg/l was an indication of infection or inflammation (TABLE 2.13). Bivariate analysis of anaemia was done by combining those with low haemoglobin, low MCV, low haemoglobin and low ferritin, while trivariate analysis was done by combining those with low Hb, low MCV and low ferritin.

TABLE 2.12: Diagnosis of anaemia based on haemoglobin levels

	Altitude	Non-anaemia	Anaemia	Mild	Moderate	Severe
				Anaemia (further classification)		
	Children 5 – 11 years					
Haemoglobin	Sea level	≥ 11.5	< 11.4	11.0–11.4	8.0–10.9	< 8.0
(g/dl)	Adjusted[1]	≥ 11.0	< 10.9	10.5–10.9	7.5–10.4	< 7.5
	Children 12 – 14 years					
Haemoglobin	Sea level	≥ 12.0	< 11.9	11.0–11.9	8.0–10.9	< 8.0
(g/dl)	Adjusted[1]	≥ 11.5	< 11.4	10.4–11.4	7.5–10.3	< 7.5
		Degree of public health significance				
		None		Mild	Moderate	Severe
Prevalence of anaemia (percent of the population)		≤ 4.9		5.0–19.9	20.0–39.9	≥ 40

Source: World Health Organisation[299]
[1] haemoglobin adjusted for attitude by subtraction of 0.5 g/dl for 1 500 m above sea level

TABLE 2.13: Relative extent of iron stores based on serum ferritin concentration

	Normal iron stores	Depleted body iron stores[1]	Depleted body iron stores in the presence of an infection	Risk of iron overload
Serum ferritin (ug/l)	≥ 15	< 15	< 30	> 200 (males) > 150 (females)
	No indication of infection/inflammation	Indication of infection/inflammation		
CRP (mg/l)	< 10	≥ 10		

Source: World Health Organisation[300]; [1] Serum iron, total iron-binding capacity and percentage saturation were not determined. A clear distinction between iron deficiency and iron depletion cannot therefore be made

Vitamin A status

Vitamin A status was based on serum retinol concentration. Children with a serum retinol concentration < 0.35 μmol/L were classified as vitamin A deficient, those with <0.70 μmol/L were classified as having inadequate vitamin A status, while children with a serum retinol concentration ≥ 0.70 μmol/L were classified as having adequate vitamin A status[301] (TABLE 2.14). The degree of significance of subclinical vitamin A deficiency as a public health problem was determined by the prevalence of low serum retinol, i.e. < 0.70μmols/L, as mild (2 to 9%), moderate (10 to 19%) or severe (20% of a population).

TABLE 2.14: Criteria for classification of vitamin A status

Serum retinol (ug/dl)[a]	Vitamin A status		
<0.35μmol/L	Vitamin A deficiency		
0.35 -0.70μmol/L	Marginal vitamin A status		
< 0.70μmol/L	Inadequate vitamin A status		
≥ 0.70μmol/L	Normal vitamin A status		
	Degree of public health problem		
Prevalence of low serum retinol	Mild	moderate	severe
<0.70μmol/L	2–9%	10–19%	≥ 20%

[a] WHO[301]

2.15 Statistical Analysis

The statistical analysis was done with Statistica version 11.[302] The normality of data was checked with normal probability plots of raw residuals from the particular ANOVA or repeated measures ANOVA that was considered for the analysis of the continuous data. Means, standard deviations and 95% confidence intervals were used to describe the data, while repeat measures ANOVA were used to check for within-group differences.

A one-way ANOVA was used to check for differences between groups, and the Bonferroni test was used to test for significance within and between groups for normally distributed data at various intervals of data collection. General linear model (GLM) repeated measures analysis was used to compare means over different observation intervals. Where the data was not normally distributed, the bootstrap test was used to check for differences between and within groups. Comparisons for categorical data were done with Pearson's chi-squared test. Spearman rank order correlation was used to analyse variables for correlations, while binary logistic multivariate regression models were utilised to examine relationships between different variables. A P value < 0.05 was considered as significant.

2.16 Ethical Approval

2.16.1 Ethical review committees

Ethics approval was obtained from the Committee on Human Research of Stellenbosch University (N06/01/002; 12/04/2006), the Columbia University Institutional Research Board (AAAC5773; 10/07/2006) and Moi University's Institutional Research and Ethics Committee (N000199;04/08/2006) (Appendix 10). The research permits were renewed annually upon submission of progress reports. Permission was sought from the Ministry of Medical Services (Kenya) for sample transportation from the CDC laboratory to the micronutrient laboratory at the Division of Human Nutrition, Stellenbosch University (Appendix 6).

2.16.2 Informed consent

The procedures of the study were explained to the children and their parents before they were invited to participate in the study. The parents who accepted signed the consent in the presence of witnesses, while the children wrote their names on the consent form. The consent was translated into Swahili and Dholuo (Appendix 11).

2.16.3 Participant confidentiality

Information that could be used to identify the children was omitted from the study-related materials to ensure confidentiality. Code numbers only known to the PI were used in the data processing and analysis. Participation in the study was voluntary and the participants were free to withdraw at any point of the study without being penalised.

2.16.4 Ethical issues regarding treatment

All children with haemoglobin levels of 7 g/dl and below were referred to the Bar-Sauri Health Clinic, where they were treated for anaemia, investigated for malaria and treated if found to have malaria. This referral was done within 10 days of the haemoglobin measurement. Only 11 children were found to have severe anaemia and were referred to the health facility for treatment. Out of those 11, 5 were treated for malaria and anaemia while the rest were treated for anaemia only. Records on the type of diagnosis and the treatment of the children were kept in the database. There were no children with severe vitamin A deficiency who needed to be referred for further investigation or treatment.

3   CHAPTER THREE: RESULTS

3.1   Enrolment of Subjects in the Study

A total of 240 children who met the inclusion criteria were invited to participate in the study during the recruitment period from May to June 2006. Of the potential 240 candidates, 235 children gave assent and their parents/guardians consented for them to be included in the study. Of the 235 children enrolled, 118 were from the MVP and 117 were from the control group (TABLE 3.1).

TABLE 3.1: Enrolment of subjects in the study

Category	Total (N = 235)	MVP (N = 118)	Control (N = 117)	Chi-square test: p
		N (%)		
Sex				
Male	123 (52.3)	64 (54.2)	59 (50.4)	
Female	112 (47.7)	54 (45.8)	58 (49.6)	0.325
Class				
Nursery	66 (28.1)	41 (34.7)	25 (21.4)	
Class 1	57 (24.3)	29 (24.6)	28 (23.9)	0.07
Class 2	60 (25.5)	31 (26.3)	29 (24.8)	
Class 3	52 (22.1)	17 (14.4)	35 (29.9)	
School				
Bar Sauri		46 (19.6)		
Bar Turo		37 (15.7)		
Nyamninia		35 (14.9)		
Muhanda			40 (17.0)	
Migosi			45 (19.1)	
Kanyuto			32 (13.6)	

[1]P value determined by one-way analysis of variance (ANOVA)

Household socio-demographics, the subject's dietary intake, blood samples and anthropometric data were collected for all the children in the MVP at baseline (June 2006). In the control group, socio-demographic and dietary data were collected for 102 children because 15 parents/guardians were not available for interviewing even after four home visits during the baseline data collection period. Blood samples were completed for the 117 children in this group at baseline (Figure 3.1).

The random sub-sample for serum ferritin and retinol analysis was 100 (N = 50 in the MVP and N = 53 in the control group). However, ferritin results were not available for eight samples in the MVP and 11 samples in the control group because the sample volumes were too small for analysis. In addition, serum retinol results were not available for 6 samples in the control group because the results were below the limit of detection. Therefore serum ferritin and CRP analysis was done for a total of 84 samples (N = 42 in the MVP and N = 42 in the controls), while serum retinol was done for a total of 94 samples (N = 50 in the MVP and N = 44 in controls; see Figure 3.1).

Study population, children aged 6 to 13 years
N = 1211

All eligible

MVP; eligible subjects
N = 627

MVP; sampled N = 118

Control; eligible subjects
N = 583

Control; sampled N = 117

Baseline data collection
- Household sociodemographics (N = 118)
- Dietary intake (N = 118)
- Anthropometry (N = 118)
- Full haemogram (N = 118)
- Serum ferritin and CRP (N = 42); eight samples too little for analysis
- Serum retinol (N = 50)

Baseline data collection
- Household sociodemographics (N = 102); 15 parents/guardians unavailable
- Dietary intake (N = 102) (15 parents/guardians unavailable)
- Anthropometry (N = 105); 12 children unavailable for measurements
- Full haemogram (N = 117)
- Serum ferritin and CRP (N = 42); 11 samples were too little for analysis
- Serum retinol (N = 44); six samples were below the limit of detection of serum

30 months of data collection*

Completeness of data collection
- Household sociodemographics (N = 118); complete
- Dietary intake (N = 118); complete
- Anthropometry and full haemoglobin (N = 118); complete
- Serum ferritin (N = 42); eight samples not analysed at baseline were excluded
- Serum retinol (N = 50); complete

Completeness of data collection
- Household sociodemographics (N = 102); 15 subjects excluded from baseline
- Dietary intake (N = 102); 15 subjects excluded from baseline
- Anthropometry and full haemograms (N = 105); complete
- Serum ferritin (N = 42); 11 samples not analysed at baseline were excluded
- Serum retinol (N = 44); six samples not analysed at baseline were excluded

FIGURE 3.1: Results of recruitment and follow-up

*The last point at which data was collected for blood and serum samples was 24 months

3.2   Sequence of Results

The results are presented in three main sections, namely section A, B and C. Section A contains the baseline findings and is subdivided into three parts. Part 1 gives the baseline findings of the combined group of children from the first level of the demographic, dietary, anthropometry, full haemogram, serum ferritin, CRP and serum retinol analysis. Part 2 contains the bivariate results for the combined group of children, testing associations between the anthropometry, prevalence of anaemia, body iron stores and vitamin A status, and socio-demographic characteristics. Part 3 of section A presents comparisons of the various outcomes in the two separate study groups (MVP and controls), between sexes (male and female) and according to age groups (6 to 8 years and 9 to 13 years).

Section B of this chapter presents results following the intervention. This section is also subdivided into two parts. Part one of this section shows the findings on anthropometric indicators at 6 months compared with baseline for the two study groups. Part 2 of section B presents the findings on the trends in growth, haemoglobin concentration, serum ferritin and serum retinol concentration. Part 3 of section B has findings on the impact of the SFP on nutritional status and body composition based on anthropometric measurements, as well as the impact on anaemia, body iron stores and vitamin A status.

Section C of the chapter presents findings on the initial nutritional status on the responses to the SFP to growth, prevalence of anaemia, body iron stores and vitamin A for all the children and for the groups compared.

3.3   Section A: Baseline Findings

This section provides baseline findings on demographic, dietary, anthropometry, full haemogram, serum ferritin, CRP and Serum retinol analysis.

3.4   Part 1: Results for the Combined Group at Baseline

This part of the section provides baseline findings of the combined group of children from the first level of the demographic, dietary, anthropometry, full haemogram, serum ferritin, CRP and serum retinol analysis

3.4.1   Demographic and socio-economic characteristics for all the children at baseline

The mean age of the children was 7.9 years (2.0 SD; range 6 to 13; TABLE 3.2). About half (51.4%) of the children were male. Almost three quarters (69.5%) came from male-headed

households, and many of the household heads were self-employed (45.5%). The majority of the mothers had primary school level of education (70.5%) and were married (72.7%), and about a quarter did not have any form of employment. Reported total household income was low in this population, with more than half (56%) of the households having a monthly income less than 1,000 Kenyan shillings (KES) (equivalent to about US$12.5).

TABLE 3.2: **Household and child socio-demographic characteristics for all the children at baseline**

Characteristic	Category	Total N = 220
	Range	Mean (SD)
Age in years	6 to 13 years	7.9 (2.0)
		N (%)
Age groups	6 to 8 years	130 (55.3)
	9 to 13 years	105 (44.7)
Sex of child	Males	113 (51.4)
	Females	107 (48.6)
Head of household	Father	153 (69.5)
	Mother	35 (15.9)
	Grandparent or other relatives	32 (14.5)
Employment of head of household	Self-employed	100 (45.5)
	Employed	50 (22.7)
	Unemployed	28 (12.7)
	Deceased	42 (19.1)
Mother's education	Primary school	155 (70.5)
	Secondary school	41 (18.6)
	None	14 (6.4)
	Deceased	10 (4.5)
Mother's marital status	Married	160 (72.7)
	Single	50 (22.7)
	Deceased	10 (4.5)
Mother's employment	Self-employed	142 (64.5)
	Unemployed	55 (25.0)
	Employed	13 (5.9)
	Deceased	10 (4.5)
Total household income/month	Less than KES 1,000	123 (55.9)
	More than KES 1,000	97 (44.1)

3.4.2   Dietary intake for all the children at baseline

The variety of foods consumed by the children was obtained from the food frequency questionnaire at baseline. There were 50 different foods mentioned as having been consumed in the previous 6 months. These foods were grouped into eleven food groups, namely staple starches, vegetables, fruit, meat, fish and poultry, sugars, nuts and seeds, roots and tubers, legumes, milk, fats and oils,

and grains (TABLE 3.3). Consumption of the staple starches group was highest (21.0%), while legumes, milk and grains were consumed the least (4%, 2.6% and 0.5% respectively).

TABLE 3.3: Frequency with which food groups were consumed for all the children at baseline

Food group	Food items	Frequency[1] [N (%)]
Staple starches	green bananas, green maize, *githeri* (maize and bean mixture), maize meal, *ugali* and porridge, rice, chapatti (made from wheat flour)	2 057 (21.0)
Vegetables	kales (*sukuma-wiki*), pumpkin, cowpea leaves, indigenous vegetables, bean leaves, spinach, carrots, cabbage, mushroom, onion and tomatoes	1 882 (19.2)
Fruit	mango, ripe pawpaw, guava, passion fruit, orange, pineapple, lemon, avocado, ripe bananas	1 438 (14.7)
Meat, fish and poultry	beef, lamb, mutton, chicken, fish, liver, egg, offal	1 370 (14.0)
Sugars	sugar, honey, sugar cane	748 (7.6)
Nuts and seeds	ground nuts, sesame seeds	561 (5.7)
Roots and tubers	sweet potatoes, Irish potatoes, arrowroot and cassava	546 (5.3)
Fats and oils	vegetable cooking fat (shortening), margarine, vegetable cooking oil (liquid), ghee, and milk cream used in cooking vegetables	516 (5.3)
Legumes	cowpeas and varieties of beans	389 (4.0)
Milk and milk products	milk	254 (2.6)
Grains	millet, wheat and sorghum	50 (0.5)

[1] Number of times the items in each food group was consumed in the 6 months prior to data collection

3.4.3   Nutrient intake for all the children at baseline

The overall mean daily energy intake was 1 677 (399 SD) [95% CI; 930 to 1 817] kcals/7 021 (1 671 SD) [95% CI; 3 894, 7 607] kJ, mean total protein intake was 40 g, while the mean total fat intake was 19 g (TABLE 3.4). Mean vitamin A intake was 753 (374 SD) [95% CI; 705 to 800] µg, with a non-significant difference between the study groups, gender and age groups. The daily mean folate intake was 180 (89 SD) [95% CI; 169, 192] mg against a daily EAR of 160 mg for the 6- to 8-year-olds, and 250 mg for the 9 to 13-year-olds. The calcium intake of 153 (61 SD) [95% CI; 145,

161] mg among the children was notably lower than the EAR, which is 800 mg for 6- to 8-year-olds and 1,100 mg for the older children (9 to 13 years).

TABLE 3.4:   Nutrient intake for all the children at baseline

Nutrient	Mean (sd)	[95% CI]	DRI[a] 6–8 years	DRI[a] 9–13 years
	N= 220			
Energy (kcals)	1 677 (399)	[930, 1 817]	1 702[b]	2 303[b]
Kilojoule	7 021(1 671)	[3894, 7 607]	7 126	9 642
Carbohydrate (g)	182 (67)	[173, 190]	130[c]	130[c]
Total protein (g)	40 (15)	[38, 42]	19[c]	34[c]
Total fat (g)	19 (7)	[18, 20]	ND	ND
Vitamin A (ug)	753 (374)	[705, 800]	275[d]	445[d]
Folate (mg)	180 (89)	[169, 192]	160[d]	250[d]
Vitamin C (mg)	156 (72)	[146, 165]	22[d]	39[d]
Zinc (mg)	4 (1)	[3, 4]	4[d]	7[d]
Iron (mg)	11 (5)	[11, 12]	4.1[d]	5.9[d]
Calcium (mg)	153 (61)	[145, 161]	800[d]	1 100[d]
Selenium (mg)	26 (11)	[24, 27]	23[d]	35[d]

kcals (kilocalories); g (grams); ug (micrograms); mg (milligrams); [a] DRI: dietary reference intakes; sourced from Ross et al.[40] [b] EER: estimated energy requirements; ND: not determined; [c] RDA: recommended dietary allowances; [d] EAR: estimated average requirement

3.4.4   Prevalence of inadequate nutrient intake for all the children at baseline

More than half of the children (66%) had energy intakes less than the EER for age, gender and active physical activity level (

TABLE 3.5). Compared to the AMDR, the likelihood of inadequate fat intake was 100%, as all children had an intake of fat that contributed to less than 25% of the daily energy requirement. Inadequate intake of vitamin A, zinc, folate and selenium was likely for 6%, 59%, 38% and 36% respectively. Most notable was that all the children had a likelihood of insufficient calcium intake.

TABLE 3.5: Prevalence of inadequate nutrient intake for all the children and by age group at baseline

Nutrient	% children with inadequate intake	DRI[a] 6–8 years	DRI[a] 9–13 years
	N = 220		
Energy (kcals)	66	1 702[b]	2 303[b]
Carbohydrate (g)	4	130[c]	130[c]
Total protein (g)	0	19[c]	34[c]
Total fat (g)	100	25-35[d]	25-35[d]
Vitamin A (ug)	6	275	445
Folate (mg)	38	160	250
Vitamin C (mg)	0	22	39
Zinc (mg)	59	4	7
Iron (mg)	1	4.1	5.9
Calcium (mg)	100	800	1 100
Selenium (mg)	36	23	35

DRI: dietary reference intakes; sourced from Ross et al.[40]; [a] refers to an average estimated average requirement (EAR) for the specific age group, unless otherwise stated. [b] EER: estimated energy requirements; [c] RDA: recommended dietary allowances; [d] AMDR: acceptable macronutrient distribution range

3.4.5   Anthropometry for all the children at baseline

Anthropometric data was analysed for 223 children at baseline, with 118 from the MVP and 105 in the control group. The WHO 2007 growth reference standards[296] was used to calculate the growth indices for the children. This reference population calculates height-for-age and BMI-for-age indicators for children aged 5 to 19 years and weight-for-age indicators for children aged 5 to 10 years, meaning that weight-for-age data is not available for children above this age. The mean height of all the children was 123.2 cm (11.3 SD) [95% CI; 121.8–124.7] and the mean weight was 24 kg (5.1 SD) [95% CI;23. 4-24.7; Table 3.6). The mean HAZ, WAZ and BMIAZ were below the median of the reference population. The children were generally lean, with a mean triceps skinfold measurement of 4.6 mm and a mean subscapular skinfold measurement of 4.3 mm. Arm muscle area was within the 50th percentile, while arm fat area was below the 5th percentile of Frisancho's norms of upper limb fat and muscle areas.[303]

3.4.6   Prevalence of undernutrition among all the children at baseline

Stunting (< -2 HAZ) was found in 16.6% of the children, with 3.6% severely (< -2 HAZ) stunted. Underweight (< -2 WAZ) occurred in 6% of the children (TABLE 3.7). Mild underweight and mild wasting (WAZ and BMIAZ -1 to -2 sd) were seen in 24.6% and 13.5% of the children respectively. There were no children with severe underweight or severe wasting. Overweight (> +1 BMI-AZ) was found in 5% of the children, and no children with obesity were observed.

TABLE 3.6: Anthropometry and body composition for all the children at baseline

Variable	Mean (SD)	95% CI
	N = 223	
Height (cm)	123.2 (11.3)	121.8, 124.7
Weight (kg)	24 (5.1)	23.4, 24.7
HAZ	-0.8 (1.3)	-1.0, -0.7
WAZ[a]	-0.5 (1.0)	-0.7, -0.4
BMIAZ	-0.2 (0.8)	-0.3, -0.1
MUAC (cm)	17.4 (1.5)	17.2, 17.6
Triceps (mm)	4.6 (1.2)	4.4, 4.7
Subscapular (mm)	4.3 (1.1)	4.1, 4.4
MUAFA (mm)^2	400.8 (131.5)	383.3, 418.2
Body fat (%)	7.5 (2.3)	7.2, 7.8
Fat mass (kg)	1.8 (0.7)	1.7, 1.9
MUAMA (mm)^2	2 048.4 (365.4)	1 999.9, 2 097
Fat free mass (kg)	22.2 (4.7)	21.6, 22.8

HAZ: height-for-age z-score, WAZ: weight-for-age z-score, BMIZ: BMI-for-age z-score, MUAC: mid-upper arm circumference, MUAFA: mid-upper arm fat area, MUAMA: mid-upper arm muscle area, [a] Weight-for-age z-score available for children aged up to 10 years

TABLE 3.7: Prevalence of undernutrition among all the children at baseline

Indicator	N(%)
Stunting	**N = 223**
Severe[a]	8 (3.6)
Moderate[b]	30 (13.5)
All stunted (combines severe and moderate)	37 (16.6)
Mild[c]	70 (31.5)
Normal[d]	115 (51.8)
Underweight [a,e]	
Moderate[b]	11 (6.0)
Mild[c]	45 (24.6)
Normal[d]	127 (69.4)
Wasting[a]	
Moderate[b]	7 (3.2)
Mild[c]	30 (13.5)
Normal[d]	175 (78.8)
Overweight[f]	11 (5.0)

[a] Severe is defined as height-for-age < -3 sd from the WHO 2007 growth reference standard
[b] Moderate is defined as height-for-age/weight-for-age and BMI-for-age between -2 and -3 standard deviations below the median of the WHO 2007 growth reference
[c] Mild is defined as height-for-age/weight-for-age and BMI-for-age from -1 to -2 standard deviations below the median of WHO 2007growth reference.
[d] Normal is defined as height-for-age and weight-for-age above -1 standard deviations of the median of the WHO 2007 growth reference
[e] Weight-for-age available for children up to 10 years of age: N = 183
[f] Overweight was defined as BMI-for-age above 1 standard deviation above the median of the WHO 2007 growth reference standards

3.4.7   Initial nutritional status of all the children at baseline

Initial nutritional status was determined by height-for-age, weight-for-age and BMI-for-age z-scores. All children with mild, moderate and severe malnutrition based on a z-score of less than -1.0 standard deviations from the WHO 2007 growth reference standard were classified as having inadequate nutritional status. Almost half (48%) had inadequate initial nutritional status based on height-for-age z-score. About one third (30%) of the children had inadequate initial nutritional status based on weight-for-age z-score, while 16% had an initial inadequate nutritional status based on BMI-for-age z-scores (TABLE 3.8)

TABLE 3.8:   Prevalence of initial inadequate nutritional status for all the children at baseline

Indicator	Nutritional status	Frequency	percent
	N = 223		
Height-for-age	Inadequate[a]	107	48.0
	Adequate[b]	116	52.0
Weight-for-age	Inadequate[a]	55	30.0
	Adequate[b]	128	70.0
BMI-for-age	Inadequate[a]	36	16.1
	Adequate[b]	187	83.9

[a] Height-for-age, weight-for-age and BMI-for-age < -1standard deviations from the WHO 2007 growth reference standard

[b] Height-for-age, weight-for-age and BMI-for-age > -1 standard deviations from the WHO 2007 growth reference standard

3.4.8   Full haemogram, serum ferritin, CRP and serum retinol concentration for all the children at baseline

Full haemograms were analysed for 235 children at baseline (TABLE 3.9). Overall, the mean haemoglobin (Hb) concentration was 10 g/dl (1.6 SD) [95% CI; 9.8, 10.2]. The mean white blood cell count was 6.8 (SD 1.8) [95% CI; 6.5, 7.0] 10^3cells/uL. Overall, the mean MCV and mean platelet count were 81.3 (SD 9.3) [95% CI 81.3 (9.3) fl and 290.7 (116.3 SD) [95% CI; 276.9, 306.5] 10^3/cells uL respectively. Serum ferritin samples were analysed for a subsample of 84 children. The mean serum ferritin concentration was 41.3 (27.0 SD) [95% CI; 35.4- 47.2] µg/l, while the mean CRP concentration was 4.1 (6.2 SD) [95% CI; 2.8,5.5; TABLE 3.9].

3.4.9   Prevalence of anaemia and depleted body iron stores for all the children at baseline

The prevalence of anaemia was 70.2% overall at baseline (TABLE 3.10). Mean cell volume was low (MCV < 82 fl) among 61.3% of the children, and 15.5% had depleted body iron stores as depicted by serum ferritin < 15 µg/l. Of the 235 children, 11.9% had elevated CRP levels, indicating the presence of inflammation/infections. None of the children with depleted body iron

stores had elevated CRP levels. The prevalence of anaemia overall indicated a severe, significant public health problem in the study area using WHO cut-off data for public health significance in a population (TABLE 3.11).

TABLE 3.9: Full haemogram parameters, serum ferritin and CRP levels for all the children at baseline

Full haemogram parameter (N = 235)	Normal ranges	Mean (SD)	95% confidence interval
WBC (10^3 cells/uL)	6–14	6.8 (1.8)	6.5, 7
RBC (10^6 cells /uL)	4–5.2	4.1 (0.7)	4.0, 4.2
Hb (g/dL)	11.5–15.5	10 (1.6)	9.8, 10.2
Hct (%)	35–45	35.8 (30.3)	30.1, 39.9
MCV (fL)	77–95	81.3 (9.3)	80.1, 82.5
MCH (pg)	23–31	24.1 (3.3)	23.7, 24.6
MCHC (g/dL)	28–33	29.9 (3.3)	29.5, 30.3
Plt (10^3/cells uL)	150–400	290.7 (116.3)	276.9, 306.5
ALC (%)	25–50	44.4 (8.9)	43.7, 45.8
Lymph abs (10^3 uL)	5–8.5	3 (0.9)	2.9, 3.1
Serum ferritin (µg/l) N = 84	15–55	41.3 (27.2)	35.4, 47.2
CRP (mg/l) N = 84	1–3	4.1 (6.2)	2.8, 5.5

Note: WBC (white blood cells), RBC (red blood cells), HB (haemoglobin), HCT (haematocrit), MCV (mean cell volume), Fl (femtolitres), MCH (mean cell haemoglobin), MCHC (mean cell haemoglobin concentration), Plt (platelets), Lymph abs (lymphocytes), ALC (absolute lymphocyte count), CRP (C-reactive protein)

TABLE 3.10: Prevalence of anaemia and depleted body iron stores for all the children at baseline

Variable	Indicator	N (%)
Anaemia based on haemoglobin[1] (N = 235)	Low	165 (70.2)
	Normal	70 (29.8)
Classification of anaemia	Severe[2]	11 (4.7)
	Moderate[3]	88 (37.4)
	Mild[4]	66 (28.1)
Microcytic anaemia based on MCV[5] (N = 235)	Low[5]	144 (61.3)
	Normal	91 (38.7)
Depleted body iron stores based on serum ferritin[6] (N = 84)	Depleted stores[6]	13 (15.5)
	Normal stores	71 (84.5)
CRP levels (N = 84)	Elevated CRP	10 (11.9)
	Normal CRP	74 (88.1)

[1] Classification of anaemia by haemoglobin; Hb < 11.0 g/dl for 6- to 11-year-olds; Hb cut-off for 12- to 13-year-olds was Hb < 11.5 g/dl; [2]Severe anaemia (all ages) – Hb < 7.5 g/dl); [3]Moderate anaemia: Hb 7.5 to 10.4 g/dl (6 to 11 years) and Hb 7.5 to 10.3 (up to 13 years); [4]Mild anaemia: Hb 10.5 to 10.9 g/dl (6 to 11 years) and Hb 10.4 to 11.4 g/dl (12 to 14 years) ; [5]Not anaemic: Hb ≥ 11.0 g/dl (6 to 11 years) and Hb ≥ 11.5 g/dl (12 to 14 years); [6] Low MCV – mean cell volume < 82 fl; [7]depleted body iron stores: serum ferritin < 15 ug/l for children ages 5 to 11 years and less than 12 ug/l for children 12 to 14 years; CRP (C-reactive protein)

TABLE 3.11: Degree of public health significance of anaemia for all the children at baseline

Public health significance for anaemia		
		Percentage with anaemia
Degree of public health significance for anaemia	WHO cut-off[a]	Study population
Severe	≥ 40	70.2
Moderate	20.0–39.9	
Mild	5.0–19.9	
None	≤ 4.9	

[a]Source: World Health Organisation[300]

3.4.10 Anaemia and body iron stores by bivariate and trivariate analysis for all the children at baseline

The results of the bivariate analysis of Hb and MCV at baseline showed that 42.1% of the children had microcytic anaemia (low Hb and low MCV; TABLE 3.12). Out of the 235 children, 10.7% had iron deficiency anaemia by bivariate analysis of low Hb and low serum ferritin. Trivariate analysis (low Hb, low MCV and low ferritin) showed that 8.3% of the children had microcytic iron deficiency anaemia (TABLE 3.12).

TABLE 3.12:   Multivariate and bivariate analysis for iron status for all the children at baseline

Indicator	Frequency (%)
N	235
Bivariate analysis by haemoglobin (g/dl) and MCV (fl) [N(%)];	
Normal Hb and normal MCV	26 (11.1)
Low Hb[1] and normal MCV	66 (28.1)
Normal Hb and low MCV[2]	45 (19.1)
Low Hb and low MCV	98 (41.7)
Bivariate analysis by haemoglobin (g/dl) and ferritin (ug/l) [N(%)]	
N	84
Normal Hb and ferritin[3]	9 (10.7)
Low Hb and normal ferritin	62 (73.8)
Normal Hb and low ferritin	4 (4.8)
Low Hb and low ferritin	9 (10.7)
Trivariate analysis by haemoglobin (g/dl), ferritin (ug/l) and MCV (fl)	
N	84
Low Hb, low ferritin and low MCV	7 (8.3)

* Statistically significant at p value < 0.001; [1]low haemoglobin (adjusted) ≤ 11.0 g/dl for 5 to 11 years and < 11.5 g/dl for 12 to 14 years; [2]low MCV ≤ 82 fl; [3]low ferritin ≤ 15 ug/l for children aged 5 and above

3.4.11 Serum retinol and vitamin a status for all the children at baseline

Serum retinol data was analysed for a subsample of 94 children overall. The mean serum retinol concentration was 0.73 (0.2SD) [95% CI; 0.69, 0.77] µmol/L (TABLE 3.13). A Pearson's correlation test did not show any significant correlations between vitamin A intake and serum retinol levels (Pearson's r = 0.041; data not in the table). Only one child was found to be vitamin A deficient (serum retinol <0.35 µmol/L) at baseline. A further 39.4% of the children had marginal vitamin A status (serum retinol 0.35-0.70 µmol/L), while 59.6% of the children had adequate vitamin A status. When vitamin A deficiency and marginal status were combined, a total of 37 (40.4%) of the children had inadequate vitamin A status (serum retinol <0.70 µmol/L. This was considered a severe public health problem in a study based on the WHO cut-off for public health significance in a population (TABLE 3.14).

TABLE 3.13: Serum retinol concentration and vitamin A status for all the children baseline

Serum retinol	Indicator	
	Mean (SD)	*95% confidence interval*
Serum retinol (µmols/l) concentration [N = 94] Normal range ≥ 0.70 µmol/L	0.73 (0.2)	0.69, 0.77
Vitamin A status	*N*	*%*
Vitamin A deficiency (Serum retinol <0.35 µmol/L)	1	1.1
Marginal vitamin A status (Serum retinol 0.35-0.70 µmol/L)	37	39.4
Inadequate vitamin A status (Serum retinol <0.70 µmol/L)	38	40.4
Normal vitamin A status (Serum retinol ≥ 0.70 µmol/L)	56	59.6

Source: World Health Organisation[301]

TABLE 3.14: Degree of public health significance of inadequate vitamin A status for all the children at baseline

Public health significance of inadequate vitamin A status		
	Prevalence of inadequate vitamin A status (<0.70 µmol/L)	
Degree of public health significance of inadequate vitamin A status	WHO cut-off[a]	Prevalence in the study population (N = 94)
Severe	≥ 20%	40.4%
Moderate	10–19%	
Mild	2–9%	

[a]World Health Organisation[301]

3.5   Part 2: Bivariate Results: Anthropometry, Haemoglobin, Serum Ferritin, CRP and Serum Retinol Measurements by Socio-demographic Characteristics for all the Children at Baseline

Part 2 of section A presents the bivariate results for the combined group of children, testing associations between the anthropometry, prevalence of anaemia, body iron stores and vitamin A status, with socio-demographic characteristics.

3.5.1   Anthropometric indicators by socio-demographic characteristics for all the children at baseline

This analysis was done for 220 children for whom there was anthropometry and socio-demographic data at baseline, with 118 in the MVP and 102 in the control group. Initial nutritional status was defined by height-for-age, weight-for-age and BMI-for-age $\leq$ -1 standard deviations (SD). Based on a height-for-age z-score of less than -1.0 SD, almost half (42.3%) of the total number of children had inadequate nutritional status or linear growth deficit, while 23.6% and 12.3% had inadequate nutritional status based on weight-for-age and BMI-for-age $\leq$ -1 standard deviation z-scores respectively (TABLE 3.15). There were no significant differences in the level of inadequate nutritional status between the two study groups at baseline.

When grouped together, age was significantly associated with initial nutritional status. Linear growth deficit (height-for-age < -1 SD) and inadequate nutritional status based on BMI-for-age < -1 SD were more common among the older children than the younger ones (chi-square test: $p = 0.034$ and chi-square test: $p = 0.005$ for linear growth failure and for BMI-for-age respectively). The prevalence of linear growth deficit was higher among children of unemployed mothers compared to those of employed mothers (chi-square test: $p = 0.05$; TABLE 3.15).

TABLE 3.15: Nutritional status based on anthropometric indices by social demographic characteristics for all the children at baseline

Characteristic	N	Height-for-age ≤ -1 SD N (%)	Height-for-age > -1 SD N (%)	Chi-square test: p	Weight-for-age ≤ -1 SD N (%)	Weight-for-age > -1 SD N (%)	Chi-square test: p	BMI-for-age ≤ -1 SD N (%)	BMI-for-age > -1 SD N (%)	Chi-square test: p
Total	220	93 (42.3)	127 (57.7)		45 (23.6)	146 (76.4)		27 (12.3)	193 (87.7)	
Gender										
Male	113	52 (46)	61 (54)	0.154	23 (24.2)	72 (75.8)	0.484	10 (8.8)	103 (91.2)	0.083
Female	107	41 (38.3)	66 (61.7)		22 (22.9)	74 (77.1)		17 (15.9)	90 (84.1)	
Age										
6 to 8 years	131	47 (36.7)	81 (63.3)	0.034*	27 (21.1)	101 (78.9)	0.167	9 (7.0)	74 (80.4)	0.005*
9 to 13 years	92	46 (50)	46 (50)		18 (28.6)	45 (71.4)		18 (19.6)	119 (93)	
Head of household										
Mother and others	67	26 (38.8)	41 (61.2)	0.295	12 (20.7)	46 (79.3)	0.337	9 (13.4)	58 (86.6)	0.442
Father	153	67 (43.8)	86 (56.2)		33 (24.8)	100 (75.2)		18 (11.8)	135 (88.2)	
Employment of head of household										
Not employed	78	33 (42.3)	45 (57.7)	0.553	17 (24.3)	53 (75.7)	0.496	10 (12.8)	68 (87.2)	0.506
Employed	142	60 (42.3)	82 (57.7)		28 (23.1)	93 (76.9)		17 (12)	125 (88)	
Mother's education										
None	13	4 (30.8)	9 (69.2)	0.662[a]	2 (20)	8 (80)	0.823	4 (30.8)	9 (69.2)	0.112[a]
Primary	156	68 (43.6)	88 (56.4)		31 (23.3)	102 (76.7)		17 (10.9)	35 (85.4)	
Secondary	41	17 (41.5)	24 (58.5)		11 (27.5)	29 (72.5)		6 (14.6)	139 (89.1)	
Mother's marital status										
Single	50	22 (44)	28 (56.0)	0.458	9 (22)	32 (78)	0.449	4 (8)	46 (92)	0.176
Married	160	67 (41.9)	93 (58.1)		35 (24.6)	107 (75.4)		23 (14.4)	137 (85.6)	
Mother's employment status										
None	55	29 (52.7)	26 (47.3)	0.05	15 (30.6)	34 (69.4)	0.144	9 (16.4)	46 (83.6)	0.247
Employed	155	60 (38.7)	95 (61.3)		29 (21.6)	105 (78.4)		18 (11.6)	137 (88.4)	
Total household income/month										
Less than KES 1,000	123	52 (42.3)	71 (57.7)		25 (23.6)	81 (76.4)		14 (11.4)	109 (88.6)	
More than KES 1,000	97	41 (42.3)	56 (44.1)	0.554	20 (23.5)	65 (76.5)	0.566	13 (13.4)	84 (86.6)	0.401

* Statistically significant at the level of $P < 0.05$; [a] Fisher's exact test; the mother's education, employment and household monthly income

3.5.2 Anaemia and body iron stores by socio-demographic characteristics for all the children at baseline

The prevalence of anaemia and body iron stores were analysed on the basis of household socio-demographic characteristics. The association between household headship and anaemia was significant (chi-square test: p = 0.021). There was a higher prevalence of anaemia (71.6%) among children living in households headed by the mother or other relatives compared to those in households headed by fathers (56.2%; TABLE 3.16). Although not statistically significant, the prevalence of anaemia and depleted body iron stores was higher among children from households in which the household head did not have any form of employment.

TABLE 3.16: Anaemia and body iron stores by socio-demographic characteristics for all the children at baseline

Characteristic	N	Anaemic N(%)	Not anaemic N(%)	Chi-square tests: p	N	Depleted body iron stores N(%)	Normal iron stores N(%)	Chi-square tests: p
Head of household								
Mother and others	67	48 (71.6)	19 (28.4)	0.021*	30	5 (16.7)	25 (83.3)	0.612
Father	153	86 (56.2)	67 (43.8)		54	9 (16.7)	45 (83.8)	
Employment of head of household								
Not employed	78	49 (62.8)	29 (37.2)	0.389	34	8 (23.5)	26 (76.5)	0.137
Employed	142	85 (59.9)	57 (40.1)		50	6 (12)	44 (88.0)	
Mother's education[a]								
None	13	9 (69.2)	4 (30.8)		4	1 (25)	3 (75)	0.804[b]
Primary	41	24 (58.5)	17 (20.5)	0.785[b]	19	4 (21.1)	15 (78.9)	
Secondary	156	94 (60.3)	62 (39.7)		57	9 (15.8)	48 (84.2)	
Mother's marital status[a]								
Single	50	34 (68)	16 (32)	0.14	24	7 (29.2)	17 (70.8)	0.073
Married	160	93 (58.1)	67 (41.9)		56	7 (12.5)	49 (87.5)	
Mother's employment status[a]								
None	55	34 (61.8)	21 (38.2)	0.472	18	4 (22.2)	14 (77.8)	0.387
Employed	155	93 (60)	62 (40)		62	10 (16.1)	52 (83.9)	
Total household income/month								
Less than KES 1,000	123	74 (60.2)	49 (39.8)		51	8 (15.7)	43 (84.3)	
More than KES 1,000	97	60 (61.9)	37 (38.1)	0.454	33	6 (18.2)	27 (81.8)	0.494

[a] Number excluded data for children whose mothers were deceased; n = 10 for anaemia and n = 4 for body iron stores; [b] Fisher's exact test

3.5.3   Vitamin A status by household socio-demographic characteristics for all the children at baseline

Vitamin A status was analysed on the basis of household socio-demographic characteristics, including the one child who was vitamin A deficient in the inadequate status category. This was done in order to obtain adequate statistical power for the chi-square test. A significant association was found between vitamin A status and the employment status of the household head. Children from households in which the household head had no form of employment had a higher prevalence of inadequate vitamin A status (chi-square test: $p = 0.021$) when compared to those from households with employed household heads (TABLE 3.17).

TABLE 3.17: Vitamin A status by household socio-demographic characteristics

Characteristic		Inadequate vitamin A status (<0.7 μmol/L)	Adequate vitamin A status (≥0.7 μmol/L)	Chi-square tests; p values
	N		N (%)	
Total	*94*			
Head of household				
Mother and others	31	12 (38.7)	19 (61.3	0.381
Father	63	28 (44.4)	35 (55.6)	
Employment of head of household				
Not employed	37	21 (56.8)	16 (43.2)	0.021*
Employed	57	19 (33.3)	38 (66.7)	
Mother's education[b]				
Primary or none	25	11 (44.0)	14 (56.0)	0.561
Secondary	65	28 (43.1)	37 (56.9)	
Mother's marital status[b]				
Single	24	12 (50)	12 (50)	0.297
Married	66	27 (40.9)	39 (59.1)	
Mother's employment status[b]				
None	23	10 (43.5)	29 (43.3)	0.588
Self-employed	67	13 (56.5)	38 (56.7)	
Total household income/month				
Less than KES 1,000	59	21 (35.6)	38 (64.4)	
More than KES 1,000	35	19 (54.3)	16 (45.7)	0.06

[a] Includes one case with serum retinol < 0.35 μmol/L; [b] Number excludes data for children whose mothers were deceased; n = 4; *Significantly at P < 0.05

3.5.4   Multiple nutritional deficiencies amongst all the children at baseline

Overall, 8% of the 84 children whose data was available for serum ferritin and serum retinol had iron deficiency and inadequate vitamin A status, while 31% of 94 children had anaemia and inadequate vitamin A status. Almost 5% were found to have iron deficiency and anaemia (Figure 3.2).
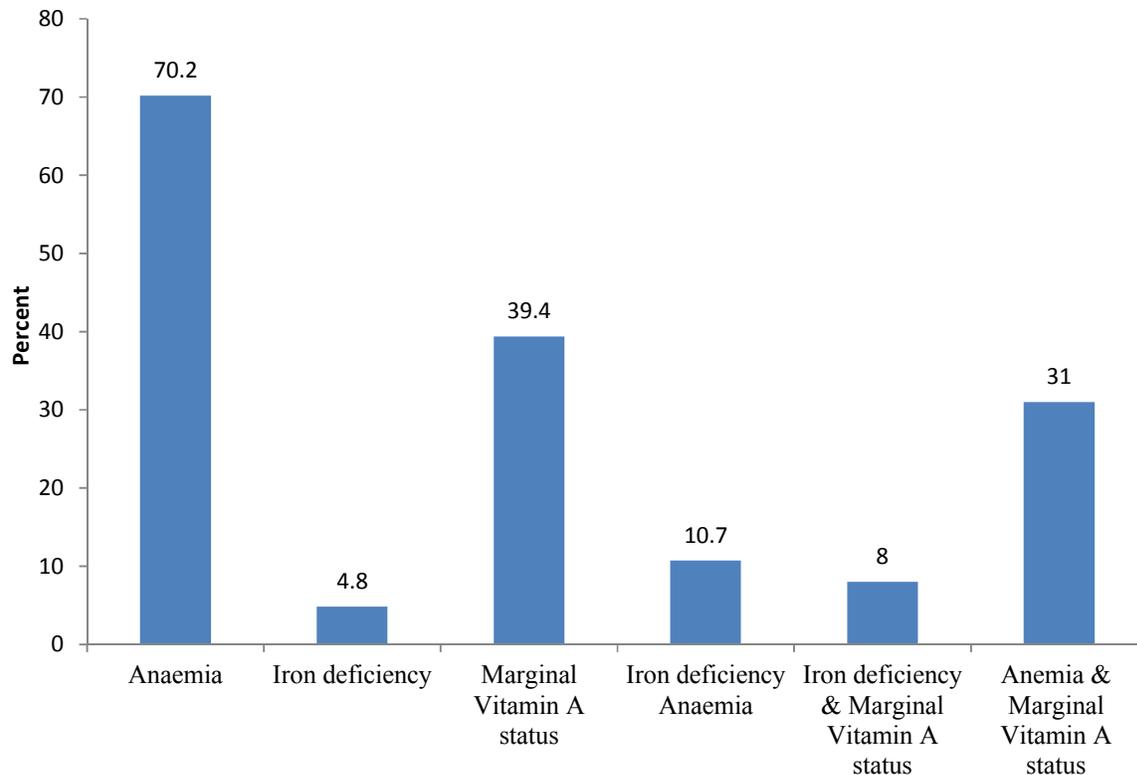
FIGURE 3.2: Prevalence of multiple nutritional deficiencies amongst all the children at baseline

3.5.5   Full haemogram parameters, serum ferritin and serum retinol by anthropometric indicators for all the children at baseline

Full haemogram parameters, serum ferritin and serum retinol were compared by anthropometric indicators for all the children at baseline. Children with low anthropometric indicators ($\leq$ -1 SD) based on the three indices had significantly lower mean red blood cell counts (ANOVA: $p = 0.001$, 0.002 and 0.005 and Hb for height-for-age, weight-for-age and BMI-for-age z-scores respectively) and significantly lower haemoglobin concentrations (ANOVA: $p = 0.033$, 0.026 and 0.009 for height-for-age, weight-for-age and BMI-for-age z-scores respectively; TABLE 3.18).

Children with low BMI-for-age z-scores ($\leq$ -1 SD) had a significantly higher mean MCV of 85.5 fl (13.9 SD) [95% CI; 80.2, 90.8] compared to those who had a high BMI-for-age z-score (> -1 SD) (ANOVA: $p = 0.014$). The mean serum ferritin concentration was lower in children with weight-for-age and BMI-for-age z-scores > -1 compared to their counterparts with weight-for-age and BMI-for-age z-scores $\leq$ -1 SD. However, these differences were not statistically significant.

Similarly, the mean serum retinol concentration was lower among children with weight-for-age and BMI-for-age z-scores ≤ -1, although the differences were not statistically significant.

3.5.6   Prevalence of anaemia, depleted body iron stores and vitamin A status by anthropometric indicators for all the children at baseline

The results of the bivariate analysis comparing anaemia, depleted body iron stores and vitamin A status by anthropometric indicators for all the children at baseline based on height-for-age, weight-for-age and BMI-for-age are shown in TABLE 3.19. The prevalence of linear growth deficit (height-for-age z-scores ≤ -1 SD) was higher (51.9%) amongst anaemic children as compared to those without anaemia (chi-square test: $p = 0.05$). The proportion of children with initial inadequate nutritional status based on weight-for-age and BMI-for-age was significantly higher among the anaemic children than amongst those without anaemia (chi-square test: $p = 0.041$ and 0.003 respectively). There was no association between the prevalence of depleted body iron stores and prevalence of inadequate vitamin A status with anthropometric indicators (TABLE 3.19).

TABLE 3.18: Full haemogram, serum ferritin and retinol by anthropometric indicators for all the children at baseline

Indicator	Height-for-age ≤ −1 N = 107	Height-for-age > -1 N = 116	ANOVA test: p	Weight-for-age ≤ −1 N = 55	Weight-for-age > -1 N = 129	ANOVA test: p	BMI-for-age ≤ −1 N = 36	BMI-for-age > -1 N = 187	ANOVA test: p
				Blood cell count: Mean (SD) [CI]					
WBC (10³ cells/ul)	6.6 (1.6) [6.2, 6.9]	6.8 (2.0) [6.5, 7.2]	0.293	6.9 (1.9) [6.4, 7.4]	6.7 (2.0) [6.4, 7.1]	0.649	6.4 (1.5) [5.8, 7.0]	6.8 (1.9) [6.5, 7.0]	0.325
RBC (10⁶cells/ul)	4.0 (0.6) [3.9, 4.1]	4.2 (0.6) [4.1, 4.3]	0.001*	3.9 (0.5) [3.7, 4.0]	4.2 (0.6) [4.1, 4.3]	0.002*	3.8 (0.6) [3.6, 4.1]	4.2 (0.6) [4.1, 4.2]	0.005*
Hb (g/dl)	9.7 (1.7) [9.4, 10.0]	10.2 (1.5) [9.9, 10.4]	0.033*	9.4 (1.4) [9.0, 9.8]	10 (1.6) [9.7, 10.3]	0.026*	9.3 (1.5) [8.7, 9.8]	10.1 (1.6) [9.8, 10.3]	0.009*
Hct (%)	32.1 (4.0) [31.3, 32.9]	39.3 (41.5) [31.9, 46.7]	0.09	31.8 (3.5) [30.8, 32.8]	38.1 (39.3) [31.5, 44.7]	0.249	32.2 (3.6) [30.8, 33.5]	36.7 (33.5) [31.9, 41.5]	0.469
MCV (fl)	81.6 (8.5) [79.8, 83.3]	81.4 (10.3) [79.6, 83.3]	0.919	83.2 (10.3) [80.4, 86.1]	81 (9.6) [79.4, 82.6]	0.154	85.5 (13.9) [80.2, 90.8]	80.9 (8.6) [79.6, 82.1]	0.014*
MCH (pg)	24.5 (2.7) [23.9, 25.0]	23.9 (3.8) [23.2, 24.5]	0.173	24.5 (2.8) [23.7, 25.2]	23.8 (3.7) [23.2, 24.4]	0.254	24.2 (3.2) [23, 25.4]	24.1 (3.4) [23.6, 24.6]	0.894
MCHC (g/dl)	30.0 (2.8) [29.5, 30.6]	32.0 (29.5) [26.7, 37.3]	0.512	29.5 (3.1) [28.6, 30.3]	31.8 (27.9) [27.1, 36.5]	0.542	28.8 (3.6) [27.4, 30.2]	31.5 (23.7) [28.1, 34.9]	0.54
Plt (10³ cells/ul)	276 (99.1) [256, 296.0]	296.6 (121.9) [274.8, 318.3]	0.18	289.8 (125.1) [255.4, 324.3]	289.6 (114.4) [270.3, 308.9]	0.99	277.4 (132.8) [226.9, 327.9]	289 (109.6) [273.4, 304.7]	0.606
ALC (%)	44.9 (8.4) [43.2, 46.6]	44.4 (9.2) [42.8, 46.1]	0.705	45.2 (8.0) [43.0, 47.4]	44.5 (9.3) [42.9, 46.1]	0.639	46.5 (9.0) [43.1, 49.9]	44.4 (8.8) [43.1, 45.6]	0.221
LY (10³ cells /ul)	2.9 (0.9) [2.8, 3.1]	3.1 (0.9) [2.9, 3.2]	0.326	3.1 (1.0) [2.8, 3.4]	3.0 (0.9) [2.8, 3.2]	0.535	2.9 (0.7) [2.7, 3.2]	3.0 (0.9) [2.9, 3.1]	0.688
				Serum ferritin: Mean (SD) [CI]					
N	35	49		19	55		12	72	
Serum ferritin (µg/l)	42.0 (27.4) [32.6, 51.4]	40.8 (27.1) [33.0, 48.5]	0.839	41.0 (33.8) [24.7, 57.3]	39.8 (22.2) [33.8, 45.8]	0.864	44.8 (29.7) [25.8, 63.7]	40.7 (26.7) [34.4, 47]	0.634
				Serum retinol: Mean (SD) [CI]					
N	39	55		22	62		12	82	
Serum retinol (µmol/L)	0.74(0.28) [0.65,0.83]	0.73(0.19) [0.67,0.78]	0.778	0.69(0.25) [0.58,0.8]	0.73(0.21) [0.68,0.79]	0.473	0.77(0.28) [0.59,0.95]	0.73(0.22) [0.68,0.77]	0.538

Note: WBC (white blood cells), RBC (red blood cells), HB (haemoglobin), HCT (haematocrit), MCV (mean cell volume), Fl (femtolitres), MCH (mean cell haemoglobin), MCHC (mean cell haemoglobin concentration), Plt (platelets), Ly (lymphocytes), ALC (absolute lymphocyte count); * statistically significant at p value < 0.05

TABLE 3.19:   Prevalence of anaemia, depleted body iron stores, and inadequate vitamin A status by initial nutritional status based on anthropometric indicators for all the children at baseline

Variable	N	Height-for-age ≤ -1	Height-for-age > -1	Chi-square test; p value	Weight-for-age ≤ -1[c]	Weight-for-age > -1	Chi-square test; p value	BMI-for-age ≤ -1	BMI-for-age > -1	
	N	107	116		55	129		36	187	
					N(%)					
Anaemia present	154	80 (51.9)	74 (48.1)	0.052	45 (33.8)	88 (66.2)	0.041*	32 (20.8)	122 (79.2)	0.003*
No anaemia	69	27 (39.1)	42 (60.9)		10 (19.6)	41 (80.4)		4 (5.8)	65 (94.2)	
Depleted body iron stores[b]	14	7 (50.0)	7 (50.0)	0.239	4 (33.3)	8 (66.7)	0.278	1 (7.1)	13 (92.9)	0.473
Normal iron stores	70	25 (35.7)	45 (64.3)		13 (21.0)	49 (79.0)		9 (12.9)	61 (87.1)	
Inadequate vitamin A status (serum retinol <0.70 µmol/L)	40	15 (37.5)	25 (62.5)	0.532	9 (24.3)	28 (75.7)	0.47	2 (5.0)	38 (95.0)	0.116
Normal vitamin A status (serum retinol ≥ 0.70 µmol/L)	54	21 (38.9)	33 (61.1)		10 (21.3)	37 (78.7)		8 (14.8)	46 (85.2)	

[a] Hb < 11.0 g/dl for 6 to 11-year-olds; and Hb < 11.5 g/dl for 12-to-13-year-olds; [d] Depleted body iron stores: serum ferritin < 15 ug/l for children aged 5 to 11 years and less than 12 ug/l for children aged 12 to 14 years; [c] weight-for-age determined only for children aged up to 10 years old; N = 133 for anaemia present, 51 for no anaemia, 12 for depleted body iron stores, 62 for normal iron stores, 37 for serum retinol <0.70 µmol/L and 47 for serum retinol ≥ 0.70 µmol/L; *Statistically significant at P value < 0.005

3.6   Part 3: Findings Comparing Two Study Groups, Sex and Age Groups at Baseline

Part 3 of section A presents comparisons of the various outcomes in the two separate study groups (MVP and controls), between sexes (male and female) and according to age groups (6 to 8 years and 9 to 13 years).

3.6.1   Demographic and socio-economic characteristics by study group, sex and age at baseline

The mean age of the children was 7.8 years among the MVP children and 8 years for the control group (TABLE 3.20). The sex distribution did not differ between the groups. The percentage of male-headed households was significantly higher in the control group (chi-square test: $p = 0.004$), with a higher percentage of households headed by mothers (22%) and grandparents (17.8%) in the MVP group than in the control group. There was no significant difference in the reported total household income of the study groups.

3.6.2   Nutrient intake by study group, sex and age at baseline

The mean daily energy intake was similar for both groups of children (TABLE 3.21). This intake is about equal to the average estimated energy requirements for the 6 to 8-year-olds with an active physical activity level, but below the requirement for older children (9 to 13 years). There were no significant differences in carbohydrate, total protein and total fat intake between the study groups at baseline. The mean daily intake of macronutrients did not differ between the males and females or between the two age groups (6 to 8 years and 9 to 13 years). Mean daily vitamin A intake was similar for the two groups of children; 760 µg (378 SD) [95% CI; 696, 823] for the MVP group and 743 µg (371 SD) [95% CI; 671, 816] for the control group. The daily mean intake for folate and selenium was below the estimated average requirement for the older children. More striking, though, was the very low calcium intake of 154 mg (55 SD) [95% CI; 145, 164] and151 mg (69 SD) [137, 164] among the MVP and control groups respectively, against a daily EAR of 800 mg for the 6- to 8-year-olds and 1 100 mg for the 9 to 13-year-olds. There were no significant differences between the mean intake of the seven selected micronutrients between the study groups or the sex and age groups (TABLE 3.21).

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TABLE 3.20: Household and child socio-demographic characteristics by study group at baseline

Characteristic	Characteristic	MVP (N = 118)	Control (N = 102)	Chi-square; p
Age in years	Mean (SD) (Range 6 to 13 years)	7.8 (2.0)	8.0 (1.9)	0.336^{a}
	Age groups		[N (%)]	
	6–8 years	71 (60.2)	59 (50.4)	0.85
	9–13 years	47 (39.8)	58 (49.6)	
Sex of child	Males	60 (50.8)	53 (52.0)	0.488
	Females	58 (49.2)	49 (48.0)	
Head of household	Father	71 (60.1)	82 (80.4)	0.004*
	Mother	26 (22)	9 (8.8)	
	Grandparent or other relatives	21 (17.8)	11 (10.8)	
Employment of head of household	Self-employed	53 (44.9)	47 (46.1)	0.085
	Employed	28 (23.7)	22 (21.6)	
	Unemployed	20 (16.9)	8 (7.8)	
	Deceased	17 (14.4)	25 (24.5)	
Mother's education	Primary school	80 (67.8)	75 (73.5)	0.534
	Secondary school	23 (19.5)	18 (17.6)	
	None	10 (8.5)	4 (3.9)	
	Deceased	5 (4.2)	5 (4.9)	
Mother's marital status	Married	83 (70.3)	77 (75.5)	0.587
	Single	30 (25.4)	20 (19.6)	
	Deceased	5 (4.2)	5 (4.9)	
Mother's employment	Self-employed	76 (64.4)	66 (64.7)	0.685
	Unemployed	28 (23.7)	27 (26.5)	
	Employed	9 (7.6)	4 (3.9)	
	Deceased	5 (4.2)	5 (4.9)	
Total household income/month	Less than KES 1,000	66 (55.9)	57 (55.9)	0. 551
	More than KES 1,000	52 (44.1)	45 (44.1)	

[a] P value determined by ANOVA comparison in of means; *Statistically significant for differences between MVP1 and controls at P < 0.05

TABLE 3.21: Nutrient intake at baseline by study group, sex and age

Nutrient	MVP n = 118	Control n = 102	ANOVA test: p	Male n = 113	Female n = 107	ANOVA test: p	6–8 years n = 128	DRI[a] 6–8 years	9–13 years n = 92	DRI[a] 9–13 years	ANOVA test: p
Energy (kcals)	1 660 (395) [905, 1 722]	1 696 (404) [922, 1 857]	0.5	1 677 (411) [923, 1 757]	1 678 (389) [903, 1 714]	0.98	1 645 (405) [893, 1 803]	1 702[b]	1 737 (384) [953, 1 988]	2 303[b]	0.1
Energy (kJ)	6 950 (1 654) [3 789, 7 210]	7 101 (1 691) [3 860, 7 775]		7 021 (1 721) [3 864, 7 356]	7 025 (1 629) [3 781, 7 176]		6 887 (1 696) [3 739, 7 549]	7 126	7 272 (1 608) [3 990, 8 323]	9 642	
Carbohydrate (g)	179 (69) [167, 190]	185 (65) [172, 198]	0.4	183 (73) [171, 197]	180 (61) [168, 190]	0.7	176 (69) [164, 185]	130[c]	192 (61) [180, 207]	130[c]	0.1
Total protein (g)	40 (16) [37, 42]	40 (14) [37, 43]	0.9	40 (16) [37, 43]	39 (14) [37, 42]	0.6	40 (16) [37, 42]	19[c]	39 (13) [37, 43]	34[c]	0.8
Total fat (g)	19 (7) [17, 20]	19 (7) [18, 21]	0.5	19 (8) [18, 20]	19 (6) [17, 20]	0.6	19 (7) [18, 20]	ND	19 (6) [18, 20]	ND	0.93
Vitamin A (ug)	760 (378) [696, 823]	743 (371) [671, 816]	0.7	790 (384) [721, 858]	727 (370) [650, 782]	0.2	767 (364) [695, 806]	275[d]	743 (405) [675, 849]	445[d]	0.63
Folate (mg)	185 (92) [170, 201]	174 (84) [157, 190]	0.3	190 (100) [173, 208]	181 (94) [156, 184]	0.1	181 (83) [163, 192]	160[d]	186 (86) [169, 205]	250[d]	0.96
Vitamin C (mg)	155 (67) [144, 166]	156 (80) [141, 172]	0.9	160 (83) [146, 175]	151 (63) [139, 161]	0.3	157 (76) [144, 167]	22[d]	152 (69) [144, 174]	39[d]	0.63
Zinc (mg)	4 (1) [3, 4]	4 (1) [3, 4]	0.8	4 (2) [3, 4]	4 (1) [3, 4]	0.4	4 (2) [3, 4]	4[d]	4 (1) [3, 4]	7[d]	0.64
Iron (mg)	12 (5) [11, 12]	11 (5) [10, 12]	0.8	12 (5) [11, 13]	11 (4) [10, 12]	0.6	12 (5) [11, 12]	4.1[d]	11 (4) [11, 13]	5.9[d]	0.87
Calcium (mg)	154 (55) [145, 164]	151 (69) [137, 164]	0.7	158 (62) [147, 169]	149 (62) [137, 159]	0.3	157 (63) [144, 164]	800[d]	149 (60) [139, 165]	1 100[d]	0.35
Selenium (mg)	26 (11) [24, 28]	25 (10) [23, 27]	0.8	26 (12) [24, 28]	25 (10) [23, 27]	0.4	26 (12) [24, 28]	23[d]	24 (9) [23, 27]	35[d]	0.17

kcals (kilocalories); g (grams); ug (micrograms); mg (milligrams); [a] DRI: dietary reference intakes; sourced from reference[40]; [b] EER: estimated energy requirements; ND: not determined; [c] RDA: recommended dietary allowances; [d] EAR: estimated average requirement

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3.6.3 Prevalence of inadequate intake by study groups, sex and age at baseline

Seventy percent of children in the MVP group had energy intake less than the EER for age, gender and active physical activity, compared to 62% of those in the control group (TABLE 3.22). The older children (9 to 13 years old) had a higher prevalence of inadequate energy intake of 12 percentage points compared to the younger ones. Compared to the AMDR, the likelihood of inadequate fat intake was 100% across the study groups, and the sex and age groups. Inadequate intake of vitamin A was likely among 5% of the children in the MVP group and 8% of the children in the control group. A further 10% of children aged 9 to 13 years did not have enough vitamin A intake compared to 3% of the younger children. Similarly, the prevalence of inadequate folate intake was higher among the older children than the younger ones; 46% versus 31%. This pattern was repeated for the likelihood of inadequate zinc and selenium intakes.

The probability of inadequate calcium intake was 100%, as all the children had a calcium intake less than the EAR across all the comparative groups. There were no noticeable differences in the prevalence of inadequate intake of the seven selected macronutrients between the MVP and the control groups.

TABLE 3.22: Prevalence of inadequate nutrient intake at baseline by study group, gender and age

Nutrient	MVP N = 118	Control N = 102	Male N = 113	Female N = 107	6–8 years N = 128	DRI[a]	9–13 years N = 92	DRI[a]
% children with inadequate intake								
Energy (kcals)	70	62	68	64	62	1 702[b]	74	2303[b]
Carbohydrate (g)	5	3	2	5	5	130[c]	2	130[c]
Total protein (g)	1	0	0	0	0	19[c]	0	34[c]
Total fat (g)	100	100	100	100	100	25–35[d]	100	25–35[d]
Vitamin A (ug)	5	8	6	7	3	275	10	445
Folate (mg)	38	38	39	37	31	160	46	250
Vitamin C (mg)	0	1	1	0	1	22	0	39
Zinc (mg)	60	58	55	63	49	4	71	7
Iron (mg)	1	0	1	1	1	4.1	1	5.9
Calcium (mg)	100	100	100	100	100	800	100	1 100
Selenium (mg)	35	37	37	35	28	23	46	35

DRI: dietary reference intakes; sourced from reference[40]; [a] refers to an average estimated average requirement (EAR) for the specific age group, unless otherwise stated; [b] EER: estimated energy requirements; [c] RDA: recommended dietary allowances; [d] AMDR: acceptable macronutrient distribution range

3.6.4   Anthropometry by study group, sex and age at baseline

Anthropometric measurements of growth and body composition were not significantly different between the MVP and control groups at baseline (TABLE 3.23). The mean BMI-for-age z-score was significantly lower among females than males (ANOVA: p = 0.02). The mean body composition measurements were significantly higher for the females compared to the males (ANOVA: p = 0.021, 0.001, 0.006, 0.001 and 0.001 for triceps, subscapular, mid-upper arm fat area, percent body fat and fat mass respectively). Although not statistically significant, the boys had a higher mean fat-free mass of 22.7 kg (4.8 SD) [95% CI; 21.8, 23.6] than the girls, at 21.7 kg (4.4 SD) [95% CI; 20.8, 22.5]. As expected, the older children were taller and heavier, but they had a significantly lower mean HAZ and BMI-Z (ANOVA: p = 0.004 and 0.000 respectively).

The children were generally lean, with a mean triceps skinfold measurement within the third percentile based on the 2010 skinfold sex- and age-specific reference data from NHANES[304] for boys and below the third percentile for girls. Subscapular skinfold measurements were within the 25th percentile of NHANES for both boys and girls. The older children had significantly higher measurements of body composition, namely mean MUAC (ANOVA: p = 0.000), subscapular skinfold thickness (ANOVA: p = 0.038), fat mass (ANOVA: p = 0.000), fat-free mass (ANOVA: p = 0.000) and mid-upper arm muscle area (ANOVA: p = 0.000), when compared to the younger children (TABLE 3.23).

3.6.5   Prevalence of undernutrition by study group, sex and age at baseline

The prevalence of undernutrition at baseline was compared between study groups, sex and age groups (TABLE 3.24). Of the 118 children in the MVP group, 17.8% were stunted (< -2 HAZ), compared to 15.2% in the control group. This difference was not statistically significant. The proportion of children with mild wasting was 16.2% in the MVP group compared to 9.5% among the controls. When severe and moderate stunting were combined (< -2 HAZ), the males had a significantly higher percentage of stunting (21.2%) than the females (11.4%) (chi-square; p = 0.04). Wasting (BMIZ < -2) was significantly different between the males and females (chi-square test: p = 0.001).

The prevalence of mild wasting was higher among the females (21.0%) than the males (6.0%), while more males (8.5%) were overweight compared to females (1.0%). There was a significant difference in the prevalence of stunting and wasting between the age groups (chi-square test: p =

0.017 and p value < 0.001). The prevalence of stunting was higher among the older children (9 to 13 years) than among the younger ones (6 to 8 years), at 22.8% and 13% respectively. There were no wasted children in the youngest age group, while 7.6% were wasted in the oldest group. In addition, 19.6% of the older children had mild wasting compared to 8.5% in the younger age group (chi-square test: p = 0.000; TABLE 3.24).

3.6.6   Initial nutritional status by study group, sex and age at baseline

Initial nutritional status was determined by height-for-age, weight-for-age and BMI-for-age z-scores for a total of 223 children, with 118 from the MVP and 105 from the control group (TABLE 3.25). All children with mild, moderate and severe malnutrition based on a z-score of less than -1.0 standard deviations from the WHO 2007 growth reference standard were classified as having inadequate nutritional status. The prevalence of inadequate nutritional status was 44.2% in the MVP and 47.6% in the control group. The prevalence of inadequate nutritional status based on BMI-for-age was significantly higher (chi-square test: p = 0.003) among the females (23.8%) than the males (9.3%). More than half (56.5%) of the older children had inadequate nutritional status on the basis of height-for-age z-score compared to the younger ones (42%; chi-square; p = 0.023). Similarly, inadequate nutritional status on the basis of BMI-for-age z-scores was higher among the older children than the younger ones.

TABLE 3.23: Anthropometry and body composition by study group, gender and age at baseline

Variable	MVP (N = 118)	Control (N = 105)	ANOVA test: p	Male N = 118	Female N = 105	ANOVA test: p	6–8 years n = 131	9–13 years n = 92	ANOVA test: p
				[Mean (SD) [95% CI]					
Height (cm)	123.6 (11.6) [121.5, 125.7]	122.8 (11.1) [120.7, 125.0]	0.59	123.1 (11.5) [121, 125.2]	123.4 (11.2) [121.3, 125.6]	0.83	117.1 (9.3) [115.5, 118.7]	132.1 (7.5) [130.5, 133.6]	0.000**
Weight (kg)	23.5 (5.1) [23.1, 25.0]	24 (5.1) [23.0, 25.0]	0.96	24.3 (5.3) [23.3, 25.2]	23.8 (5) [22.8, 24.7]	0.46	21.3 (3.7) [20.7, 22]	27.9 (4.3) [27, 28.8]	0.000**
HAZ	-0.9 (1.2) [-0.1, -0.7]	-0.8 (1.4) [-1.05, -0.5]	0.56	-0.9 (1.4) [-1.1, -0.6]	-0.8 (1.1) [-1.1, -0.6]	0.74	-0.6 (1.4) [-0.9, -0.4]	-1.1 (1.1) [-1.4, -0.9]	0.004**
WAZ[1]	-0.6 (0.9) -0.7, -0.4	-0.5 (1.1) [-.07, -0.2]	0.51	-0.5 (1.1) [-0.7, -0.2]	-0.6 (0.9) [-0.7, -0.2]	0.4	-0.5 (1) [-0.6, -0.3]	-0.7 (1) [-1, -0.4]	0.137
BMIAZ	-0.3 (0.8) [-0.5, -0.1]	-0.1 (0.8) [-0.3, 0.0]	0.09	-0.1 (0.8) [-0.3, -0.02]	-0.4 (0.8) [-0.5, -0.2]	0.02*	0 (0.7) [-0.2, 0.1]	-0.5 (0.8) [-0.7, -0.3]	0.000**
MUAC (cm)	17.4 (1.5) [17.1, 17.7]	17.4 (1.4) [17.2, 17.7]	0.949	17.4 (1.4) [17.1, 17.6]	17.5 (1.6) [17.2, 17.8]	0.686	16.9 (1.3) [16.7, 17.2]	18.1 (1.5) [17.8, 18.4]	0.000***
Triceps (mm)	4.5 (1.2) [4.3, 4.7]	4.6 (1.2) [4.4, 4.9]	0.35	4.4 (0.9) [4.1, 4.6]	4.8 (1.4) [4.5, 5.0]	0.021*	4.6 (1.3) [4.4, 4.9]	4.5 (1) [4.3, 4.7]	0.33
Subscapular (mm)	4.3 (1.2) [4.1, 4.6]	4.2 (1) [4, 4.4]	0.22	3.9 (1) [3.8, 4.1]	4.6 (1.1) [4.4 , 4.8]	0.000**	4.1 (1) [4, 4.3]	4.4 (1.2) [4.2, 4.7]	0.038*
Body fat (%)	7.5 (2.3) [7.1, 7.9]	7.4 (2.3) [7, 7.9]	0.88	6.3 (1.7) [6, 6.6]	8.8 (2.2) [8.4, 9.2]	0.000**	7.3 (2.5) [6.9, 7.8]	7.7 (2) [7.2, 8.1]	0.33
Fat mass (kg)	1.8 (0.8) [1.7, 2]	1.8 (0.7) [1.7, 1.9]	0.74	1.6 (0.6) [1.4, 1.7]	2.1 (0.8) [2, 2.3]	0.000**	1.6 (0.7) [1.5, 1.7]	2.2 (0.7) [2, 2.3]	0.000**
MUAFA (mm)[2]	405 (151.7) [377.3, 432.6]	395.9 (103.8) [375.5, 416.3]	0.613	377.2 (90) [360.5, 394]	425.6 (161.1) [394.7, 456.5]	0.006*	393.5 (130.7) [370.6, 416.3]	410.9 (132.6) [383.5, 438.4]	0.332
MUAMA (mm)[2]	2 044.6 (371.7) [1 976.8, 2 112.3]	2 052.9 (359.7) [1 982.3, 2 123.6]	0.866	2 058.8 (353) [1 993, 2124.6]	2 037.5 (379.4) [1 964.8, 2 110.2]	0.666	1 915.5 (312.3) [1 860.9, 1 970.1]	2 233.4 (354.6) [2 159.9, 2 306.8]	0.000**
Fat-free mass (kg)	22.2 (4.6) [21.4, 23.1]	22.2 (4.7) [21.3, 23.1]	1.0	22.7 (4.8) [21.8, 23.6]	21.7 (4.4) [20.8 , 22.5]	0.092	19.7 (3.3) [19.2, 20.3]	25.8 (3.9) [24.9, 26.6]	0.000**

HAZ: height-for-age z-score; WAZ: weight-for-age z-score; BMIZ: BMI-for-age z-score; MUAC: mid-upper arm circumference; MUAFA: mid-upper arm fat area; MUAMA: mid-upper arm muscle area; *Statistically significant at p value < 0.05; **Statistically significant at p value < 0.01; ***Statistically significant at p value < 0.01 [1]Weight-for-age z-score available for children aged up to 10 years

TABLE 3.24: Prevalence of undernutrition by study group, gender and sex at baseline

Indicator	MVP n = 118	Control n = 105	Fisher's exact test: p[1]	Male n = 118	Female n = 105	Fisher's exact test: p	6 to 8 years n = 131	9 to 13 years n = 92	Fisher's exact test: p
				N(%)					
Stunting									
Severe[a]	5 (4.2)	3 (2.9)	0.385	7 (5.9)	1 (1.0)	0.004*	6 (4.6)	2 (2.2)	0.017*
Moderate[b]	17 (14.4)	13 (12.4)		18 (15.2)[a]	11 (10.5)		11 (7.7)	19 (20.7)	
Mild[c]	31 (26.5)	39 (37.1)		33 (28.0)	38 (36.2)		39 (30)	31 (33.7)	
Normal[d]	65 (55.6)	50 (47.6)		60 (50.8)	55 (52.4)		75 (57.7)	40 (43.5)	
Stunted (combines severe and moderate)	22 (18.6)	16 (15.2)		25 (21.2)	12 (11.4)		17 (13.0)	21 (22.8)	
Underweight[a,e]									
Moderate[b]	5 (5.3)	6 (6.8)	0.89	8 (8.4)	3 (3.4)	0.35	7 (5.4)	4 (7.5)	0.41
Mild[c]	23 (24.2)	22 (25.0)		22 (23.2)	23 (26.1)		30 (22.3)	16 (30.2)	
Normal[d]	67 (70.4)	60 (68.2)		65 (68.4)	62 (70.5)		94 (72.3)	33 (62.3)	
Wasting[a]									
Moderate[b]	4 (3.4)	3 (2.9)	0.18	53 (4.2)	3 (2.9)	0.001**	0	7 (7.6)	0.000**
Mild[c]	20 (16.9)	10 (9.5)		7 (5.9)	22 (21.0)		12 (8.5)	18 (19.6)	
Normal[d]	91 (77.1)	84 (80.0)		96 (81.3)	79 (75.2)		110 (84.6)	65 (70.7)	
Overweight[f]	3 (2.5)	8 (7.6)		10 (8.5)	1 (1.0)		9 (6.9)	2 (2.2)	

*Statistically significant at the level of P < 0.05; ** Statistically significant at the level of P < 0.01

[a] Severe is defined as height-for-age < -3 sd from the WHO 2007 growth reference standard

[b] Moderate is defined as height-for-age/weight-for-age and BMI-for-age between -2 and -3 standard deviations below the median of the WHO 2007growth reference standard

[c] Mild is defined as height-for-age/weight-for-age and BMI-for-age from -1 to -2 standard deviations below the median of the WHO 2007 growth reference standard

[d] Normal is defined as height-for-age and weight-for-age above -1 standard deviations of the median of the WHO 2007 growth reference standard

[e] Overweight is defined as BMI-for-age above 1 standard deviation above the median of the WHO 2007 growth reference standard

[f] Weight-for-age available for children up to 10 years of age: MVP N = 95; control N = 88; male N = 95; female N = 88; 6 to 8 years N = 130; 9 to 13 years N = 53

TABLE 3.25: Prevalence of initial inadequate nutritional status by study group, sex and age at baseline

Indicator	Nutritional status	MVP N = 118	Controls N = 105	Chi-square test: p	Male n = 118	Female n = 105	Fisher's exact test: p	6 to 8 years n = 131	9 to 13 years n = 92	Fisher's exact test: p
Height-for-age	Inadequate[a]	52 (44.1)	50 (47.6)	0.134	57 (48.7)	50 (47.5)	0.513	55 (42.3)	52 (56.5)	0.023*
	Adequate[b]	66 (55.9)	50 (52.4)		61 (51.3)	55 (52.5)		76 (57.7)	40 (43.5)	
Weight-for-age[c]	Inadequate[a]	27 (28.4)	28 (31.8)	0.350	30 (31.3)	25 (28.4)	0.398	36 (27.5)	19 (35.8)	0.172
	Adequate[b]	69 (71.6)	60 (68.2)		66 (68.7)	63 (71.6)		95 (72.5)	34 (64.2)	
BMI-for-age	Inadequate[a]	23 (19.5)	13 (12.4)	0.104	11 (9.3)	25 (23.8)	0.003*	11 (8.4)	25 (27.2)	0.000**
	Adequate[b]	95 (80.5)	92 (87.6)		107 (90.7)	80 (76.2)		120 (91.6)	67 (72.8)	

,*Statistically significant at P value < 0.05; **Statistically significant at P value < 0.001

[a] Height-for-age, weight-for-age and BMI-for-age < -1 standard deviations from the WHO 2007 growth reference standard

[b] Height-for-age, weight-for-age and BMI-for-age > -1 standard deviations from the WHO 2007 growth reference standard

[c] Weight-for-age available for children up to 10 years of age: MVP N = 95; control N = 88; Male N = 95; Female N = 88; 6 to 8 years N = 130; 9 to 13 years N = 53

3.6.7   Full haemogram serum ferritin, CRP and serum retinol concentration by study group, sex and age at baseline

Full haemograms were analysed for a total of 235 children; 118 in the MVP and 117 in the control. Of these, 123 were male and 112 were female, and 130 children were aged between 6 and 9 years while 105 children were aged from 9 to 13 years (TABLE 3.26). Children in the control group had a significantly higher mean Hb (chi-square test: $p < 0.0001$) when compared to those in the MVP group. The mean white blood cell (WBC) and red blood cell (RBC), mean cell haemoglobin (MCH), mean cell haemoglobin concentration (MCHC) and absolute lymphocyte counts (ALC) were also significantly higher in the control group than in the MVP group (ANOVA: $p = 0.026$, 0.017, 0.000, 0.000 and 0.029 respectively). On the other hand, mean cell volume (MCV) was significantly higher (ANOVA: $p = 0.000$) in the MVP group than in the control group. The full haemogram parameters were not significantly different between males and females.

The mean WBC was higher ANOVA: among the younger children than the older ones (ANOVA: $p = 0.02$). The mean Hb and RBC concentrations were significantly higher among the older children when compared to the younger ones (chi-square test: $p = 0.003$ and 0.000). The mean serum ferritin concentration was higher in the control group, at 46.9 µg/l (28.1 SD) [95% CI; 27.9, 43.7] compared to the MVP group, at 35.8 µg/l (25.4 SD) [95% CI; 38.1, 55.6]. This difference was not statistically significant (ANOVA: $p = 0.06$). The mean CRP level was higher among children in the control group at 5.1 (7.5 SD) [95% CI; 2.7, 7.4] than among those in the MVP group which was 3.2 (4.4 SD) [95% CI; 1.8, 4.5] (ANOVA: $p = 0.067$). Children in the MVP group had a significantly higher mean serum retinol concentration at baseline (ANOVA: $p = 0.036$) when compared to children in the control group. Mean serum retinol was not significantly different between gender and age (TABLE 3.26).

TABLE 3.26: Full haemogram parameters, serum ferritin, CRP and serum retinol concentration by study group, gender and age at baseline

Full haemogram parameter	Normal ranges	MVP n = 118	Control n = 117	ANOVA test: p	Male n = 123	Female n = 112	ANOVA test: p	6 to 8 years n = 130	9 to 13yrs n = 105	ANOVA test: p
				Mean (SD) [95% confidence interval]						
WBC (10^3cells/uL)	6 – 14	6.5 (1.7) [6.2, 6.8]	7.0 (1.9) [6.7, 7.3]	0.026*	6.8 (1.8) [6.5, 7.1]	6.7 (1.8) [6.4, 7]	0.62	7.0 (2) [6.6, 7.4]	6.5 (1.4) [6.2, 6.7]	0.02*
RBC (10^6 cells /uL)	4-5.2	4 (0.7) [3.9, 4.1]	4.2 (0.6) [4.1, 4.3]	0.017*	4.1 (0.6) [4.1, 4.3]	4.1 (0.7) [4, 4.2]	0.62	4 (0.6) [3.9, 4.1]	4.3 (0.6) [4.1, 4.4]	0.003**
Hb (g/dL)	11.5-15.5	9.5 (1.6) [9.3, 9.8]	10.5 (1.4) [10.2, 10.7]	0.000**	10.1 (1.5) [9.8, 10.4]	9.9 (1.7) [9.6, 10.2]	0.48	9.7 (1.6) [9.4, 9.9]	10.4 (1.5) [10.1, 10.7]	0.000**
Hct (%)	35-45	33.1 (4.6) [33.4, 44.4]	33 (3.7) [27.5, 38.5]	0.863	33.1 (4.5) [30.6, 41.4]	33.0 (3.9) [30.3, 41.6]	0.98	34.8 (28.5) [29.9, 39.8]	37.4 (32.4) [31.1, 43.7]	0.533
MCV (fL)	77-95	83.9 (10.9) [82.2, 85.4]	78.8 (6.5) [77.1, 80.4]	0.000**	80.7 (8.3) [79.1, 82.4]	82 (10.3) [80.2, 83.7]	0.32	81.3 (9) [79.7, 82.8]	81.4 (9.8) [79.5, 83.3]	0.716
MCH (pg)	23-31	23.4 (3.7) [22.7, 24.0]	24.9 (2.6) [22.4, 25.4]	0.000**	24.1 (3.2) [23.5, 24.7]	24.2 (3.3) [23.6, 24.8]	0.78	24 (3.1) [23.4, 24.5]	24.4 (3.5) [23.7, 25]	0.107
MCHC (g/dL)	28-33	28.3 (3.8) [27.6, 29.5]	31.5 (1.6) [31.2, 31.8]	0.000**	30.1 (2.7) [29.5, 30.7]	29.7 (4) [29.1, 30.3]	0.32	29.6 (3.6) [29, 30.3]	30.2 (3) [29.7, 30.8]	0.059
Plt (10^3/ cells uL)	150-400	282.1 (111.2) [264.3, 303.9]	299.4 (121.1) [277.2, 321.5]	0.257	297.4 (114.3) [278.9, 319.8]	283.3 (118.6) [261.9, 304.7]	0.35	301.3 (113.1) [281.7, 320.9]	279.8 (116.9) [257.2, 302.4]	0.343
ALC (%)	25-50	43.8 (9.9) [43, 46.2]	45 (7.6) [43.6, 46.4]	0.313	43.5 (9.3) [42.3, 45.2]	45.4 (8.2) [44.4, 47.4]	0.10	44.848 (7.7) [43.5, 46.2]	44.649 (8.9) [42.9, 46.4]	0.666
Lymph abs (10^3uL)	5-8.5	2.9 (0.9) [2.7, 3.0]	3.1 (0.9) [3.0, 3.3]	0.029*	3 (1) [2.8, 3.1]	3 (0.8) [2.9, 3.2]	0.61	3.107 (1) [2.9, 3.3]	2.874 (0.8) [2.7, 3]	0.05
Serum ferritin and CRP										
N		42	42		43	41		50	34	
Serum Ferritin (µg/l)	15 - 55	35.8 (25.4) [38.1, 55.6]	46.9 (28.1) [27.9, 43.7]	0.06	41.3 (30.7) [33, 49.6]	41.4 (23.4) [32.9, 49.9]	0.98	42.6 (26.5) [35.1, 50.1]	39.5 (28.5) [29.5, 49.4]	0.99
CRP (mg/l)		3.2 (4.4) [1.8, 4.5]	5.1 (7.5) [2.7, 7.4]	0.163	4.8 (7.2) [2.6, 7.0]	3.3 (4.9) [1.8, 4.9]	0.293	5.1 (7.0) [3.2, 7.1]	2.6 (4.6) [1, 4.2]	0.067
Serum retinol										
N		50	44		48	46		35	59	
Serum retinol (µmol/L)		0.77 (0.22) [0.71, 0.84]	0.68 (0.2) [0.62, 0.74]	0.034*	0.75 (0.21) [0.68, 0.81]	0.72 (0.22) [0.65, 0.78]	0.97	0.73 (0.21) [0.67, 0.78]	0.77 (0.25) [0.69, 0.86]	0.326

* Statistically significant at P value < 0.05; ** statistically significant at P value < 0.01; Note: WBC (white blood cells), RBC (red blood cells), HB (haemoglobin), HCT (haematocrit), MCV (mean cell volume), Fl (femtolitres), MCH (mean cell haemoglobin), MCHC (mean cell haemoglobin concentration), Plt (platelets), Lymphs abs (lymphocytes), ALC (absolute lymphocyte count), CRP (creatinine reactive protein)

3.6.8   Prevalence of anaemia by study group, sex and age at baseline

A very high percentage of children (82.2%) in the MVP were anaemic compared to 58.1% in the control group (chi-square test: p = 0.000; TABLE 3.27). Further, the prevalence of severe and moderate anaemia was higher in the MVP group compared to the control group (7.6% versus 1.7% for severe anaemia and 46.6% versus 28.2% for moderate anaemia, p = 0.000). The proportion of children with microcytic anaemia as defined by MCV < 82 fl was higher among the control group (71.8%) compared to the MVP group (50.6%; chi-square test: p = 0.001). There were no significant differences in the prevalence of anaemia between males and females. More than two thirds (77.7%) of the younger children (6 to 8 years old) were anaemic compared to 61% of the older ones (9 to 13 years; chi-square test: p = 0.004). In addition, 63.4% of the younger children had moderate anaemia compared to 43.8% of the controls (chi-square test: p = 0.013).

3.6.9   Body iron stores and CRP levels by study group, sex and age at baseline

There was a significant difference between the prevalence of depleted body iron stores among the males (23.3%) and the females (7.3%; chi-square test: p = 0.014; TABLE 3.27). Of the 42 children in the MVP with data on CRP levels, 9.5% had elevated CRP levels, compared to 14.3% in the control group. Elevated CRP levels were higher among males (16.3%) than females (7.3%), and among the younger children (16.0%) than the older children (5.9%).

3.6.10 Vitamin A status by study group, sex and age at baseline

Serum retinol data was analysed for a subsample of 94 children (MVP N = 50; control N = 44) (TABLE 3.28). The mean serum retinol was significantly higher among the MVP group (22.2 (SD 6.4) [95% CI 20.4, 24]) than the control group (19.5 (SD 5.6) [95% CI 17.8, 21.2; chi-square test: p = 0. 036]). Mean serum retinol was not significantly different between gender and age groups. Only one child (1.1%) aged 8 years from the control group had vitamin A deficiency (serum retinol <0.35 μmol/L. Half of the children in the control group had marginal vitamin A status (serum retinol 0.35-0.70 μmol/L), which was significantly different (chi-square test: p = 0.044) from the prevalence among the MVP children (30%). The prevalence of marginal vitamin A status was higher among females than males, and higher in younger children in comparison to the older ones. These differences were not statistically significant.

TABLE 3.27: Prevalence of anaemia, depleted body iron stores and CRP levels by study group, gender and age at baseline

Variable	MVP N = 118	Control N = 117	Chi-square test: p	Male N = 123	Female N = 112	Chi-square test: p	6–8 years N = 130	9–13 years N = 105	Chi-square test: p
Anaemia based on haemoglobin[1]					[N (%)]				
Anaemic	97 (82.2)	68 (58.1)	0.000**	86 (69.9)	79 (70.5)	0.484	101 (77.7)	64 (61.0)	0.004*
Not anaemic	21 (17.8)	49 (41.9)		37 (30.1)	33 (29.5)		29 (22.3)	41 (39.0)	
Classification of anaemia based on haemoglobin									
Severe[2]	9 (7.6)	2 (1.7)	0.000**	2 (1.7)	9 (8.1)	0.374	6 (4.6)	5 (4.8)	0.013*
Moderate[3]	55 (46.6)	33 (28.2)		55 (44.7)	33 (29.5)		83 (63.4)	46 (43.8)	
Mild[4]	33 (28)	33 (28.2)		29 (23.6)	37 (33)		12 (9.2)	13 (12.4)	
Microcytic anaemia based on MCV[5]									
Anaemic	60 (50.6)	84 (71.8)	0.001*	78 (63.4)	66 (58.9)	0.337	82 (63.1)	65 (61.9)	0.480
Not anaemic	58 (49.2)	33 (28.2)		45 (36.6)	46 (41.1)		48 (36.9)	40 (38.1)	
Depleted body iron stores based on serum ferritin[6]	N = 42	N = 42		N = 43	N = 41		N = 50	N = 34	
Depleted body iron stores	8 (19.1)	5 (11.9)	0.27	10 (23.3)	3 (7.3)	0.041*	6 (12.0)	7 (20.6)	0.22
Normal stores	34 (81)	37 (88.1)		33 (76.4)	38 (92.7)		44 (88.0)	27 (79.4)	
CRP levels									
Elevated CRP	4 (9.5)	6 (14.3)	0.36	7 (16.3)	3 (7.3)	0.77	8 (16.0)	2 (5.9%)	0.14
Normal CRP	38 (90.5)	36 (85.7)		36 (83.7)	38 (92.7)		42 (84.0)	32 (94.1)	

*Statistically significant at p value < 0.05; *statistically significant at p value < 0.001

Notes: ug/l: micrograms per litre; [1]Classification of anaemia by haemoglobin; Hb < 11.0 g/dl for 6 to 11 years old; and Hb cut-off for 12 to 13 years was Hb < 11.5 g/dl [2]Severe anaemia (all ages): Hb < 7.5 g/dl); [3]Moderate anaemia: Hb 7.5 to 10.4 g/dl (6 to 11 years) and Hb 7.5 to 10.3 (12 to 13 years); [4]Mild anaemia: Hb 10.5 to 10.9 g/dl (6 to 11 years) and Hb 10.4 to 11.4 g/dl (12 to 14 years) ; [5] Not anaemic: Hb ≥ 11.0 g/dl (6 to 11 years) and Hb ≥ 11.5 g/dl (12 to 14 years); [6] Low MCV – mean cell volume < 82 fl; [7]Depleted body iron stores: serum ferritin < 15 ug/l for children aged 5 to 11 years and less than 12 ug/l for children 12 to 14 years; CRP (C-reactive protein)

TABLE 3.28: Vitamin A status by study group, gender and age at baseline

Characteristic	MVP N = 50	Control N = 44	Chi-square test: p	Male N = 48	Female N = 46	Chi-square test: p	6–8 years N = 35	9–13 years N = 59	Chi-square test: p
N	50	44		48	46		35	59	
Serum retinol (μmol/L) Mean (SD) [CI]	22.2 (6.4) [20.4, 24]	19.5 (5.6) [17.8, 21.2]	0.036*[d]	21.4 (6.1) [19.6, 23.2]	20.5 (6.3) [18.6, 22.3]	0.47[d]	20.8 (6.1) [19.2, 22.4]	22.2 (7.1) [19.8, 24.6]	0.326[d]
Vitamin A status; N (%)									
Vitamin A deficiency (Serum retinol < 0.35 μmol/L)	0	1 (2.3)		1 (1.1)	0		1 (1.1)	0	
Marginal vitamin A status (Serum retinol 0.35-0.70 μmol/L)	15 (30.0)	22 (50.0)	0.044*[e]	13 (28.9)	23 (48.9)	0.06[e]	25 (46.3)	11 (28.9)	0.114[e]
Inadequate vitamin A status (Serum retinol < 0.70 μmol/L)	35 (70)	21 (47.7)		31 (68.9)	24 (51.1)		29 (53.7)	26 (68.4)	

* statistically significant at P value < 0.05
[d]P values obtained by ANOVA test
[e]P values obtained by Fisher's exact test

3.7 Determinants of Initial Nutritional Status Based on Anthropometric Indicators

A multivariate stepwise logistic regression was used to identify determinants of inadequate initial nutritional status based on height-for-age, weight-for-age and BMI-for-age z scores < -1 SD. Each of the anthropometric indices grouped in dichotomy of 1 to represent inadequate initial nutritional status and 0 to represent adequate nutritional status was included in a regression model as the dependant variable. The independent variables included in the regression model were the study group, child's age, gender and household socio-demographic characteristics (TABLE 3.29).

Child's age was a significant determinant of low height-for-age and BMI-for-age. Older children aged 9 to 13 years were significantly more likely to have poor nutritional status based on height-for-age (OR = 3.4; logistic regression; p = 0.037) and BMI-for-age (OR = 3.7; logistic regression; p = 0.006) compared to the younger children (6 to 8 years). In addition, children whose mothers were unemployed were 4.4 times and significantly more likely (logistic regression; p = 0.031) to have poor nutritional status based on height-for-age compared to those whose mothers were employed. Children from households with a total income of less than KES 1,000 per month were 1.3 times more likely to have a low height-for-age compared to those from households earning more than KES 1,000 per month. This difference was not statistically significant (TABLE 3.29).

TABLE 3.29: Adjusted odds of inadequate nutritional status by socio-demographic characteristics at baseline

Characteristics	Height-for-age			Logistic regression; p	Weight-for-age		Logistic regression; p	BMI-for-age		Logistic regression; p
	N	OR	95% confidence interval		OR	95% confidence interval		OR	95% confidence interval	
Study group										
MVP	97	2.6	0.8, 2.6	0.218	0.9	0.4, 1.9	0.809	0.6	0.2, 1.4	0.231
Control (rc)	113	1.0			1.0			1.0		
Sex of child										
Male	104	2.5	0.8, 2.5	0.246	1.1	0.5, 2.2	0.854	0.5	0.2, 1.3	0.154
Female (rc)	106	1.0			1.0			1.0		
Age of child (years)										
9 to13	87	3.4	1, 3.4	0.037*	1.6	0.8, 3.3	0.228	3.7	1.5, 9.3	0.006*
6 to 8 (rc)	123	1.0			1.0			1.0		
Head of household										
Mother and others	59	1.7	0.4, 1.7	0.615	0.8	0.3, 1.9	0.651	1.2	0.4, 3.3	0.771
Father (rc)	151	1.0			1.0			1.0		
Employment of head of household										
Not employed	76	2.1	0.6, 2.1	0.703	1.1	0.5, 2.2	0.895	1	0.4, 2.6	0.976
Employed (rc)	134	1.0			1.0			1.0		
Maternal education										
None	13	1.6	0.1, 1.6	0.206	0.7	0.1, 3.6	0.657	2.9	0.7, 12.2	0.149
Secondary	41	0.9	0.3, 2.8	0.685	1.2	0.5, 2.9	0.63	1.8	0.6, 5.3	0.309
Primary (rc)	156	1.0			1.0			1.0		
Maternal marital status										
Single	50	2.7	0.7, 2.7	0.416	0.9	0.4, 2.2	0.85	0.4	0.1, 1.3	0.117
Married (rc)	160	1.0			1.0			1.0		
Maternal employment status										
None	55	4.4	1.1, 4.4	0.031*	1.8	0.8, 4	0.163	1.9	0.7, 5.7	0.22
Employed (rc)	155	1.0			1.0			1.0		
Total household income/month										
More than KES 1,000	97	1.3	0.7, 2.4	0.46	1	0.5, 2.3	0.919	1.5	0.6, 4.1	0.397
Less than KES 1,000 (rc)	113	1.0			1.0			1.0		

rc: reference category; *statistically significant at P < 0.05; total number of subjects = 210, logistic model excludes 10 subjects with deceased mothers

3.8   Determinants of Anaemia, Body Iron Stores and Vitamin A Status at Baseline

The determinants of anaemia, low body iron stores and inadequate vitamin A status were identified with a multivariate logistic regression model. Age, study group, linear growth deficit and wasting were significant determinants of anaemia. The odds of being anaemic were higher among younger children aged 6 to 8 years (OR = 1.0) than older ones (9 to 13 years; OR = 0.5; logistic regression; p = 0.019; TABLE 3.30). Children in the MVP group were 2.8 times more likely to be anaemic compared to those in the control group (p = 0.001), while children with mild to moderate stunting and wasting were 1.9 times and 5.1 times more likely to be anaemic (logistic regression; p = 0.046 and 0.007) compared to those with a height-for-age and BMI-for-age z-score > -1 SD respectively (Table 3.20). Although not significant, males tended to be 4.4 times more likely to have depleted body iron stores than females. Study group, employment status of the household head, household income and BMI-for-age were significant determinants of vitamin A status.

Children in the MVP group were 0.2 times and significantly (logistic regression; p = 0.005) less likely to have inadequate vitamin A status compared to their control counterparts. Those from households with unemployed household heads were 3.4 times and significantly (logistic regression; p = 0.041) more likely to have inadequate vitamin A status compared to those from households with an employed household head. In addition, children in households with a total monthly household income less than KES 1,000 were significantly more likely (4.6 times) to have inadequate vitamin A status (logistic regression; p = 0.014) compared to those in households with a total monthly income above KES 1,000 (TABLE 3.30).

TABLE 3.30: Adjusted odds of anaemia, depleted body iron stores and inadequate vitamin A status by anthropometric indicators and socio-demographic characteristics at baseline

Characteristic	Anaemia				Depleted body iron stores				Inadequate Vitamin A status			
	N[a]	OR	CI	Logistic regression; p	N	OR	CI	Logistic regression; p	N	OR	CI	Logistic regression; p
Age of child (years)												
9 to 13	87	0.5	0.2, 0.9	0.019*	32	0.7	0.2, 3.2	0.689	32	0.4	0.1, 1.4	0.152
6 to 8 (rc)	123	1.0			48	1.0			58	1.0		
Height-for-age												
≤ -1 SD	89	1.9	1, 3.6	0.046*	31	2.1	0.5, 10.1	0.339	35	2	0.6, 6.5	0.241
> -1 SD (rc)	121	1.0			49	1.0			55	1.0		
BMI-for-age												
≤ -1 SD	27	5.1	1.6, 16.3	0.007*	10	0.8	0.1, 8.9	0.887	10	0.1	0, 0.9	0.034*
> -1 SD (rc)	183	1.0			70	1.0			80	1.0		
Study group												
MVP	113	2.8	1.5, 5.2	0.001*	41	2.1	0.5, 9.4	0.336	49	0.2	0.1, 0.6	0.005*
Control (rc)	97	1.0			39	1.0			41	1.0		
Sex of child												
Male	104	0.7	0.4, 1.3	0.307	37	4.4	0.9, 21	0.063	43	0.6	0.2, 1.8	0.353
Female (rc)	106	1.0			43	1.0			47	1.0		
Head of household												
Mother and others	26	0.6	0.2, 1.6	0.307	12	2	0.2, 17.1	0.53	12	1.6	0.3, 8.9	0.604
Father (rc)	184	1.0			68	1.0			78	1.0		
Employment of head of household												
Not employed	76	1.1	0.6, 2.2	0.69	32	1.7	0.3, 8.6	0.501	35	3.4	1.1, 11	0.041*
Employed (rc)	134	1.0			48	1.0			55	1.0		
Mother's education												
None	13	1.3	0.3, 5.4	0.709	4	0.3	0,5	0.412	4	0.2	0, 4.1	0.289
Secondary	41	0.7	0.3,1.7	0.472	19	0.6	0.1, 3.2	0.551	21	0.7	0.2, 2.4	0.555

rc: reference category; [a]model excludes, for anaemia, 10 children with deceased mothers, and for depleted body iron stores and vitamin A status, 4 children whose mothers were deceased; weight-for-age is excluded because data is only available for children < 10 years; *statistically significant at P < 0.05

TABLE 3.30 Adjusted odds of anaemia, depleted body iron stores and inadequate vitamin A status by anthropometric indicators and socio-demographic characteristics at baseline (Continued)

Characteristic	Anaemia				Depleted body iron stores				Inadequate Vitamin A status			
	N[a]	OR	CI	Logistic regression; p	N	OR	CI	Logistic regression; p	N	OR	CI	Logistic regression; p
Primary(rc)	156	1.0			57	1.0			65	1.0		
Mother's marital status												
Married	50	1.8	0.9, 3.9	0.123	24	3.4	0.7, 15.9	0.121	24	2.5	0.7, 8.5	0.143
Single (rc)	160	1.0			56	1.0			66	1.0		
Mother's employment status												
None	55	0.9	0.4, 2	0.839	18	1.5	0.3, 8.7	0.636	23	1.2	0.4, 4.1	0.774
Employed (rc)	155	1.0			62	1.0			67	1.0		
Total household income/month												
Less than KES 1 000	113	1.0	0.5, 1.9	0.906	47	0.6	0.1,3	0.559	55	4.6	1.4, 15.6	0.014*
More than KES 1 000 (rc)	97	1.0			33	1.0			35	1.0		

rc: reference category; [a]model excludes, for anaemia, 10 children with deceased mothers, and for depleted body iron stores and vitamin A status, 4 children whose mothers were deceased; weight-for-age is excluded because data is only available for children < 10 years; *statistically significant at $P < 0.05$

3.9   Summary of Baseline Findings

A majority of the children lived in male-headed households. A majority of the mothers had attained a primary level of education. The socioeconomic characteristics of the two study groups were comparable at baseline, except for household headship, in terms of which the percentage of households headed by mothers and grandparents was higher in the control group than in the MVP group.

In general, the dietary intake of the children lacked in variety, with predominantly staple starches, and a limited intake of milk, fruit, vegetables and total fats. The probability of inadequate nutrient intake was high for energy, total fat, folate, calcium, selenium and zinc. The groups were similar in the levels of nutrient intake at baseline and in the prevalence of inadequate nutrient intake.

Stunting, underweight and wasting were found among 16.6%, 6% and 3.2% of the children respectively. Inadequate nutritional status, defined by height-for-age, weight-for-age and BMI-for-age z-scores < -1 SD was observed in 48%, 30% and 16% of the children respectively. Five percent of the children were overweight at baseline. The study groups were comparable in nutrition outcomes at baseline. Stunting was higher among the males and older children, while wasting was higher among the females and older children. Children's age was a significant determinant of inadequate nutritional status based on low height-for-age and BMI-for-age z-scores, where younger children were more likely to be thin and have linear growth deficit compared to older children. Maternal employment status was a determinant of linear growth deficit among the children. Children whose mothers were not self-employed or in any form of employment were more likely to have height-for-age < -1 SD compared to children whose mothers were in any form of employment.

Anaemia was highly prevalent (70.2%) in this population, with a significantly higher prevalence among the MVP group compared to the controls, and higher among younger children than older ones. Children in the control group had a significantly higher prevalence of microcytic anaemia (low MCV and low HB) compared to children from the intervention group. Household headship was associated with anaemia, with a higher prevalence among children living in households headed by mothers or other relatives compared to children in households headed by fathers. Age, linear growth deficit and study group were significant determinants of anaemia at baseline. Younger children had higher odds of being anaemic compared to older children, while children in the intervention group were more likely to be anaemic compared to the control group. A height-for-age

z-score < -1 SD increased the odds of being anaemic compared to a higher height-for-age z-score. depleted body iron stores were more common among the males than the females. Hb and RBC levels were lower in children with inadequate nutritional status at baseline.

Inadequate vitamin A status was observed among 39.4% of the population and was more common in the control group. Employment status of the household head was associated with vitamin A status, with a higher prevalence of inadequate status among children staying in households with unemployed household heads. Children from households with a total monthly income of less than KES 1,000 per month were more likely to have inadequate vitamin A status. Multiple micronutrient deficiencies were found in this population, with 8% of the children having iron deficiency combined with inadequate vitamin A status.

3.10   Section B: Results Following the Intervention

The school feeding programme in the MVP was started in June 2006. The school feeding programme (SFP) in the control group was introduced in January 2007 (6 months into the study), with a basic feeding intervention (maize and beans – *githeri*), as described in Chapter 2, section 2.7.2. The introduction of the SFP in the control group was a result of a scale-up programme within the Millennium Villages Project to expand the project area to other areas within Yala Division, where the MVP is located. Part 1 of this section shows the findings on the anthropometric indicators at 6 months compared with baseline for the two study groups. The reason for presenting the findings in this way is because, although the control group had not been participating in the SFP from baseline, all data was collected for them at baseline, as presented in Section A, Part 3 above. This comparison at 6 months provides perspective on the nutritional status of the control group at the start of the feeding programme. Part 2 presents the findings on the trends in growth, haemoglobin concentration, serum ferritin and serum retinol concentration. Part 3 contains findings on the impact of the SFP on nutritional status and body composition based on anthropometric measurements, as well as the impact on anaemia, body iron stores and vitamin A status.

3.11   Part 1: Results of Anthropometric Indicators at Six Months

Part 1 of section B presents the findings on the anthropometric indicators at 6 months compared with baseline for the two study groups.

3.11.1 Anthropometry at six months

Repeated measures ANOVA was done to compare anthropometric measurements at baseline and at the 6th month interval in each of the two groups and between the groups (TABLE 3.31). As expected, growth was observed among all the children between baseline and 6 months. In the MVP

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group, the anthropometric measurements and indicators that increased significantly between baseline and 6 months were: height (ANOVA: p = 0.000), weight (ANOVA: p = 0.000), MUAC (ANOVA: p = 0.005), triceps (ANOVA: p = 0.000), percent body fat (ANOVA: p = 0.000), fat mass (ANOVA: p = 0.000), MUAFA (ANOVA: p = 0.000), and fat-free mass (ANOVA: p = 0.000).

There was a significant increase in height (ANOVA: p = 0.000), weight (ANOVA: p = 0.000), percent body fat (ANOVA: p = 0.000), fat mass (ANOVA: p = 0.001), MUAFA (ANOVA: p = 0.03), and fat-free mass (ANOVA: p = 0.000) from baseline to 6 months among children in the control group. There was a significant decline in the height-for-age z scores for both the MVP and the control group (ANOVA: p = 0.003 and 0.007 respectively). Unlike in the MVP group, the MUAC and triceps skinfold measurements did not change significantly in the control group (TABLE 3.31). The F test to compare the two groups at baseline and 6 months showed significant interactions of the mean weight F(1, 218) = 9.21, p = 0.003], height [F (1, 218) = 4.9, p = 0.0003] and fat-free mass [F(1, 218) = 9.96, p = 0.002] (TABLE 3.31). These interactions are explained by the changes observed in individual groups from baseline to 6 months, rather than differences between groups, because the Bonferroni post hoc test did not reveal any significant differences between groups in any of the anthropometric indicators at the 6-month interval (TABLE 3.31).

TABLE 3.31: Growth and body composition measurements by study group at baseline and 6 months

Growth and body composition measurement		MVP (N = 118)			Control (N = 102)			Repeated measures ANOVA; F statistics	Bonferroni post hoc test: p[b]
		Mean (SD)	95%CI	Bonferroni post hoc test: p[a]	Mean (SD)	95%CI	Bonferroni post hoc test: p[a]		
Height (cm)	Baseline	123.6 (11.6)	121.5, 125.7	0.000**	122.8 (11.1)	120.7, 125.0	0.000**	$F(1, 218)=13.8$,	
	6 months	125.1 (11)	123.1, 127.1		125 (10.4)	122.9, 127		$p = 0.0003$**	1.0
Weight (kg)	Baseline	23.5 (4.9)	22.6, 24.4	0.000**	24.0 (5.0)	23.0, 25.0	0.000**	$F(1, 218) = 9.21$,	
	6 months	25.2 (5.2)	24.2, 26.1		25 (4.9)	24.0, 25.9		$p = 0.003$*	1.0
HAZ	Baseline	-0.6 (0.9)	-0.7, -0.4	0.03*	-0.7 (1.4)	-1.0, -0.4	0.007*	$F(1, 218) = 1.60$,	
	6 months	-0.7 (1.2)	-0.9, -0.4		-0.8 (1.4)	-1.1, -0.5		$p = 0.21$	1.0
WAZ	Baseline	-0.6 (0.9)	-0.7, -0.4	0.87	-0.5 (1.1)	-.07, -0.2	1.0	$F(1, 169)=.92, p=0.34$	
	6 months	-0.4 (0.8)	-0.5, -0.2		-0.4 (1.1)	-0.6, -0.2			1.0
BMI-AZ	Baseline	-0.3 (0.8)	-0.5, -0.1	0.55	-0.1 (0.8)	-0.3, 0.0	1.0	$F(1, 218)=3.4, p=0.06$	
	6 months	-0.1 (0.8)	-0.3, 0.0		-0.1 (0.8)	-0.3, 0			1.0
MUAC (cm)	Baseline	17.4 (1.5)	17.1, 17.7	0.005*	17.4 (1.4)	17.2, 17.7	0.53	$F(1, 217)=1.1, p=0.30$	
	6 months	17.7 (1.7)	17.4, 18.0		17.6 (1.3)	17.3, 17.8			1.0
Triceps (mm)	Baseline	4.5 (1.2)	4.3, 4.7	0.000**	4.6 (1.2)	4.4, 4.9	1.0	$F(1, 218)=2.39, p=0.12$	
	6 months	5.3 (1.6)	5.0, 5.6		5.0 (1.4)	4.7, 5.3			1.0
Subscapular (mm)	Baseline	4.3 (1.2)	4.1, 4.6	0.47	4.2 (1)	4, 4.4	0.93	$(1, 218)=0.02, p=0.89$	
	6 months	4.5 (1.4)	4.2, 4.7		4.3 (1)	4.1, 4.5			1.0
Body fat (%)	Baseline	7.5 (2.3)	7.1, 7.9	0.000**	7.4 (2.3)	7, 7.9	0.000**	$F(1, 218)=2.49, p=0.12$	
	6 months	9.9 (2.8)	9.4, 10.4		10.3 (2.9)	9.7, 10.8			1.0
Fat mass (kg)	Baseline	1.8 (0.8)	1.7, 2.0	0.000**	1.8 (0.7)	1.7, 1.9	0.001*	$F(1, 218)=1.79, p=0.18$	
	6 months	2.2 (1.1)	2.2, 2.4		2 (0.9)	1.8, 2.2			1.0
MUAFA (mm)^2	Baseline	405 (151.7)	377.3, 432.6	0.000**	395.9 (103.8)	375.5, 416.3	0.03*	$F(1, 217)=1.41, p=0.26$	
	6 months	455.3 (176.0)	423.1, 487.6		427.8 (132.4)	405.8, 453.8			1.0
MUAMA (mm)^2	Baseline	2 044.6 (371.7)	1 976.8, 2112.3	0.96	2 052.9 (359.7)	1 982.3, 2 123.6	1.0	$F(1, 217)=0.45, p=0.50$	
	6 months	2 076.5 (388.5)	2 005.4, 2147.6		2 062.2 (314.7)	2 000.4, 2 124.0			1.0
Fat-free mass (kg)	Baseline	22.2 (4.6)	21.4, 23.1	0.000**	22.2 (4.7)	21.3, 23.1	0.000**	$F(1, 218)=9.96$,	
	6 months	23(4.7)	22.2, 23.9		23 (4.4)	22.1, 23.8		$p=0.002$*	1.0

HAZ: height-for-age z-score, WAZ: weight-for-age z-score, BMIZ: BMI-for-age z-score, MUAC: mid-upper arm circumference, MUAFA: mid-upper arm fat area, MUAMA: mid-upper arm muscle area, *significant at $P < 0.05$; **significant at $P < 0.001$
[a] Bonferroni post hoc test explores differences between baseline and 6 months within individual groups
[b] Bonferroni post hoc test for differences between groups at 6 months

3.12   Part 2: Results of Trends in Growth, Haemoglobin, Serum Ferritin and Serum Retinol
        Concentration Following the Intervention

Part 2 presents the findings on the trends in growth, haemoglobin concentration, serum ferritin and serum retinol concentration. Trends in growth are presented over a 30 months study period while the trends of haemoglobin, serum ferritin and serum retinol concentration are presented for a period of 24 months.

3.12.1 Trends in height

Figure 3.3 shows the mean height for the two groups of children compared from baseline to 30 months. The mean height from baseline to 30 months differed significantly over the 30-month study period [$F_{(5, 1090)} = 2.7846$, $p = 0.016$]. The Bonferroni post hoc test showed significant gains in each of the individual groups (Bonferroni p values < 0.0001), but no significant differences were observed in the mean height between the two groups at any time interval.
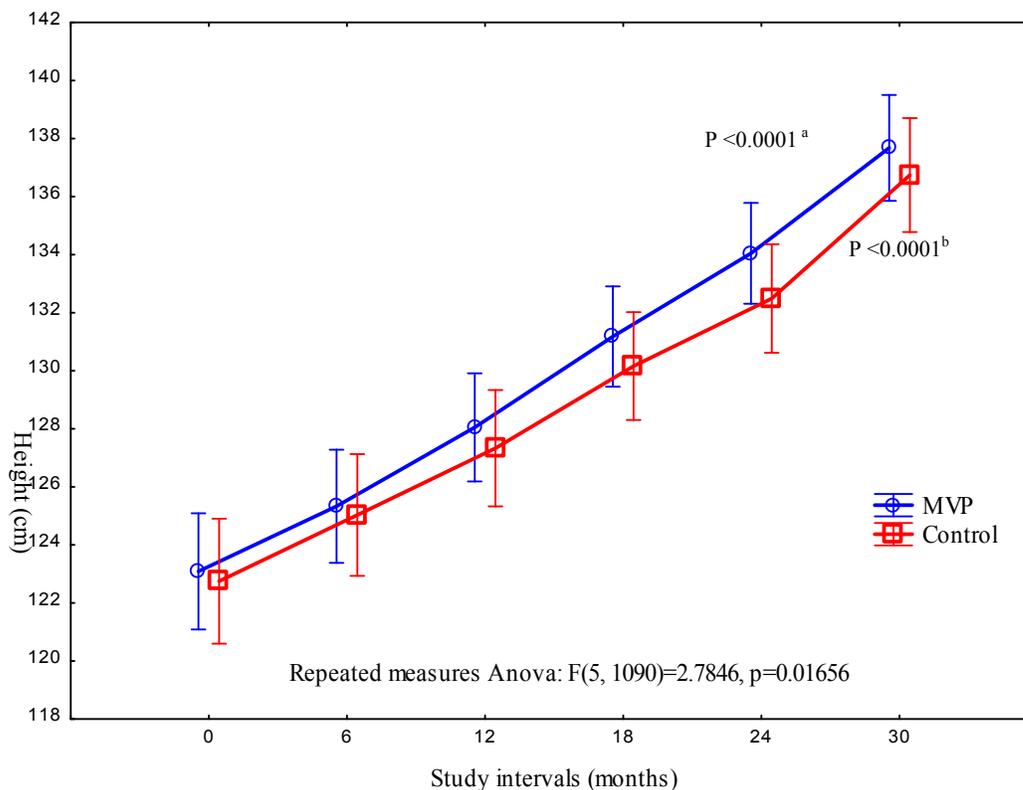
FIGURE 3.3: Trend in mean height by study group and time

Notes: Vertical bars denote 95% CI; P values were obtained by Bonferroni post hoc test; [a] All p values comparing the mean height between each data point for the MVP were < 0.0001; [b] All p values comparing mean height between each data point for the control group were < 0.0001; P values comparing the two study groups at each data point showed no statistically significant differences (all p values = 1.0)

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3.12.2 Trends in height velocity

The mean height velocity increased steadily for all the children from 6 months to 30 months (Figure 3.4). A significant interaction $[F_{(4, 872)} = 3.0017, p = 0.01781]$ was observed between the mean height velocity and the study groups with time. The significant changes occurred from the 18[th] month up to the 30[th] month, when children in the MVP group showed more accelerated height velocity compared to the control group children (Bonferroni; p = 0.01, 0.000 and 0.001 for 18, 24 and 30 months respectively; Figure 3.4).
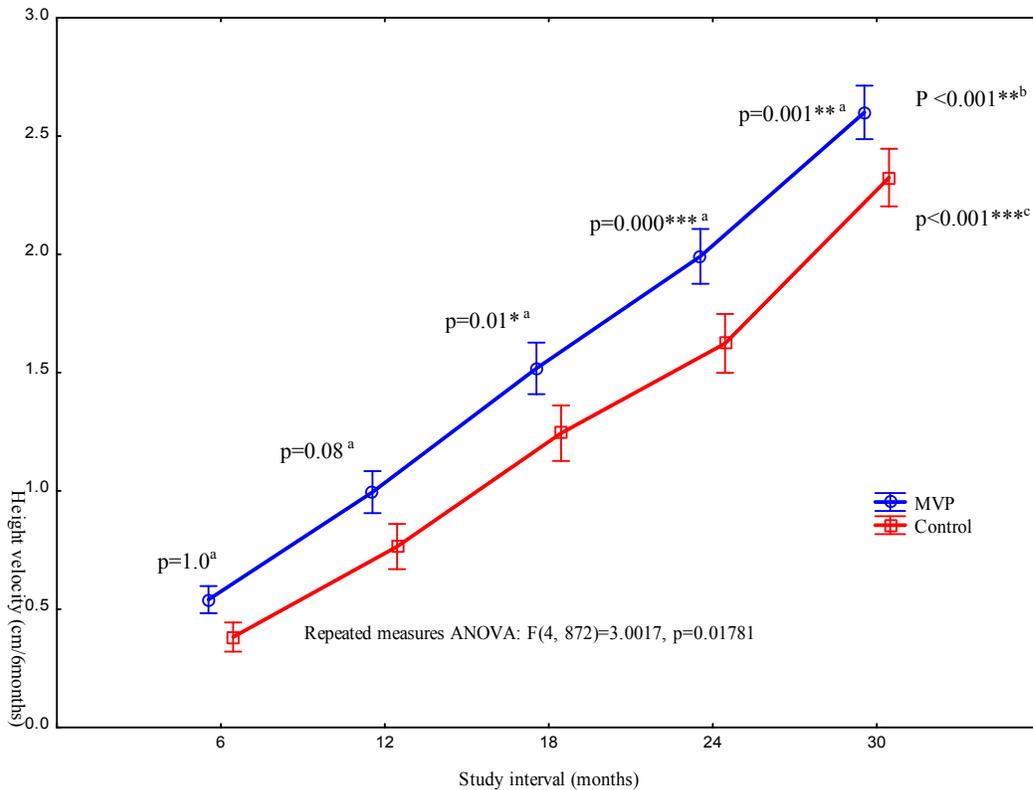
FIGURE 3.4: Trend in mean height velocity by study group and time

Vertical bars denote 95% CI; P values determined by Bonferroni post hoc test. [a] P values compares the mean height velocity between the two study groups at each interval. *Statistically significant at p values < 0.05; ** Statistically significant at p values < 0.005; *** Statistically significant at p values < 0.0001. [b]P values comparing change between each data point for MVP group were all statistically significant at the level < 0.001. [c]P values comparing change between each data point for control group were all statistically significant at the level < 0.001.

3.12.3 Trends in mean weight

Analysis by repeated measures ANOVA over 6 intervals of anthropometric measurements showed significant interactions between the mean weights and time by study group $[F_{(5, 1090)} = 2.48, p = 0.03]$ (Figure 3.5). In the MVP group, the mean weight was significantly higher at the 6[th] month time point compared to the baseline (Bonferroni; p = 0.000). The mean weight was also significantly higher in the 18[th] month time point compared to the 12[th] month and in the 30[th] month

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time point compared to the 24[th] month time point (Bonferroni; p = 0.000, 0.000 and 0.000 for 6[th], 12[th] and 30[th] month time points respectively). Of interest to note in the control group is that the mean weight was significant at the 12[th] month compared to the 6[th] (Bonferroni; p value = 0.000), considering that the school feeding programme was started among the controls in the 6th month. On the contrary, the mean weight of the children in the MVP was not significant in the 12[th] month compared to the 6[th] month.
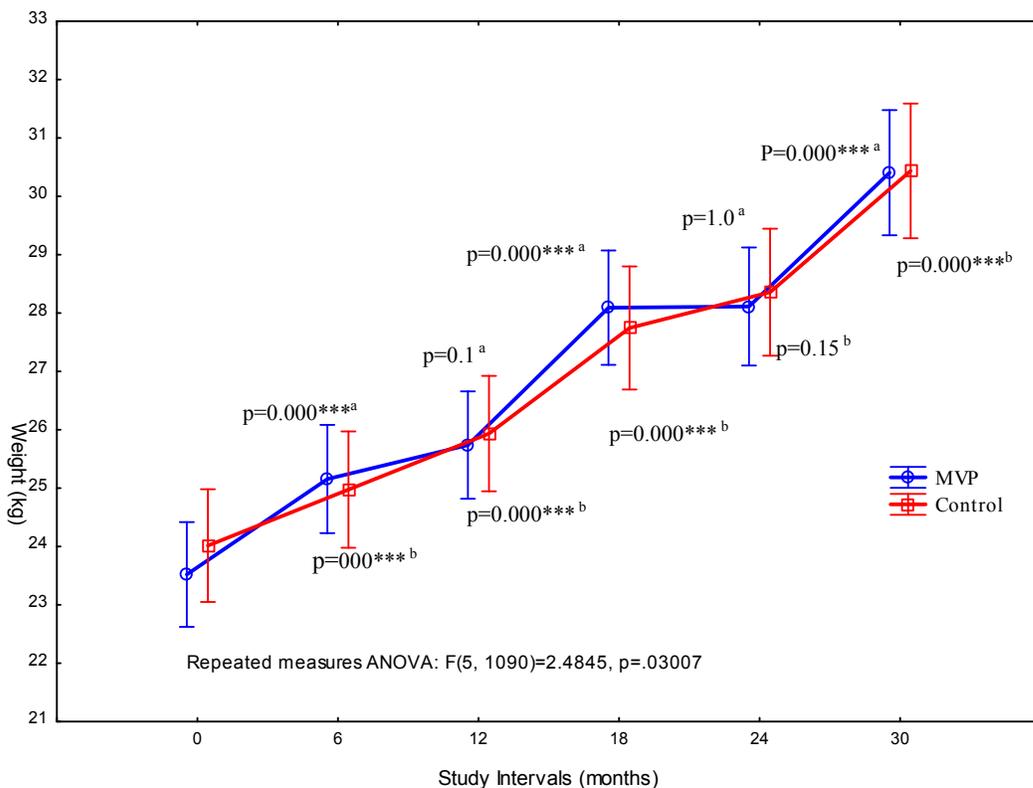
FIGURE 3.5: Trend in mean weight by group and time

Notes: Vertical bars denote 95% CI; P values were obtained by Bonferroni post hoc test; [a] p values compare the mean weight between each data point for the MVP; [b]p values compare mean weight between each data point for the control group. ***Significant differences at p values < 0.0001

3.12.4 Trends in weight velocity

Weight velocity was compared between the 6[th] month and the 30[th] month of the study (Figure 3.6). There was no significant interaction between weight velocity and the study groups over time [$F_{(4,872)} = 1.5$, p = 0.2]. The mean weight velocity also did not differ significantly between the two groups over time. However, significant changes were observed within each group separately. In the MVP group, weight velocity was significantly higher by the 18[th] month (Bonferroni; p = 0.000) and by the 30[th] month (Bonferroni; p = 0.000). Weight velocity was significant for the control children in the 6[th] (Bonferroni; p = 0.000), 18[th] (p = 0.000) and 30[th] month (Bonferroni; p = 0.000).

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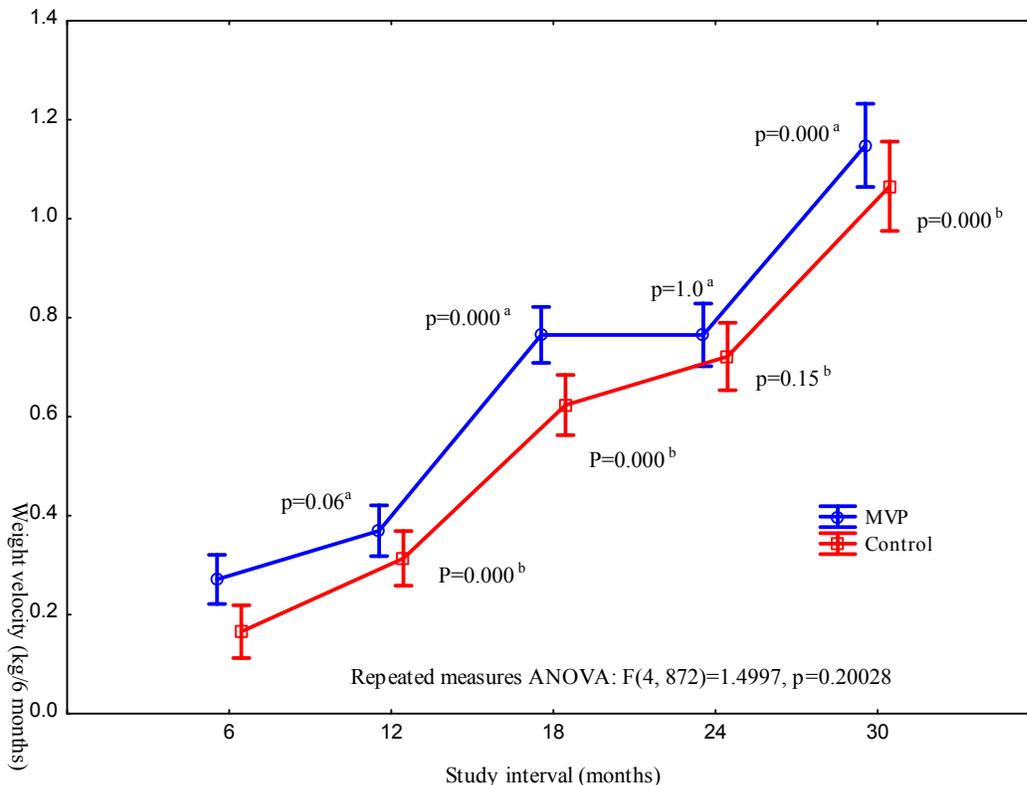
FIGURE 3.6: Trends in mean weight velocity by group and time

Notes: Vertical bars denote 95% CI; P values were obtained by Bonferroni post hoc test; [a] p values compare the mean weight velocity between each data point for the MVP; [b]p values compare the mean weight velocity between each data point for the control group.***Significant differences at p values < 0.0001

3.12.5 Changes in growth and body composition measurements

Table 3.32 presents the change in mean anthropometric measurements over time by study group. Height gain by the 18[th], 24[th] and 30[th] months of the study was significant in the MVP group compared to the control group (Bonferroni; p values = 0.008, < 0.0001, 0.02). The changes in the mean triceps, percent body fat, fat mass, fat-free mass and mid-upper arm fat area showed an increasing trend from baseline, although the differences were not statistically significant between groups. The mid-upper arm muscle area declined between baseline and 6 months, with a higher decline in the control (-28 mm^2) compared to the MVP group (-9 mm^2; TABLE 3.32).

TABLE 3.32: Change in anthropometric measurements by group and time

Measurement and timeline	MVP		Control		
	Mean[a] (sd)	95% CI	Mean (sd)	95% CI	Bonferroni test: p
Weight (kg)					
6 months	1.6 (1.9)	1.3, 2.0	1.0 (1.3)	0.7, 1.2	0.7
12 months	2.2 (2.0)	1.9, 2.6	1.9 (1.3)	1.7, 2.2	1.0
18 months	4.6 (2.0)	4.2, 4.9	3.7 (1.7)	3.4, 4.1	0.1
24 months	4.6 (2.3)	4.2, 5.0	4.3 (1.8)	4.0, 4.7	1.0
30 months	6.9 (3.0)	6.3, 7.4	6.4 (2.4)	5.9, 6.9	1.0
Height (cm)***					
6 months	3.2 (2.2)	2.8, 3.6	2.3 (1.5)	2.0, 2.6	1
12 months	6.0 (3.3)	5.4, 6.5	4.6 (2.5)	4.1, 5.1	0.09
18 months	9.1 (3.8)	8.4, 9.8	7.4 (3.4)	6.7, 8.1	0.008**
24 months	11.9 (4.2)	11.2, 12.7	9.7 (3.4)	9.1, 10.4	0.000***
30 months	15.6 (4.2)	14.8, 16.3	14.0 (3.1)	13.4, 14.6	0.02*
MUAC (cm)					
6 months	0.3 (0.9)	0.1, 0.4	0.2 (0.9)	0, 0.3	1.0
12 months	0.2 (0.9)	0, 0.3	0.2 (0.8)	0, 0.3	1.0
18 months	0.8 (1.1)	0.6, 1	0.7 (1)	0.5, 0.9	1.0
24 months	0.9 (1.1)	0.7, 1.1	0.8 (1)	0.6, 1	1.0
30 months	1.4 (1.4)	1.1, 1.6	1.5 (1.5)	1.2, 1.8	1.0
Triceps (mm)					
6 months	0.5 (1.4)	0.3, 0.8	0.3 (1.2)	0.1, 0.6	1.0
12 months	1.0 (1.4)	0.8, 1.3	1.2 (1.5)	0.9, 1.5	1.0
18 months	1.2 (1.5)	0.9, 1.5	1.5 (1.5)	1.2, 1.8	1.0
24 months	1.6 (1.4)	1.4, 1.9	1.6 (1.7)	1.3, 2.0	1.0
30 months	1.4 (1.7)	1.1, 1.7	1.7 (1.7)	1.4, 2.0	1.0
Subscapular (mm)					
6 months	0.1 (1.4)	-0.1, 0.4	0.2 (0.9)	0, 0.4	1.0
12 months	0.7 (1.2)	0.5, 0.9	0.9 (0.9)	0.7, 1.1	1.0
18 months	0.5 (1.2)	0.2, 0.7	0.7 (1.0)	0.5, 0.9	1.0
24 months	0.4 (1.3)	0.2, 0.6	0.7 (1.1)	0.5, 0.9	1.0
30 months	0.7 (1.4)	0.4, 0.9	0.9 (1.0)	0.7, 1.1	1.0
Body fat (%)					
6 months	0.8 (2.2)	0.4, 1.2	0.6 (1.8)	0.2, 0.9	1.0
12 months	1.6 (2.1)	1.3, 2.0	2.2 (1.9)	1.8, 2.6	1.0
18 months	2.3 (2.3)	1.9, 2.7	2.7 (2.1)	2.3, 3.1	1.0
24 months	2.4 (2.5)	1.9, 2.8	2.7 (2.3)	2.2, 3.1	1.0
30 months	2.3 (2.8)	1.8, 2.8	3.0 (2.3)	2.5, 3.4	1.0
Fat mass (kg)					
6 months	0.3 (0.7)	0.2, 0.4	0.2 (0.5)	0.1, 0.3	1.0
12 months	0.6 (0.7)	0.5, 0.7	0.7 (0.6)	0.6, 0.8	1.0
18 months	0.9 (0.8)	0.7, 1.0	0.8 (0.7)	0.7, 1.0	1.0
24 months	0.9 (0.8)	0.8, 1.1	1.0 (0.8)	0.8, 1.2	1.0
30 months	1.4 (1.2)	1.2, 1.6	1.4 (0.9)	1.2, 1.5	1.0
Mid-upper arm muscle area (mm^2)					
6 months	29.6 (228)	-12.2, 71.3	10.6 (219.5)	-33.2, 54.4	1.0
12 months	-8.9 (200.4)	-45.6, 27.8	-28 (190.7)	-66, 10.1	1.0
18 months	118.7 (262.3)	70.6, 166.7	52.5 (226)	7.4, 97.6	1.0
24 months	108.8 (272.8)	58.8, 158.7	78.8 (256.3)	27.7, 130	1.0
30 months	246.7 (337.9)	184.8, 308.6	265.1 (429.8)	179.3, 350.8	1.0

*Significant at P < 0.05; **Significant at P < 0.005; ***Significant at P < 0.001; [a] mean change from the shown data point to the preceding data point

TABLE 3.32 Change in anthropometric measurements by group and time (Continued)

Measurement and Timeline	MVP		Control		
	Mean[a] (sd)	95% CI	Mean (sd)	95% CI	Bonferroni test: p
Mid-upper arm fat area (mm^2)					
6 months	50.2 (123.5)	27.6, 72.9	30.4 (103.6)	9.8, 51.1	1.0
12 months	88 (126.9)	64.7, 111.2	103.1 (132.7)	76.6, 129.5	1.0
18 months	120.8 (142)	94.8, 146.8	136 (150)	106.1, 165.9	1.0
24 months	157.3 (133.3)	132.9, 181.7	157.3 (153.8)	126.6, 187.9	1.0
30 months	152 (169.2)	121, 183	184.1 (169.2)	150.4, 217.9	1.0
Fat free mass (kg)					
6 months	1.3 (1.6)	1.0, 1.6	0.7 (1.2)	0.5, 1.0	1.0
12 months	1.6 (1.7)	1.3, 1.9	1.2 (1.1)	1.0, 1.4	1.0
18 months	3.0 (2.3)	2.6, 3.4	2.2 (2.2)	1.8, 2.7	1.0
24 months	2.8 (2.6)	2.4, 3.3	2.6 (2.4)	2.1, 3.1	1.0
30 months	5.9 (2.8)	5.4, 6.4	5.2 (2.3)	4.7, 5.6	1.0

*Significant at $P < 0.05$; **Significant at $P < 0.005$; ***Significant at $P < 0.001$; [a] mean change from the shown data point to the preceding data point

3.12.6 Trends of full haemogram parameters and serum ferritin by study group and time

The mean Hb concentration increased significantly (ANOVA: $p = 0.000$) in the MVP children from baseline to 6 months and from 6 months to 24 months (Bonferroni; $p = 0.000$; Figure 3.7). Children in the MVP group had a significantly lower Hb concentration at baseline (Bonferroni; $p = 0.000$) compared to the control group. This trend changed at the 6-month interval, when children in the MVP group had a significantly higher mean Hb concentration (Bonferroni; $p = 0.000$) compared to the control group (Figure 3.7). The mean Hb was not significantly different between the two groups at 24 months. Repeated measures ANOVA did not show significant interaction between ferritin levels by time and study groups. There was a reduction in the mean serum ferritin levels in the 6[th] month and an increase in the 24[th] month for both groups (TABLE 3.33).

A significant interaction was found between white blood cell count, red blood cell count, mean cell volume, mean cell haemoglobin, and mean cell haematocrit count and study groups and time (TABLE 3.33). The white blood cell count was significantly lower among the control children in the 6[th] and 24[th] months of the study (Bonferroni; p values = 0.000) compared to baseline. The mean cell volume was also significantly lower at the 6[th] and 24[th] month intervals for the MVP group compared to baseline (Bonferroni; p values = 0.000).
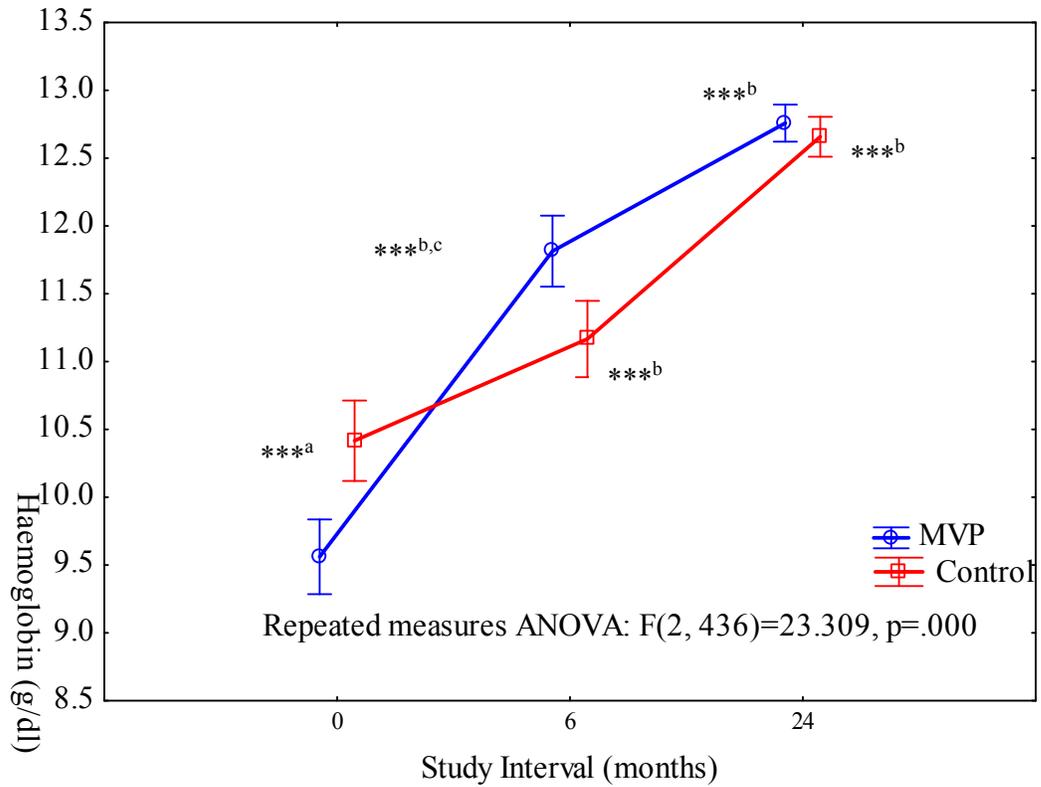
FIGURE 3.7: Trends in the mean haemoglobin concentration by study group and time

Notes: Vertical bars denote 95% CI; *** significantly different at P values < 0.001; [a] mean Hb significantly higher in control than MVP group at baseline; [b] mean Hb significantly higher than the preceding study interval for each study group; [c] Hb significantly higher in MVP group compared to control group at 6 months

TABLE 3.33: Full haemogram and serum ferritin by study group and time

	Baseline	6 months	24 months	Repeated measures ANOVA	Bonferroni test: p[a]	Bonferroni test: p[b]	Bonferroni test: p[c]
	Mean (sd) [CI]	Mean (sd) [CI]	Mean (sd) [CI]	F statistics			
WBC (10^3cells/uL)							
MVP	6.5 (1.7) [6.2, 6.8]	6.2 (1.5) [5.9, 6.5]	6.1 (1.3) [5.9, 6.4]	F(2, 436) = 3.59, p = 0.028*	0.95	0.24	1.0
Control	7.0 (1.9) [6.7, 7.3]	6.2 (1.6) [5.9, 6.5]	6.1 (1.4) [5.8, 6.3]		0.000**	0.000**	1.0
RBC (10^6 cells /uL)							
MVP	4.0 (0.7) [3.9, 4.1]	4.9 (0.5)[d] [4.8, 5]	5.1 (0.4) [5, 5.1]	F(2, 436) = 8.67, p = 0.000**	0.000**	0.000**	1.0
Control	4.2 (0.6) [4.1, 4.3]	4.7 (0.9) [4.5, 4.8]	5 (0.4) [4.9, 5]		0.000**	0.000**	0.000**
Hct (%)							
MVP	33.1 (4.6) [33.4, 44.4]	35.8 (2.7) [35.3, 36.3]	38.7 (1.9) [38.4, 39.1]	F(2, 436) = 1.3783, p = 0.253	1.0	1.0	1.0
Control	33.0 (3.7) [27.5, 38.5]	33.6 (6.5) [32.3, 34.9]	38.4 (1.8) [38.1, 38.8]		1.0	0.46	0.9
MCV (fL)							
MVP	83.9 (10.9) [82.2, 85.4]	75.6 (5.5) [74.6, 76.6]	78.2 (4.5) [77.4, 79]	F(2, 436) = 13.29, p = 0.000**	0.000**	0.000**	0.000**
Control	78.8 (6.5) [77.1,80.4]	75.1 (5.3) [74, 76.1]	78.1 (4.6) [77.2, 79]		0.000**	1.0	0.000**
MCH (pg)							
MVP	23.4 (3.7) [22.7, 24.0]	25.1 (1.9) [24.8, 25.5]	25.9 (1.8) [25.6, 26.2]	F(2, 436) = 7.36, p = 0.000**	0.000**	0.000**	0.09
Control	24.9 (2.6) [22.4, 25.4]	25.6 (3.5) [24.9, 26.3]	25.9 (1.8) [25.6, 26.3]		0.61	0.01*	1.0
MCHC (g/dL)							
MVP	28.3 (3.8) [27.6, 29.5]	33.3 (0.7) [33.2, 33.4]	33 (0.6) [32.9, 33.1]	F(2, 436) = 0.052, p = 0.95	1.0	1.0	1.0
Control	31.5 (1.6) [31.2,31.8]	34.1 (3.8) [33.3, 34.8]	33 (0.8) [32.9, 33.2]		1.0	1.0	1.0

[a] P value compares baseline and 6 months; [b] p value compares baseline and 24 months;
[d] RBC significantly higher among MVP children (p = 0.009)
[c] p value compares 6 months and 24 months
Note: WBC (white blood cells), RBC (red blood cells), HCT (haematocrit), MCV (mean cell volume), Fl (femtolitres), MCH (mean cell haemoglobin), MCHC (mean cell haemoglobin concentration), Plt (platelets), Lymph abs (lymphocytes), ALC (absolute lymphocyte count), * statistically significant at p value < 0.05; **statistically significant at p value < 0.001; Total N = 220; MVP N = 118; control N = 102

TABLE 3.33 Full haemogram and serum ferritin by study group and time (Continued)

	Baseline	6 months	24 months	Repeated measures ANOVA	Bonferroni test: p[a]	Bonferroni test: p[b]	Bonferroni test: p[c]
	Mean (sd) [CI]	Mean (sd) [CI]	Mean (sd) [CI]	F statistics			
Plt (10^3/ cells uL)							
MVP	282.1 (111.2) [264.3, 303.9]	300.3 (62.4) [288.9, 311.6]	257 (61.6) [245.8, 268.3]	F(2, 436) = 1.0, p = 0.37	0.84	0.18	0.000**
Control	299.4 (121.1) [277.2, 321.5]	294.6 (85) [277.9, 311.3]	263.6 (61.9) [251.5, 275.8]		1.0	0.03	0.049*
ALC (%)							
MVP	43.8 (9.9) [43, 46.2]	49.3 (8.5) [47.8, 50.9]	45.3 (6.9) [44.1, 46.6]	F(2, 436) = 1.45, p = 0.24	0.000**	1.0	0.000**
Control	45 (7.6) [43.6, 46.4]	48.7 (9.8) [46.8, 50.6]	47.1 (6.9) [45.7, 48.4]		0.004*	0.51	1.0
Lymph abs (10^3uL)							
MVP	2.9 (0.9) [2.7, 3.0]	3.8 (3.7) [3.2, 4.5]	2.7 (0.6) [2.6, 2.8]	F(2, 436) = 0.29, p = 0.75	0.02*	1.0	0.000**
Control	3.1 (0.9) [3.0, 3.3]	3.7 (3.8) [3, 4.5]	2.8 (0.6) [2.7, 3]		0.69	1.0	0.05
Serum Ferritin (µg/l)							
MVP	35.8 (25.4) [38.1, 55.6]	31.6 (23) [24.5, 38.7]	34.7 (24.4) [27.2, 42.2]	F(2, 164) = 0.97, p = 0.38	1.0	1.0	1.0
Control	46.9 (28.1) [27.9, 43.7]	35.1 (23.6) [27.7, 42.6]	44.3 (28.5) [35.3, 53.3]		0.07	1.0	0.46

[a] P value compares baseline and 6 months; [b] p value compares baseline and 24 months;
[d] RBC significantly higher among MVP children (p = 0.009)
[c] p value compares 6 months and 24 months
Note: WBC (white blood cells), RBC (red blood cells), HCT (haematocrit), MCV (mean cell volume), Fl (femtolitres), MCH (mean cell haemoglobin), MCHC (mean cell haemoglobin concentration), Plt (platelets), Lymph abs (lymphocytes), ALC (absolute lymphocyte count), * statistically significant at p value < 0.05; **statistically significant at p value < 0.001; Total N = 220; MVP N = 118; control N = 102

3.12.7 Trends in serum retinol concentration by study group and time

The mean serum retinol concentration decreased from 0.78 to 0.64μmols/l between the baseline and the 6[th] month study interval in the MVP group, while there was a slight increase in the control group (Figure 3.8). Both groups showed an increase in mean serum retinol concentration between 6 and 24 months. The changes were not statistically significant.
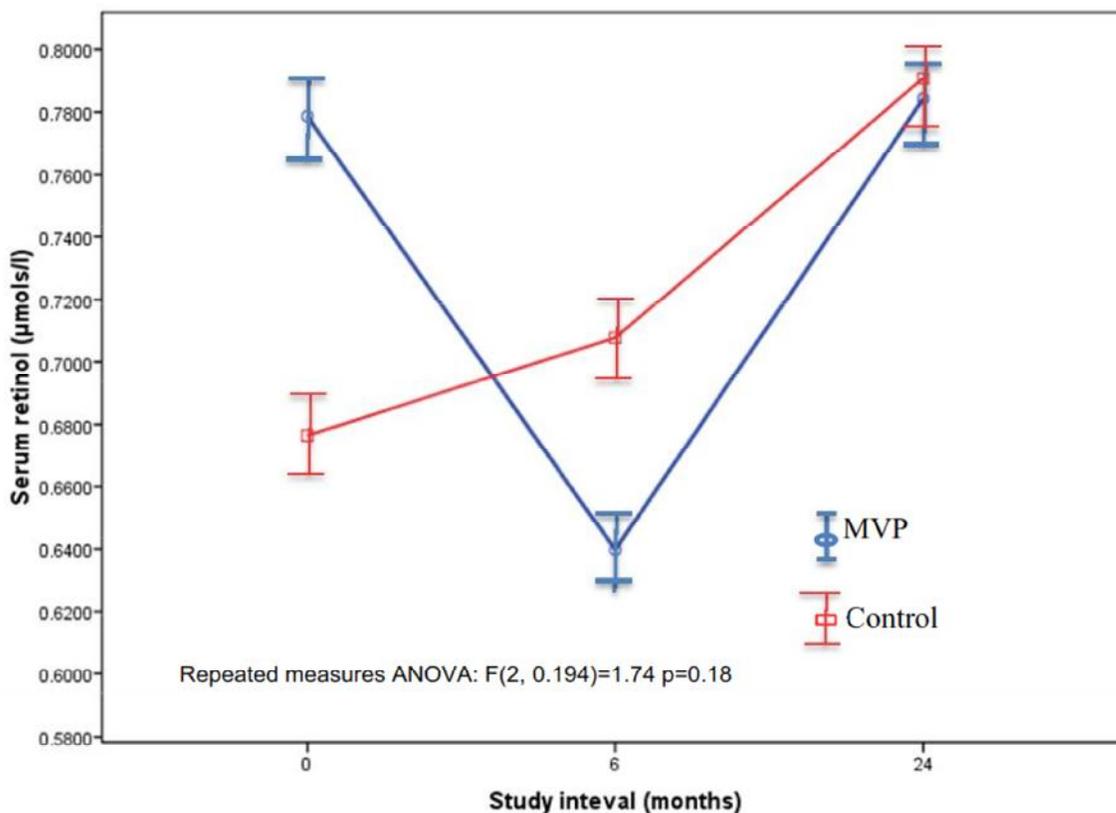
FIGURE 3.8: Trends in the mean serum retinol concentration by study group and time

Note: Vertical bars denote 95% CI

3.13   Part 3: Findings on Impact of the SFP on Nutritional Status, Anaemia, Body Iron Stores and Vitamin A Status

Part 3 contains findings on the impact of the SFP on nutritional status and body composition based on anthropometric measurements, as well as the impact on anaemia, body iron stores and vitamin A status.

3.13.1 Impact of school feeding programmes on nutritional status

The prevalence of inadequate nutritional status based on height-for-age of less than -1 SD was significantly associated with the study group in the 6[th], 12[th], 18[th], 24[th] and 30[th] months of follow-up

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(chi-square test: p values = 0.040, 0.02, 0.007, 0.006 and 0.009; TABLE 3.34). Almost half of the children in the control group had a height-for-age ≤ -1 sd, compared to those from the MVP group (36.4%) in the 6[th] month of the study. This trend was similar throughout the other study intervals (TABLE 3.34). There were no significant differences between the proportion of children with weight-for-age and BMI-for-age ≤ 1.0 sd by study group throughout the study period.

Figure 3.9 presents the prevalence of overweight (BMI-for-age >+1 z-scores) by time and study group. There was an increase in obesity from 5.9% at baseline to 6.4% at 6 months. This was followed by a general decline to 2.3% by the 30[th] month (Figure 3.9). The prevalence of overweight was higher among the control children and increased throughout the study, from 10.3% at baseline to 12.2% by 30 months. The prevalence of overweight was significantly higher in the control (chi-square test: p = 0.019) than in the MVP group by the 24[th] month of the study.
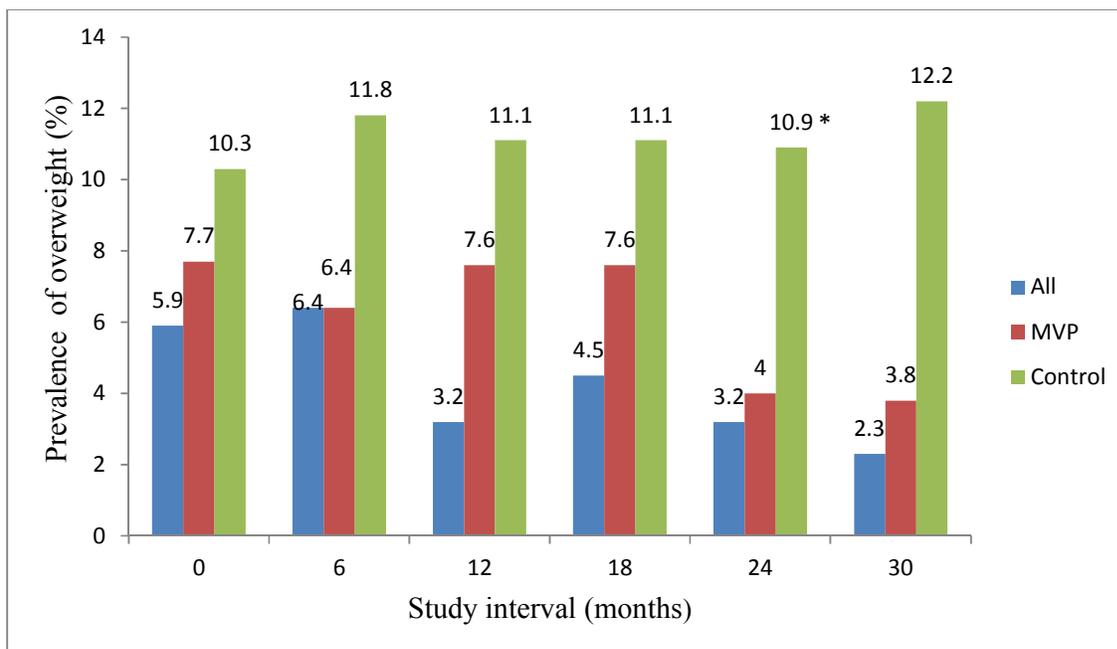
FIGURE 3.9: Trends in overweight by time for combined group

*The prevalence of overweight was significantly higher in the control group (p= 0.019)

TABLE 3.34: Prevalence of inadequate nutritional status based on anthropometric indices by study group and time

Study interval (months)	Group		Height-for-age z-score −1 sd	Height-for-age z-score > -1 sd	Chi-square test: p	Weight-for-age z-score −1 sd	Weight-for-age z-score > -1 sd	Chi-square test: p	BMI-for-age z-score - 1.0 sd	BMI-for-age z-score > -1 sd	Chi-square test: p
					N(%)						
0	MVP	N = 118	44 (37.3)	74 (62.7)	0.07	24 (23.1)	80 (76.9)	0.50	17 (14.4)	101 (84.7)	0.203
	control	N = 102	49 (48.0)	53 (52.0)		21 (24.1)	66 (75.9)		10 (9.8)	92 (90.2)	
6	MVP	N = 118	43 (36.4)	75 (63.6)	0.040*	24 (25.5)	70 (74.5)	0.446	17 (14.4)	101 (85.6)	0.318
	control	N = 102	50 (49.0)	52 (51.0)		21 (27.6)	55 (72.4)		18 (17.6)	84 (82.4)	
12	MVP	N = 118	39 (33.1)	79 (66.9)	0.024*	23 (25.6)	67 (74.4)	0.347	25 (25.2)	93 (78.8)	0.102
	control	N = 102	48 (47.1)	54 (52.9)		21 (29.6)	50 (70.4)		14 (13.7)	88 (86.3)	
18	MVP	N = 118	43 (36.4)	75 (63.6)	0.007*	13 (16.5)	66 (83.5)	0.063	20 (16.9)	98 (83.1)	0.319
	control	N = 102	55 (53.9)	47 (46.1)		18 (28.6)	45 (71.4)		14 (13.7)	88 (86.3)	
24	MVP	N = 118	34 (28.8)	84 (71.2)	0.006*	16 (21.3)	59 (78.7)	0.365	37 (31.4)	81 (68.6)	0.165
	control	N = 102	47 (46.1)	55 (53.9)		14 (25.5)	41 (74.5)		25 (24.5)	77 (75.5)	
30	MVP	N = 118	35 (29.7)	83 (70.3)	0.009*	6 (11.5)	46 (88.5)	0.218	30 (25.4)	88 (74.6)	0.246
	control	N = 102	47 (46.1)	55 (53.9)		8 (19.5)	79 (84.9)		21 (20.6)	81 (79.4)	

*Significant at P < 0.05

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3.13.2 Multiple logistic regression on the impact of school feeding programme on anthropometric indicators

The odds of inadequate nutritional status based on a height-for-age z-score $\leq$ -1 SD was on a general declining trend among the MVP children compared to the control group. The odds of inadequate nutritional status (height-for-age $\leq$ -1 SD) were 0.64 among children in the MVP group at baseline and decreased to 0.49 by the 30[th] month of the study (Figure 3.10). Children in the MVP group were significantly less likely to have inadequate nutritional status based on height-for-age $\leq$ -1 sd in the 12[th], 18[th], 24[th] and 30[th] months compared to those in the control (logistic regression; p values = 0.035, 0.01, 0.009 and 0.013). Odds ratios of inadequate nutritional status based on weight-for-age and BMI-for-age were not significantly different between the two groups throughout the study period (TABLE 3.35).
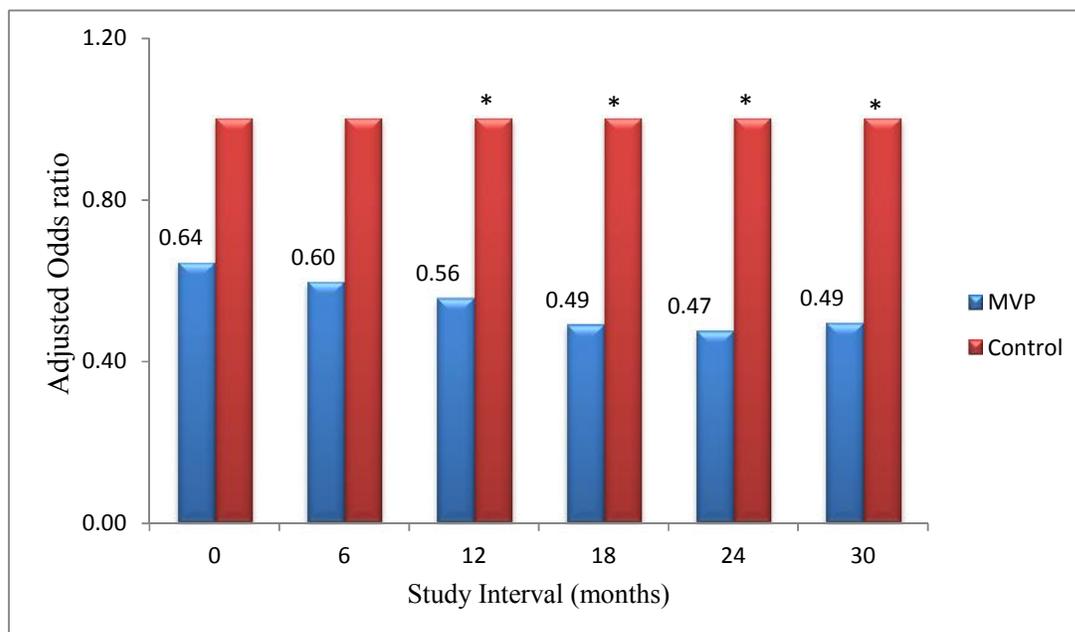
FIGURE 3.10: Trends in odds ratios of undernutrition based on height-for-age z-score by study groups and time

*Logistic regression p values significant at the level of P < 0.05. Model adjusted for background socio-demographic characteristics

TABLE 3.35: Adjusted odds of inadequate nutritional status based on weight-for-age and BMI-for-age by group and time

Study interval (months)		weight-for-age z-score ≤ -1 sd			BMI-for-age z-score ≤ -1 sd		
		OR	CI	Logistic regression P	OR	CI	Logistic regression P
0	MVP	0.943	0.48, 1.843	0.863	1.55	0.68, 3.6	0.302
	control	1.0			1.0		
6	MVP	0.898	0.453, 1.78	0.758	0.79	0.38, 1.62	0.513
	control	1.0			1.0		
12	MVP	0.817	0.41, 1.64	0.57	1.7	0.83, 3.46	0.151
	control	1.0			1.0		
18	MVP	0.49	0.22, 1.1	0.086	1.28	0.61, 2.69	0.51
	control	1.0			1.0		
24	MVP	0.79	0.35, 1.8	0.58	0.261	1.41, 0.78	0.22
	Control	1.0			1.0		
30	MVP	0.538	0.171, 1.7	0.29	1.32	0.7, 2.48	0.39
	Control	1.0			1.0		

Total N = 220; MVP N = 118; Control N = 102

3.13.3 The impact of school feeding programme on the prevalence of anaemia and depleted body iron stores

In general, the prevalence of anaemia decreased in both groups from baseline to 24 months, whereas the prevalence of those with depleted body iron stores increased in both groups from baseline to 6 months, followed by a slight decline from 6 months to the 24[th] month (TABLE 3.36. In the 6[th] month of the study, the prevalence of anaemia had declined significantly (chi-square; p = 0.000) in the MVP group – by 62% compared to the control group. The prevalence of low iron stores was not significantly different between the two study groups at baseline and 6 months. However, by the 24[th] month the prevalence of low iron stores was significantly higher among the MVP children (chi-square; p = 0.024) compared to the control children.

By the 6[th] month interval, the likelihood of children in the MVP group being anaemic was 77% less (RR = 0.23; 95% CI: 0.12–0.42; chi-square; p = 0.000) than in the control group. The likelihood of being anaemic or having depleted body iron stores was not significant between the groups at the 6- and 24-month intervals (TABLE 3.36). The degree of severity of anaemia as a public health problem was compared to the WHO classification.[299] Overall, anaemia was a severe public health problem at baseline and at the 6-month intervals, and a mild public health problem by the 24[th] month of the study (

TABLE 3.37). Comparing between groups, anaemia was a severe public health problem for the MVP and the control group at baseline. However, by the 6[th] month, anaemia was a mild public health problem for the MVP group, while it remained a severe public health problem for the control

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children. Further, anaemia was not a public health problem for the MVP group by the 24[th] month, although it remained of mild significance among the controls (
TABLE 3.37).

TABLE 3.36: Impact of school feeding programme on the prevalence of anaemia and low iron stores by study group and time

	Control	MVP	Chi-square test: p value
	Control group is the reference category; relative risk =1	*RR (95% CI)*	
Baseline			
Anaemia	58.1%	82.2%; RR = 1.45 (95% CI: 1.16–1.83)	0.001**
Depleted body iron stores	11.5%	19.1%; RR = 0.93 (95% CI: 0.77–1.11)	0.306
6 months			
Anaemia	41.2%	9.3%; RR =0.23 (95% CI: 0.12–0.42)	0.000**
Depleted body iron stores	19.5%	27.9%; RR = 1.43 (95% CI: 0.65–3.14)	0.259
24 months			
Anaemia	7.8%	4.2%; RR = 0.54 (95% CI: 0.18–1.6)	0.199
Depleted body iron stores	14.6%	23.3%; RR = 1.29 (95% CI: 0.64–43.98)	0.234

Total number of subjects for anaemia analysis: MVP N = 118; control N = 102; Total number of subjects for depleted body iron stores analysis: MVP N = 42; control N = 42

TABLE 3.37: Degree of public health significance of anaemia comparing WHO cut-offs by group and time

			Degree of public health significance			
			None	Mild	Moderate	Severe
WHO			≤ 4.9	5.0–19.9	20.0–39.9	≥ 40
Study population	Baseline	All				70.2
		MVP				82.2
		Control				58.1
	6 months	All				43.6
		MVP		9.3		
		Control				41.2
	24 months	All			13.2	
		MVP	4.2			
		Control		7.8		

Source: WHO[299]

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3.13.4 The impact of school feeding programme on vitamin A status

During the 6th month of the study, 7.8% of the children in the MVP group and 4.7% in the control group were vitamin A deficient (serum retinol <0.35 µmol/L; TABLE 3.38). This was a deterioration in vitamin A status compared to the baseline, when only one child in the control group had a vitamin A deficiency. Over half of the children in both groups (54% and 60.9%) had inadequate vitamin A status by the 6th month. There was a slight improvement by the 24th month, when the proportion of children with inadequate vitamin A status decreased to 52% in the MVP group and 40.9% in the control group. There was no significant difference in the vitamin A status of the two study groups in the 6th and 24th month study intervals.

TABLE 3.38: Trends in vitamin A status by study group and time

Timeline	MVP N = 50	Control N = 44	Chi-square tests; p
	N (%)		
Baseline			
Vitamin A deficient (<0.35 µmol/L)	0	1 (2.3)	
Inadequate Vitamin A status (0.35 –0.70 µmol/L)	15 (30.0)	22 (50.0)	0.044[a]
Adequate vitamin A status (≥0.70 µmol/L)	35 (70)	21 (47.7)	
6 months			
Vitamin A deficient (<0.35 µmol/L)	4 (7.8)	2 (4.7)	
Inadequate Vitamin A status (0.35 –0.70 µmol/L)	27 (54.0)	26 (60.5)	0.766
Adequate vitamin A status (≥0.70 µmol/L)	19 (37.3)	16 (36.4)	
24 months			
Vitamin A deficient <0.35 µmol/L)	0	0	
Inadequate Vitamin A status (0.35 –0.70 µmol/L)	26 (52.0)	18 (40.9)	0.138
Adequate vitamin A status (≥0.70 µmol/L)	24 (47.1)	26 (60.5)	

[a] P values obtained by Fisher's exact test; *statistically significant at p value < 0.05

The likelihood of having inadequate vitamin A status was significantly higher (chi-square; p = 0.015) among the control children at baseline (Figure 3.11). At 6 months, the relative risk of having inadequate vitamin A status was almost equal in children in the two study groups. Children in the MVP group were 34% more likely to have inadequate vitamin A status by 24 months compared to those in the control, but this was not statistically significant.
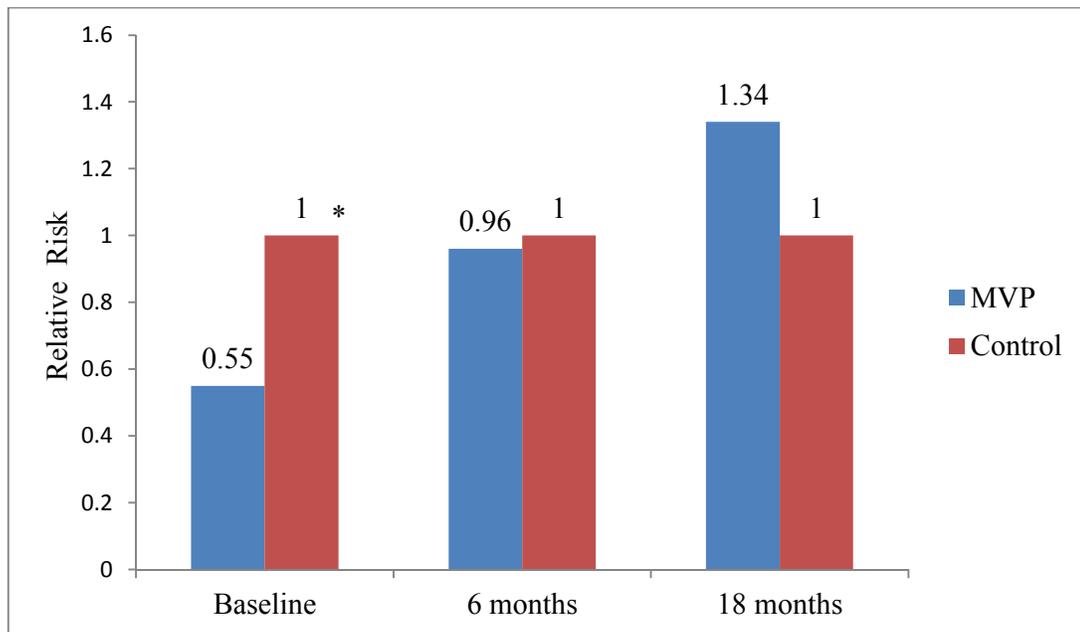
FIGURE 3.11: Relative risk of inadequate vitamin A status by study group and time

*Relative risk for inadequate vitamin A status significantly higher for the control group at baseline; p value = 0.015

3.14   Summary of Results in Section B

Trends in the growth indicators show that children in the MVP group had a bigger height velocity than the controls. Children in the control group gained significant weight and weight velocity by the 12th month after introduction of a SFP, unlike those in the MVP group, whose weight gain by the 12th month was not significant. There were increases in the body composition measurements of fat mass, although no significant differences were observed between the groups over time. The impact of the diversified school feeding programme in the MVP group yielded a higher increase in linear growth, as shown by a significantly greater height velocity by the 18th, 24th and 30th months compared to the controls. In addition, the prevalence of linear growth failure (height-for-age ≤ -1 SD) declined significantly in the MVP group between baseline and 30 months, and the odds of having a low height-for-age declined in the MVP group from baseline to 30 months.

There was an increase in the mean haemoglobin concentration among all the children. Those in the MVP group had a lower mean Hb at baseline compared to the controls, but the value increased significantly by 6 months, to be above that of the control group. The likelihood of being anaemic at the 6-month interval was less in the MVP group compared to the control group. The prevalence of anaemia reduced from 82.2% in the MVP group to 9% by the 6-month interval, and from 58% in the control group to 41%. There was a decrease in the mean serum ferritin concentration from

baseline to the subsequent study intervals. The number of subjects per group for whom serum ferritin concentration was measured was below 50 and the results therefore should be interpreted with caution.

At the 6-month study interval there was a deterioration in vitamin A status, which should also be interpreted with caution because the sample size was less than 50 subjects per group. It is also worth noting that the serum samples were collected within three weeks of school opening after the school holidays, and therefore the serum retinol concentration may not have been reflective of the impact of school feeding, since there was no school feeding programme during the school holidays.

3.15   Section C: The Effect of the Initial Nutritional Status on the Responses to the SFP on Growth, the Prevalence of Anaemia, Body Iron Stores and Vitamin A Status among School Children

This section presents the findings on the effects initial nutritional status on the responses to SFP on growth, the prevalence of anaemia, body iron stores and vitamin A status among school children. The findings for the combined group are presented first, followed by the findings on the effects within each of the study groups separately for each of the initial nutritional status indicators.

3.16   Effects of Initial Nutritional Status, Based on Height-for-Age Z-Score, on the Impact of the SFP on Growth and Body Composition in the Combined Group

The effects of initial nutritional status, based on height-for-age z-scores, on the impact of the school feeding programmes was assessed using repeated measures ANOVA and the Bonferroni post hoc test for the combined group of children first, and then for each of the study groups separately. Repeated measures ANOVA showed a significant effect of initial nutritional status (based on height-for-age z-score) on the mean height [$F_{(5, 1090)} = 5.2653$, $P < 0.0001$], height velocity [$F_{(4, 872)} = 4.3523$, $p = 0.002$] and weight [$F_{(5, 1090)} = 2.4813$, $p = 0.03$] when comparing children with an initial inadequate nutritional status (height-for-age z-scores $\leq$ -1 SD) and those with an initial adequate nutritional status (height-for-age z-scores > -1 SD; TABLE 3.39).

Those with an initial inadequate nutritional status had significantly lower mean height and weight throughout the six study intervals (p values < 0.001). However, the mean height velocity changed

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significantly for all the children (p values < 0.001) from 6 months to the 30[th] month of the study. By the 24[th] month, children with an initial inadequate nutritional status (height-for-age ≤ -1 SD) had a higher height velocity (2.0 cm/6 months) than those with an initial adequate nutritional status based on height-for-age > -1 SD (1.7 cm/6 months; p value = 0.004). The effects of initial nutritional status were not significant for the mean weight velocity, mid-upper arm circumference, triceps and subscapular skinfold measurements, percent body fat, mid-upper arm fat area, mid-upper arm muscle area, fat area and fat-free mass. However, the mean weight velocity increased significantly within each group from initial nutritional status to the 12[th], 18[th], and 30[th] month, with p values < 0.0001 (TABLE 3.39).

At the 18[th] and 30[th] month study intervals, the mean fat mass was significantly higher (P < 0.05) among the children with an initial adequate nutritional status based on height-for-age z-score compared to those with an initial inadequate nutritional status on the basis of low height-for-age. Similarly, fat-free mass was significantly higher (p values < 0.001) among children with an initial adequate height-for-age z-score in all the study intervals compared to those who had initial inadequate nutritional status based on height-for-age z-scores.

TABLE 3.39: Initial nutritional status based on height-for-age by growth, body composition and time in the combined groups

Variable	HAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd)[95%CI]				
Height (cm)	≤ -1 SD	117.2 (10.4) [115.1, 119.4]	120.2 (10) [118.2, 122.3] [a]***	123 (9.6) [121, 125] [a]***	126.2 (8.7) [124.4, 128] [a]***	129.2 (9) [127.4, 131.1] [a]***	132.8 (9.2) [130.9, 134.7] [a]***	$F_{(5, 1090)} = 5.2653$, $p = 0.00009$***
	> -1 SD	126.2 (9.9)[b]*** [124.4, 127.9]	128.8 (9.7) [b]*** [127.1, 130.5] [a]***	131.2 (9.3) [b]*** [129.5, 132.8] [a]***	134 (8.7) [b]*** [132.4, 135.5] [a]***	136.3 (8.9)[b]*** [134.8, 137.9] [a]***	140.5 (9.3)[b]*** [138.9, 142.2] [a]***	
HV (cm/6 months)	≤ -1 SD		0.5 (0.4) [0.4, 0.6]	1 (0.6) [0.8, 1.1] [a]***	1.5 (0.7) [1.4, 1.6] [a]***	2 (0.7)[b]* [1.8, 2.1] [a]***	2.6 (0.7) [2.4, 2.7] [a]***	$F_{(4, 872)} = 4.3523$, $p = 0.00172$*
	> -1 SD		0.4 (0.2) [0.4, 0.5]	0.8 (0.4) [0.8, 0.9] [a]***	1.3 (0.5) [1.2, 1.4] [a]***	1.7 (0.6) [1.6, 1.8] [a]***	2.4 (0.6) [2.3, 2.5] [a]***	
Weight (kg)	≤ -1 SD	22 (4.5) [21.1, 22.9]	23.2 (4.6) [22.2, 24.1][a]***	24 (4.4)*** [23.1, 24.9] [a]***	25.9 (4.6)*** [25, 26.9]	26.1 (4.6)[a]*** [25.2, 27.1]	28.2 (4.8)[a]*** [27.2, 29.2]	$F_{(5, 1090)} = 2.4813$, $p = 0.03026$*
	> -1 SD	25 (4.9) [b]* [24.2, 25.9]	26.5 (5) [b]*** [25.6, 27.3] [a]***	27.2 (5.1) [b]** [26.3, 28.1] [a]*	29.4 (5.5) [b]*** [28.4, 30.3]	29.8 (5.7) [b]*** [28.8, 30.8]	32.1 (6.1) [b]*** [31, 33.1] [a]***	
WV (kg/6 months)	≤ -1 SD		0.2 (0.3) [0.1, 0.3]	0.3 (0.3) [0.3, 0.4] [a]*	0.7 (0.3) [0.6, 0.7] [a]***	0.7 (0.3) [0.6, 0.8]	1 (0.4) [0.9, 1.1] [a]***	$F_{(4, 872)} = 1.8273$, $p = 0.12147$
	> -1 SD		0.2 (0.2) [0.2, 0.3]	0.4 (0.3) [0.3, 0.4] [a]*	0.7 (0.3) [0.7, 0.8] [a]**	0.8 (0.4) [0.7, 0.8]	1.2 (0.5) [1.1, 1.3] [a]***	
MUAC (cm)	≤ -1 SD	17.1 (1.4) [16.8, 17.4]	17.3 (1.3) [17, 17.5]	17.2 (1.4) [16.9, 17.5]	17.8 (1.2) [17.6, 18.1] [a]***	18 (1.2) [17.8, 18.3]	18.6 (1.5) [18.3, 19] [a]***	$F_{(5, 1070)} = 1.0086$, $p = 0.41126$
	> -1 SD	17.7 (1.5) [17.4, 18]	17.9 (1.6) [17.6, 18.2]	17.9 (1.6) [17.6, 18.1]	18.4 (1.7) [18.2, 18.7] [a]***	18.5 (1.5) [18.3, 18.8]	19 (1.6) [18.8, 19.3] [a]***	
Triceps (mm)	≤ -1 SD	4.7 (1.5) [4.4, 5]	5.2 (1.3) [4.9, 5.4]	5.8 (1.3) [5.5, 6.1] [a]**	5.8 (1.3) [5.6, 6.1]	6.3 (1.2) [6, 6.5]	6.2 (1.4) [5.9, 6.5]	$F_{(5, 1080)} = 0.78823$, $p = 0.55817$
	> -1 SD	4.9 (1.3) [4.6, 5.1]	5.3 (1.7) [5, 5.6]	6 (1.9) [5.7, 6.3] [a]***	6.3 (1.9) [6.0, 6.6]	6.5 (1.7) [6.2, 6.8]	6.4 (1.7) [6.1, 6.7]	
Sub-scapular (mm)	≤ -1 SD	4.2 (1) [4, 4.4]	5.1 (0.9) [4.9, 5.3]	4.5 (1) [4.2, 4.7] [a]***	4.8 (0.7) [4.6, 4.9]	4.7 (0.8) [4.6, 4.9]	5 (1) [4.8, 5.2]	$F_{(5, 1085)} = 0.49195$, $p = 0.78246$
	> -1 SD	4.4 (1.3) [4.2, 4.7]	5.2 (1.1) [5, 5.4]	4.5 (1.4) [4.3, 4.8] [a]***	5 (1.1) [4.8, 5.2]	5 (1.1) [4.8, 5.1]	5.2 (1.1) [5, 5.4]	

HAZ: height-for-age z-score ≤ -1 SD (N = 93); height-for-age > -2 SD (N = 127); HV: height velocity; WV: weight velocity; MUAC: mid-upper arm circumference; * $P < 0.05$, ** $P < 0.001$, ***$P < 0.0001$; [a]Data point is compared with the previous data point in the same study group. [b]Data point is compared with the same data point across the two categories of initial nutritional status.

TABLE 3.39: Initial nutritional status based on height-for-age by growth, body composition and time in the combined groups (Continued)

Variable	HAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
		Mean (sd)[95%CI]						
Body fat (%)	≤ -1 SD	7.3 (2.5) [6.8, 7.8]	8.1 (2.6) [7.6, 8.6]	9.2 (2.4) [8.7, 9.7]	9.7 (2.3) [9.2, 10.1]	9.7 (2.4) [9.2, 10.2]	9.9 (2.7) [9.3, 10.4]	F(5, 1075) = 0.42842, p = 0.82903
	> -1 SD	7.8 (2.6) [7.3, 8.3]	8.4 (3.1) [7.9, 9]	9.7 (3.2) [9.1, 10.2]	10.3 (2.8) [9.8, 10.8]	10.4 (2.9) [9.9, 10.9]	10.4 (3) [9.9, 10.9]	
Fat mass (kg)	≤ -1 SD	1.6 (0.6) [1.5, 1.7]	1.9 (0.6) [1.7, 2]	2.2 (0.7) [2.1, 2.4][a]**	2.3 (0.7) [2.2, 2.5]	2.5 (0.8) [2.3, 2.7]	2.9 (1.2) [2.6, 3.1][a]**	F(5, 1070) = 1.1573, p = 0.32830
	> -1 SD	2 (0.9)	2.3 (1.2)	2.7 (1.3) [2.4, 2.9][a]**	2.9 (1.3)[b]**	3 (1.2)	3.4 (1.4)[b]** [3.2, 3.7][a]***	
MUAFA (mm)^2	≤ -1 SD	381.8 (122.6) [356.4, 407.2]	424.3 (114.2) [400.6, 447.9]	474.4 (118.1) [450, 498.9][a]*	494.4 (113.2) [471, 517.9]	535 (119.2) [510.3, 559.7]	550.1 (130.5) [523.1, 577.2]	F(5, 1070) = 0.71775, p = 0.61015
	> -1 SD	415.7 (138) [391.2, 440.3]	455.9 (183.4) [423.3, 488.5]	512.3 (199.9) [476.7, 547.8][a]**	554.7 (208.7) [517.6, 591.8][a]*	576.1 (179.1) [544.2, 607.9]	581.3 (183.1) [548.7, 613.8]	
MUAMA (mm)^2	≤ -1 SD	1 968.1 (370.9) [1 891.3, 2 044.9]	1 976.7 (329.8) [1 908.3, 2 045]	1 941.2 (304.7) [1 878.1, 2 004.3]	2 063.2 (299.4) [2 001.2, 2 125.2][a]**	2 082 (285.1) [2 022.9, 2 141]	2 251.8 (425.7) [2 163.7, 2340][a]***	F(5, 1090) = 0.94019, p = 0.45393
	> -1 SD	2 109.4 (354.9) [2046.3, 2172.4]	2 139.4 (362.4) [2 075, 2 203.8]	2 098.6 (346.4) [2 037, 2160.2]	2192.7 (380.7) [2125, 2260.4][a]**	2190.4 (338.3) [2130.3, 2250.6]	2 343.3 (385.5) [2 274.7, 2411.8][a]***	
		[1.8, 2.1]	[2, 2.5][a]*		[2.7, 3.1]	[2.8, 3.2]		
Fat-free mass (kg)	≤ -1 SD	20.4 (4.2) [19.5, 21.3]	21.3 (4.3)[b]*** [20.4, 22.2][a]*	21.7 (4)[b]*** [20.9, 22.6]	23.1 (3.8) [22.3, 23.8][a]***	23.3 (3.9) [22.5, 24.1]	25.9 (4.1) [25.1, 26.7][a]***	F(5, 1090) = 0.59137, p = 0.70663
	> -1 SD	23.1 (4.4)[b]** [22.3, 23.8]	24.2 (4.3)[b]*** [23.5, 25][a]***	24.5 (4.3) [23.8, 25.3]	25.7 (4.1)[b]** [25, 26.4][a]***	25.7 (4.1)[b]** [25, 26.4]	28.7 (5)[b]** [25.1, 26.7][a]***	

HAZ: height-for-age z-score ≤ -1 SD (N = 93); Height-for-age > -2 SD (N = 127); MUAFA: mid-upper arm fat area; MUAMA:-mid-upper arm muscle area; * P < 0.05, **P < 0.001, ***P < 0.0001; [a]Data point is compared with the previous data point in the same study group. [b]Data point is compared with the same data point across the two categories of initial nutritional status.

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3.16.1 Effect of initial nutritional status, based on height-for-age z-scores, on the impact of the school feeding programme on growth and body composition among the MVP group

The effect of initial nutritional status based on height-for-age z-score was also considered in the intervention and control groups (TABLE 3.40). In the MVP group, initial nutritional status had an effect on the changes in mean height [$F(5, 580) = 5.1251$, $p = 0.00013$], height velocity [$F(4, 464) = 3.0933$, $p = 0.01567$], weight [$F(5, 580) = 3.0842$, $p = 0.00931$] and weight velocity [$F(4, 464) = 3.4332$, $p = 0.00883$]. Children with initial adequate nutritional status based on the three indicators (height-for-age, weight-for-age and BMI-for-age) were significantly taller from the baseline throughout the study intervals compared to those with initial inadequate nutritional status (p values < 0.0001). At the 24th month study interval, those with initial inadequate nutritional status had a higher height velocity (2.3 cm/6 months) than those who had initial adequate nutritional status (P < 0.01) in the MVP group. The mean weight was highest in the 24th and 30th months among children who had initial adequate nutritional status in the MVP group. The 24th month coincided with a mean age of 10 years. The mean weight velocity was significantly higher in the 18th month compared to the 12th month (P < 0.0001) and in the 30th month compared to the 24th month (P < 0.0001) among all the children in the MVP group. No significant effects of initial nutritional status were observed for changes in mid-upper arm circumference, triceps and subscapular skinfold measurements in the MVP group.

3.16.2 Effect of initial nutritional status, based on height-for-age z-scores, on the impact of the school feeding programme on growth and body composition in the control group

In the control group, initial nutritional status, on the basis of height-for-age z-score, had significant effects on the mean height [$F(5, 500) = 2.3792$, $p = 0.03775$] and height velocity [$F(4, 400) = 2.52$, $p = 0.04037$] (Table 3.30). Similar to the case with the MVP children, the mean height in the control group was significantly higher (P < 0.05) among those with initial adequate nutritional status (height-for-age > -1 SD) compared to those with initial inadequate nutritional status throughout the study. Height velocity increased significantly (p values < 0.001) for all the children throughout the study, but did not differ significantly between those with initial adequate and those with initial inadequate nutritional status on the basis of height-for-age z-scores in the control group. This is unlike the situation in the MVP group, where there were differences between groups in the 24th month of study. In the control group, the effect of initial nutritional status was not significant for the mean weight, weight velocity, MUAC, triceps and subscapular skinfold measurements, as well as for fat mass and fat-free mass.

TABLE 3.40: Initial nutritional status based on height-for-age by growth, body composition time and study group

Variable	HAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
	MVP							
Height (cm)	< -1 SD	116 (11.2) [112.6, 119.4]	119.7 (10.9) [116.3, 123] [a]***	122.9 (10.3) [119.7, 126] [a]***	126.3 (9.3) [123.5, 129.2] [a]***	129.5 (9.1) [126.8, 132.3] [a]***	132.8 (9.5) [129.9, 135.7] [a]***	$F_{(5, 580)} = 5.1251$, p = 0.00013**
	> -1 SD	125.7 (9.5) [b]*** [123.5, 127.9]	128.7 (9.6) [b]** [126.5, 130.9] [a]***	131.1 (9.3) [b]** [129, 133.3] [a]***	134.1 (8.8) [b]** [132, 136.1] [a]***	136.7 (8.6) [b]** [134.8, 138.7] [a]***	140.6 (9) [b]** [138.5, 142.6] [a]***	
	Control							
	< -1 SD	118.4 (9.6) [115.6, 121.1]	120.7 (9.3) [118.1, 123.4] [a]***	123.1 (9) [120.5, 125.7] [a]***	126.2 (8.3) [123.8, 128.5] [a]***	128.9 (9) [126.3, 131.5] [a]***	132.7 (9.1) [130.1, 135.3] [a]***	$F_{(5, 500)} = 2.3792$, p = 0.03775*
	> -1 SD	126.8 (10.4) [b]** [123.9, 129.6]	129 (10) [b]** [126.2, 131.8] [a]***	131.3 (9.5) [b]** [128.6, 133.9] [a]***	133.9 (8.7) [b]** [131.5, 136.3] [a]***	135.8 (9.4) [b]* [133.2, 138.3] [a]***	140.4 (10) [b]** [137.7, 143.2] [a]***	
	MVP							
HV (cm/6months)	< -1 SD		0.6 (0.5) [0.5, 0.8]	1.1 (0.7) [0.9, 1.4] [a]***	1.7 (0.8) [1.5, 2] [a]***	2.3 (0.8) [b]* [2, 2.5] [a]***	2.8 (0.9) [2.5, 3.1] [a]***	$F_{(4, 464)} = 3.0933$, p = 0.01567*
	> -1 SD		0.5 (0.2) [0.4, 0.6]	0.9 (0.4) [0.8, 1] [a]***	1.4 (0.5) [1.3, 1.5] [a]***	1.8 (0.6) [1.7, 2] [a]***	2.5 (0.6) [2.3, 2.6] [a]***	
	Control							
	< -1 SD		0.4 (0.3) [0.3, 0.5]	0.8 (0.5) [0.6, 0.9] [a]***	1.3 (0.5) [1.1, 1.5] [a]***	1.8 (0.5) [1.6, 1.9] [a]***	2.4 (0.5) [2.3, 2.5] [a]***	$F_{(4, 400)} = 2.52$, p = 0.04037*
	> -1 SD		0.4 (0.2) [0.3, 0.4]	0.7 (0.4) [0.6, 0.8] [a]***	1.2 (0.6) [1, 1.4] [a]***	1.5 (0.6) [1.3, 1.7] [a]***	2.3 (0.6) [2.1, 2.4] [a]***	
	MVP							
Weight (kg)	< -1 SD	21.7 (4.8) [20.2, 23.1]	23.3 (5.4) [21.7, 24.9] [a]***	23.9 (4.8) [22.4, 25.4]	26 (4.9) [24.5, 27.5] [a]***	25.6 (4.4) [24.3, 27]	27.9 (4.7) [26.4, 29.3] [a]***	$F_{(5, 580)} = 3.0842$, p = 0.00931*
	> -1 SD	24.6 (4.6) [23.6, 25.7]	26.3 (4.9) [25.1, 27.4] [a]***	26.8 (5.1) [25.7, 28]	29.3 (5.4) [28.1, 30.6] [a]***	29.6 (5.6) [b]* [28.3, 30.9]	31.9 (6) [b]* [30.5, 33.3]	
	Control							
	< -1 SD	22.3 (4.2) [21.1, 23.5]	23.1 (3.8) [22, 24.1] [a]***	24 (4) [22.9, 25.1] [a]***	25.9 (4.3) [24.6, 27.1] [a]***	26.6 (4.8) [25.2, 27.9] [a]***	28.5 (4.9) [27, 29.9] [a]***	$F_{(5, 500)} = 0.57984$, p = 0.71548
	> -1 SD	25.6 (5.2) [24.2, 27]	26.7 (5.3) [25.3, 28.2] [a]***	27.7 (5.2) [26.3, 29.1] [a]***	29.5 (5.6) [27.9, 31] [a]***	30 (5.9) [28.4, 31.7] [a]***	32.3 (6.3) [30.5, 34] [a]***	

HAZ: Height-for-age z-score; MVP group [N = 44 for children with < -1 SD and N = 74 for children with HAZ ≥ -1 SD]; Control group [N = 49 for children with < -1 SD and N = 53 for children with HAZ ≥ -1 SD]; HV: height velocity; * P < 0.05, ** P < 0.001, ***P < 0.000. [a]Data point is compared with the previous data point in the same study group. [b]Data point is compared with the same data point across the two categories of initial nutritional status.

TABLE 3.40: Initial nutritional status based on height-for-age by growth, body composition time and study group (Continued)

Variable	HAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
	MVP			*Mean (sd) [95% CI]*					
WV (kg/6 months)	< -1 SD		0.3 (0.4) [0.2, 0.4]	0.4 (0.3) [0.3, 0.5]	0.7 (0.3) [0.6, 0.8] [a]***	0.7 (0.4) [0.6, 0.8]	1 (0.5) [0.9, 1.2] [a]***	$F_{(4, 464)} =$ 3.4332,
	> -1 SD		0.3 (0.2) [0.2, 0.3]	0.4 (0.3) [0.3, 0.4]	0.8 (0.3) [0.7, 0.9] [a]***	0.8 (0.4) [0.7, 0.9]	1.2 (0.5) [1.1, 1.3] [a]***	$p = 0.00883*$
	Control							
	< -1 SD		0.1 (0.2) [0.1, 0.2]	0.3 (0.2) [a]* [0.2, 0.3]	0.6 (0.3) [a]*** [0.5, 0.7]	0.7 (0.3) [0.6, 0.8]	1 (0.3) [a]*** [0.9, 1.1]	$F_{(4, 400)} =$ 0.23669,
	> -1 SD		0.2 (0.2) [0.1, 0.3]	0.3 (0.2) [0.3, 0.4] [a]*	0.6 (0.3) [0.6, 0.7] [a]***	0.7 (0.3) [0.6, 0.8]	1.1 (0.5) [1, 1.2] [a]***	$p = 0.91757$
	MVP							
MUAC (cm)	< -1 SD	17 (1.4) [16.6, 17.4]	17.3 (1.4) [16.9, 17.7]	17.1 (1.5) [16.7, 17.6]	17.9 (1.3) [17.5, 18.3][a]**	18.2 (1.1) [17.8, 18.5]	18.5 (1.2) [18.1, 18.9]	$F_{(5, 575)} =$ 0.94884,
	> -1 SD	17.7 (1.6) [17.3, 18.1]	17.9 (1.8) [17.5, 18.3]	17.8 (1.7) [17.4, 18.2]	18.5 (1.8) [18.1, 18.9][a]***	18.5 (1.5) [18.1, 18.8]	19 (1.6) [18.6, 19.4] [a]*	$p = 0.44881$
	Control							
	< -1 SD	17.1 (1.5) [16.7, 17.5]	17.2 (1.2) [16.8, 17.6]	17.3 (1.3) [16.9, 17.6]	17.8 (1.2) [17.4, 18.1]	17.9 (1.3) [17.5, 18.3]	18.8 (1.7) [18.3, 19.2] [a]***	$F_{(5, 485)} =$ 0.53809,
	> -1 SD	17.7 (1.3) [17.3, 18.1]	17.9 (1.4) [17.5, 18.3]	17.9 (1.4) [17.5, 18.3]	18.4 (1.5) [18, 18.8][a]*	18.6 (1.4) [18.2, 19]	19.1 (1.5) [18.7, 19.6][a]*	$p = 0.74745$
	MVP							
Triceps (mm)	< -1 SD	4.8 (1.5) [4.3, 5.2]	5.2 (1.1) [4.9, 5.6]	5.8 (1.2) [5.4, 6.1]	5.7 (1.1) [5.3, 6]	6.2 (1.2) [5.8, 6.6]	6.1 (1.3) [5.7, 6.5]	$F_{(5, 575)} =$ 0.97559,
	> -1 SD	4.8 (1.6) [4.5, 5.2]	5.4 (1.9) [5, 5.9][a]*	5.9 (1.9) [5.5, 6.4]	6.2 (1.9) [5.8, 6.7]	6.6 (1.7) [6.2, 7]	6.3 (1.6) [6, 6.7]	$p = 0.43195$
	Control							
	< -1 SD	4.6 (1.5) [4.2, 5]	5.1 (1.4) [4.7, 5.5]	5.9 (1.4) [5.4, 6.3][a]*	6 (1.3) [5.6, 6.4]	6.3 (1.3) [5.9, 6.7]	6.4 (1.4) [5.9, 6.8]	$F_{(5, 495)} =$ 0.47954,
	> -1 SD	4.9 (0.9) [4.7, 5.2]	5.1 (1.3) [4.7, 5.4]	6.1 (2) [5.5, 6.6][a]***	6.4 (1.9) [5.8, 6.9]	6.5 (1.7) [6, 7]	6.5 (1.9) [6, 7.1]	$p = 0.79160$

HAZ: height-for-age z-score; MVP group [N = 44 for children with < -1 SD and N = 74 for children with HAZ ≥ -1 SD]; Control group [N = 49 for children with < -1 SD and N = 53 for children with HAZ ≥ -1 SD]; HV: height velocity; * $P < 0.05$, ** $P < 0.001$, *** $P < 0.000$. [a]Data point is compared with the previous data point in the same study group.

TABLE 3.40: Initial nutritional status based on height-for-age by growth, body composition time and study group (Continued)

Variable	HAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
	MVP							
Sub-scapular (mm)	< -1 SD	4.3 (1) [4, 4.6]	4.6 (1.1) [4.2, 4.9]	5.2 (0.9) [4.9, 5.5]ᵃ*	4.7 (0.6) [4.5, 4.9]	4.7 (0.7) [4.5, 4.9]	5 (1) [4.7, 5.3]	F(5, 575) = 0.80912, p = 0.54339
	> -1 SD	4.5 (1.4) [4.2, 4.8]	4.5 (1.6) [4.2, 4.9]	5.1 (1.2) [4.8, 5.4]ᵃ*	5 (1.1) [4.7, 5.2]	4.9 (1.1) [4.6, 5.1]	5.1 (1.2) [4.9, 5.4]	
	Control							
	< -1 SD	4.1 (1) [3.8, 4.4]	4.3 (1) [4.1, 4.6]	5 (1) [4.7, 5.2]ᵃ**	4.8 (0.7) [4.6, 5]	4.8 (0.9) [4.5, 5]	5 (1) [4.7, 5.3]	F(5, 500) = 0.31619,
	> -1 SD	4.3 (1.1) [4, 4.7]	4.5 (1.1) [4.2, 4.8]	5.3 (1) [5, 5.6]ᵃ***	5 (1) [4.7, 5.3]	5.1 (1.1) [4.8, 5.4]	5.3 (1.1) [5, 5.6]	p = 0.90327
	MVP							
Body fat (%)	< -1 SD	7.4 (2.5) [6.6, 8.1]	8.3 (2.5) [7.5, 9]	9.1 (2.2) [8.4, 9.8]	9.4 (2.3) [8.7, 10.1]	9.6 (2.3) [8.9, 10.3]	9.5 (2.6) [8.7, 10.3]	F(5, 570) = 0.62146,
	> -1 SD	7.9 (2.9) [7.2, 8.6]	8.7 (3.4) [7.9, 9.5]	9.5 (3.4) [8.7, 10.3]	10.4 (3) [9.7, 11.1]	10.4 (2.9) [9.7, 11.1]	10.2 (2.8) [9.6, 10.9]	p = 0.68350
	Control							
	< -1 SD	7.2 (2.5) [6.5, 7.9]	8 (2.8) [7.2, 8.8]	9.3 (2.6) [8.6, 10.1]ᵃ***	9.9 (2.3) [9.2, 10.5]	9.9 (2.5) [9.2, 10.6]	10.2 (2.7) [9.4, 11]	F(5, 495) = 0.33500,
	> -1 SD	7.7 (2.2) [7.1, 8.3]	8.1 (2.7) [7.3, 8.8]	9.9 (3) [9.1, 10.7]ᵃ***	10.3 (2.7) [9.6, 11]	10.4 (3) [9.5, 11.2]	10.6 (3.2) [9.7, 11.5]	p = 0.89175
	MVP							
Fat mass (kg)	< -1 SD	1.6 (0.7) [1.4, 1.8]	1.9 (0.6) [1.7, 2.1]	2.2 (0.7) [2, 2.4]	2.3 (0.7) [2.1, 2.5]	2.5 (0.7) [2.2, 2.7]	2.8 (1.2) [2.5, 3.2]	F(5, 580) = 1.0884,
	> -1 SD	2 (0.9) [1.7, 2.2]	2.3 (1.3) [2, 2.6]ᵃ*	2.6 (1.3) [2.3, 2.9]	2.9 (1.4) [2.6, 3.2]	3 (1.2) [2.7, 3.2]	3.4 (1.4) [3.1, 3.8]ᵃ***	p = 0.36561
	Control							
	< -1 SD	1.6 (0.5) [1.4, 1.7]	1.8 (0.6) [1.6, 2]	2.2 (0.7) [2, 2.4]ᵃ**	2.4 (0.8) [2.2, 2.6]	2.6 (0.9) [2.3, 2.8]	2.9 (1.2) [2.6, 3.2]	F(5, 500) = 0.38130,
	> -1 SD	2 (0.8) [1.8, 2.2]	2.2 (1.1) [1.9, 2.5]	2.7 (1.3) [2.4, 3.1]ᵃ***	2.8 (1.2) [2.5, 3.2]	3 (1.3) [2.6, 3.3]	3.4 (1.5) [3, 3.8]ᵃ*	p = 0.86165

HAZ: height-for-age z-score; * P < 0.05, ** P < 0.001, *** P < 0.000. ᵃData point is compared with the previous data point in the same study group.

TABLE 3.40: Initial nutritional status based on height-for-age by growth, body composition time and study group (Continued)

Variable	HAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
	MVP			*Mean (sd) [95% CI]*				
MUFA (mm)^2	< -1 SD	390.7 (134.1) [349.9, 431.5]	430.9 (100.3) [400.4, 461.3]	468.4 (113.6) [433.9, 503]	482.2 (106.9) [449.7, 514.7]	533.8 (112.7) [499.6, 568.1]	534.7 (122.3) [497.5, 571.9]	F(5, 575) = 1.3412, p = 0.24519
	> -1 SD	413.7 (162.6) [375.8, 451.7]	470.1 (208.2) [421.5, 518.7] ^{a*}	507.9 (204.9) [460.1, 555.7]	552.3 (212) [502.8, 601.7]	579.7 (176.6) [538.5, 620.9]	570.6 (161.9) [532.8, 608.4]	
	Control							
	< -1 SD	373.6 (111.9) [341.1, 406.1]	418.2 (126.4) [381.5, 454.9]	479.9 (123) [444.2, 515.7]	505.7 (118.6) [471.3, 540.1]	536.1 (126) [499.5, 572.6]	564.3 (137.4) [524.4, 604.1]	F(5, 485) = 0.87729, p = 0.49607
	> -1 SD	418.6 (93.6) [392.3, 444.9]	435.6 (140.3) [396.2, 475.1]	518.6 (194.4) [463.9, 573.3] ^{a**}	558.2 (206) [500.3, 616.2]	571 (184.3) [519.1, 622.8]	596.6 (210.5) [537.4, 655.8]	
	MVP							
MUAMA (mm)^2	< -1 SD	1 941.3 (351.1) [1 834.5, 2 048]	1987.8 (367.6) [1 876, 2 099.6]	1 940.5 (336.3) [1 838.3, 2 042.8]	2 094 (320.4) [1 9 96.6, 2 191.4] ^{a*}	2 117.7 (278.7) [2 032.9, 2 202.4]	2 226.1 (311.3) [2 131.4, 2320.7]	F(5, 575) = 1.3412, p = 0.24519
	> -1 SD	2 110.6 (372.7) [2 023.7, 2 197.6]	2 130 (393.4) [2 038.2, 2 221.8]	2 096.8 (375.8) [2 009.2, 2 184.5]	2 208.8 (407.6) [2 1 13.7, 2 303.9]	2 178.6 (346.8) [2 097.7, 2 259.5]	2 334.4 (417.9) [2 236.9, 2 431.9]	
	Control							
	< -1 SD	1 992.8 (390.3) [1879.4, 2 106.1]	1 966.5 (294.6) [1 880.9, 2052]	1 941.8 (276.2) [1 861.6, 2022.1]	2 035 (279.2) [1 954, 2116.1]	2 049.2 (289.9) [1 965, 2 133.4]	2 275.4 (510.9) [2 127.1, 2 423.8] ^{a**}	F(5, 485) = 0.87729, p = 0.49607
	> -1 SD	2 107.5 (331.5) [2 014.3, 2 200.7]	2 152.8 (316) [2 063.9, 2 241.7]	2 101.1 (303) [2 015.9, 2186.4]	2 169.7 (341.2) [2 073.7, 2 265.6]	2 207.4 (328.4) [2 115, 2 299.8]	2 356 (337.3) [2 261.1, 2450.9] ^{a**}	
	MVP							
Fat-free mass (kg)	< -1 SD	20.1 (4.5) [18.7, 21.4]	21.4 (5) [19.9, 22.9] ^{a*}	21.7 (4.4) [20.3, 23]	23 (4.2) [21.7, 24.3] ^{a*}	23.1 (3.9) [21.9, 24.3]	26 (4.1) [24.7, 27.2] ^{a***}	F(5, 580) = 0.23400, p = 0.94755
	> -1 SD	22.7 (4.1) [21.7, 23.6]	24 (4.2) [23, 25] ^{a***}	24.2 (4.2) [23.2, 25.2]	25.7 (4.1) [24.7, 26.6] ^{a***}	25.4 (3.8) [24.5, 26.2]	28.5 (4.8) [27.4, 29.6] ^{a***}	
	Control							
	< -1 SD	20.7 (4) [19.6, 21.9]	21.2 (3.6) [20.2, 22.3]	21.8 (3.6) [20.7, 22.8]	23.1 (3.4) [22.1, 24.1] ^{a***}	23.4 (3.9) [22.3, 24.5]	25.8 (4.1) [24.7, 27] ^{a***}	F(5, 500) = 0.80081, p = 0.54941
	> -1 SD	23.6 (4.7) [22.3, 24.9]	24.6 (4.5) [23.3, 25.8]	25 (4.4) [23.8, 26.2]	25.7 (4.1) [24.6, 26.8]	26.2 (4.5) [24.9, 27.4]	28.9 (5.4) [27.4, 30.4] ^{a***}	

HAZ: Height-for-age z-score; MVP group [N = 44 for children with < -1 SD and N = 74 for children with HAZ ≥ -1 SD]; Control group [N = 49 for children with < -1 SD and N = 53 for children with HAZ ≥ -1 SD]: HV: height velocity; * P < 0.05, ** P < 0.001, *** P < 0.000. ^{a}Data point is compared with the previous data point in the same study group.

3.17   Effects of Initial Nutritional Status, Based on Weight-for-age Z-score, on the Impact of the School Feeding Programmes on Growth and Body Composition in the Combined Group

Repeated measures ANOVA showed a significant effect of initial nutritional status (based on weight-for-age z-score) on the mean height [$F(5, 945) = 8.1297$, $p = 0.000001$], height velocity [$F(4, 756) = 3.8988$, $p = 0.00384$], mid-upper arm circumference [$F(5, 925) = 4.7302$, $p = 0.00028$] and mid-upper arm muscle area [$F(5, 925) = 5.2312$, $p = 0.0001$] when comparing children with an initial inadequate nutritional status (weight-for-age z-scores ≤ 1 sd) and those with an initial adequate nutritional status (height-for-age z-scores > 1 sd; TABLE 3.41) overall.

The mean height, mid-upper arm circumference and mid-upper arm muscle area were significantly lower (p values < 0.05) throughout the study among children with initial inadequate nutritional status (weight-for-age ≤ -1 SD) compared to their counterparts with weight-for-age > -1 SD (TABLE 3.41). Children with an initial weight-for-age ≤ -1 SD had a significantly higher mean height velocity in the 18[th], 24[th] and 30[th] months of the study (p values < 0.05). In addition, although the interaction between initial nutritional status and other anthropometric measurements was not significant, the mean weight, fat mass and fat-free mass were significantly lower among the children with a weight-for-age ≤ -1 SD throughout the study (P values < 0.05; TABLE 3.41).

TABLE 3.41: Initial nutritional status based on weight-for-age by growth, body composition and time in the combined groups

Variable	WAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
Height (cm)	≤ -1 SD	113.8 (8.7) [111.2, 116.5]	117.5 (8.3) [115, 120][a]***	120.7 (8.3) [118.2, 123.2][a]***	124.4 (7.4) [122.2, 126.7] [a]***	126.9 (7.3) [124.7, 129.1] [a]***	130.4 (7.1) [128.2, 132.5] [a]***	$F(5, 945) = 8.1297$, $p = 0.000001$***
	> -1 SD	122.7 (9.8)[b]*** [121.1, 124.3]	125.3 (9.7)[b]** [123.7, 126.9][a]***	127.8 (9.3)[b]* [126.3, 129.3][a]***	130.8 (8.7)[b] [129.4, 132.3][a]***	133.3 (8.6)[b] [131.9, 134.7][a]***	137.3 (9.3)[b]** [135.8, 138.9] [a]***	
HV (cm/6 months)	≤ -1 SD		0.6 (0.4) [0.4, 0.7]	1.1 (0.6) [0.9, 1.3][a]***	1.7 (0.6)[b]** [1.5, 1.9][a]***	2.1 (0.7)[b]*** [1.9, 2.4] [a]***	2.7 (0.7)[b]* [2.5, 2.9][a]***	$F(4, 756) = 3.8988$, $p = 0.00384$*
	> -1 SD		0.4 (0.2) [0.3, 0.4]	0.8 (0.3) [0.7, 0.9][a]***	1.3 (0.5) [1.2, 1.4][a]***	1.7 (0.5) [1.6, 1.8] [a]***	2.4 (0.5) [2.3, 2.5][a]***	
Weight (kg)	≤ -1 SD	19.3 (2.9) [18.4, 20.2]	20.8 (3.1) [19.9, 21.8][a]***	21.7 (3.3) [20.7, 22.7]	23.7 (3.5) [22.6, 24.8][a]***	23.8 (3.3) [22.8, 24.8]	25.8 (3.6) [24.7, 26.9][a]***	$F(5, 945) = 0.56780$, $p = 0.72475$
	> -1 SD	24 (4.2)[b]*** [23.3, 24.7]	25.3 (4.4)[b]*** [24.6, 26][a]***	26.1 (4.5)[b]*** [25.4, 26.8][a]***	28.1 (4.6)[b]*** [27.3, 28.8][a]***	28.3 (4.7)[b]*** [27.6, 29.1]	30.6 (5.3)[b]*** [29.8, 31.5][a]***	
WV (kg/6 months)	≤ -1 SD		0.2 (0.2) [0.1, 0.3]	0.4 (0.2) [0.3, 0.4]	0.7 (0.3) [0.6, 0.8][a]***	0.7 (0.3) [0.6, 0.8]	1 (0.4) [0.9, 1.2][a]***	$F(4, 756) = 0.58585$, $p = 0.67298$
	> -1 SD		0.2 (0.1) [0.1, 0.2]	0.3 (0.2) [0.3, 0.3][a]***	0.6 (0.2) [0.6, 0.7][a]***	0.7 (0.2) [0.6, 0.7]	1.1 (0.4) [1, 1.1][a]***	
MUAC (cm)	≤ -1 SD	16 (1) [15.7, 16.3]	16.3 (1) [16, 16.7]	16.2 (1.1) [15.9, 16.6]	17.1 (1) [16.8, 17.4][a]***	17.4 (1.1) [17, 17.7]	18 (1.7) [17.5, 18.5][a]*	$F(5, 925) = 4.7302$, $p = 0.00028$**
	> -1 SD	17.5 (1.3)[b]*** [17.3, 17.8]	17.8 (1.4)[b]*** [17.5 ,18]	17.7 (1.4)[b]*** [17.5, 18]	18.2 (1.3)[b]** [18, 18.5][a]***	18.3 (1.3)[b]** [18.1, 18.6]	18.9 (1.3)[b]** [18.7, 19.1][a]***	
Triceps (mm)	≤ -1 SD	4.4 (1.2) [4, 4.8]	4.8 (1.4) [4.4, 5.3]	5.4 (1.3) [5, 5.8]	5.6 (1.2) [5.2, 6]	5.8 (1.2) [5.4, 6.2]	5.7 (1.3) [5.3, 6.2]	$F(5, 935) = 0.26463$, $p = 0.93241$
	> -1 SD	4.8 (1.4) [4.6, 5.1]	5.3 (1.5) [5, 5.5][a]***	5.9 (1.6) [5.7, 6.2][a]***	6.1 (1.6) [5.8, 6.4]	6.4 (1.5) [6.2, 6.7]	6.3 (1.5) [6.1, 6.6]	

WAZ: weight-for-age z-score ≤ -1 SD (N = 45); Weight-for-age > -1 SD (N = 196); HV: height velocity; WV: weight velocity; MUAC: mid-upper arm circumference; *P=< 0.05,** P < 0.001, *** P < 0.0001; [a]Data point is compared with the previous data point in the same study group. [b]Data point is compared with the same data point across the two categories of initial nutritional status

TABLE 3.41: Initial nutritional status based on weight-for-age by growth, body composition and time in the combined groups (Continued)

Variable	WAZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
Sub-scapular (mm)	≤ -1 SD	3.8 (0.8) [3.5, 4.1]	4.9 (0.9) [4.6, 5.2]^{a}*	4.3 (1.1) [3.9, 4.6]	4.6 (0.6) [4.4, 4.8]	4.5 (0.7) [4.3, 4.7]	4.6 (0.8) [4.4, 4.9]	$F_{(5,\,940)}$ = 1.5321, p = 0.17713
	> -1 SD	4.4 (1.2) [4.2, 4.6]	5.1 (1) [4.9, 5.3]^{a}***	4.5 (1.2) [4.2, 4.7]	4.8 (0.8) [4.7, 5]	4.8 (0.9) [4.7, 5]	5.1 (1) [4.9, 5.2]	
Body fat (%)	≤ -1 SD	6.6 (2) [6, 7.2]	7.7 (2.8) [6.8, 8.5]	8.6 (2.5) [7.8, 9.3]	9.2 (2.3) [8.4, 9.9]	8.9 (2.5) [8.2, 9.7]	9 (2.7) [8.2, 9.8]	$F_{(5,\,930)}$ = 0.69282, p = 0.62896
	> -1 SD	7.8 (2.7) [7.4, 8.3]	8.5 (2.9) [8, 8.9]^{a}*	9.6 (2.9) [9.2, 10.1]^{a}***	10.1 (2.6) [9.7, 10.6]	10.2(2.7) [9.8, 10.7]	10.3 (2.8) [9.9, 10.8]	
Fat mass (kg)	≤ -1 SD	1.2 (0.4) [1.1, 1.4]	1.5 (0.5) [1.4, 1.7]	1.9 (0.6) [1.7, 2]	2 (0.6) [1.8, 2.2]	2.1 (0.7) [1.9, 2.3]	2.3 (1) [2, 2.6]	$F_{(5,\,945)}$ = 1.8996, p = 0.09187
	> -1 SD	1.8 (0.7)^{b}* [1.7, 2]	2.1 (0.9)^{b}* [1.9, 2.3]^{a}**	2.5 (1)^{b}* [2.3, 2.6]^{a}**	2.6 (0.9)^{b}* [2.5, 2.8]	2.8 (0.9)^{b}* [2.6, 2.9]	3.2 (1.1)^{b}*** [3, 3.4]^{a}***	
MUAFA (mm)^2	≤ -1 SD	339 (93.8) [310.4, 367.5]	382.8 (124.4) [345, 420.7]	420 (118.7)^{b}* [383.9, 456.1]	458.3 (113.9) [423.6, 492.9]	483.3 (121.9) [446.3, 520.4]	497.5 (128.3) [458.5, 536.5]	$F_{(5,\,925)}$ = 0.13605, p = 0.98399
	> -1 SD	413 (140.8) [389.7, 436.3]	455.2 (162.8) [428.3, 482.2]^{a}*	508.2 (172.3) [479.7, 536.7]^{a}***	535.8 (166.9) [508.2, 563.4]	565.2 (154.7) [539.6, 590.7]	573.5 (152.6) [548.2, 598.7]	
MUAMA (mm)^2	≤ -1 SD	1 738.9 (278.6) [1 654.2, 1 823.6]	1 777 (231.2) [1 706.6, 1847.3]	1 741 (219) [1 674.4, 1 807.7]	1 909.8 (247.9) [1 834.4, 1985.2]	1 961.6 (246.6) [1 886.6, 2 036.6]	2 136.2 (525.1) [1 976.6, 2 295.9]^{a}**	$F_{(5,\,925)}$ = 5.2312, p = 0.0001**
	> -1 SD	2 078.9 (329.4)^{b}*** [2 024.4, 2 133.3]	2 110.7 (337.1)^{b}*** [2 055, 2 166.4]	2 072.6 (318.9)^{b}*** [2 019.9, 2 125.3]	2 160.8 (336.9)^{b}*** [2 105.1, 2 216.5]^{a}*	2 158.3 (308.6)^{b}*** [2 107.2, 2 209.3]	2 318.9 (349.4)^{b}** [2 261.1, 2 376.7]^{a}***	
Fat-free mass (kg)	≤ -1 SD	18 (2.7)^{b}*** [17.2, 18.8]	19.2 (3)^{b}*** [18.3, 20.1]^{a}*	19.8 (3)^{b}*** [18.9, 20.7]	21.2 (2.8)^{b}*** [20.3, 22]^{a}**	21.5 (2.6)^{b}*** [20.7, 22.3]	24 (3.1)^{b}*** [23.1, 25]^{a}**	$F_{(5,\,945)}$ = 1.0970, p = 0.36039
	> -1 SD	22.1 (3.9) [21.4, 22.7]	23.2 (4) [22.5, 23.8]^{a}***	23.6 (3.9) [22.9, 24.2]	24.8 (3.6) [24.2, 25.4]^{a}***	24.8 (3.6) [24.2, 25.4]	27.7 (4.4) [26.9, 28.4]^{a}***	

WAZ: weight-for-age z-score ≤ -1 SD (N = 45); Weight-for-age > -1 SD (N = 196); MUAFA: mid-upper arm fat area; MUAMA: mid-upper arm muscle area; WAZ: weight-for-age z-score ≤ -1 SD (N = 45); Weight-for-age > -2 SD (N = 196); *P=< 0.05, ** P < 0.001, ***P < 0.0001; ^{a}Data point is compared with the previous data point in the same study group.
^{b}Data point is compared with the same data point across the two categories of initial nutritional status

3.17.1 Effect of initial nutritional status, based on weight-for-age z-scores, on the impact of the school feeding programme on growth and body composition in the MVP group

The effect of initial nutritional status, based on weight-for-age z-score, was also considered separately in the intervention and the control groups (TABLE 3.42). Initial nutritional status based on weight-for-age z-scores had an effect on the mean height $[F(5, 510) = 9.3708, P < 0.000001]$, height velocity $[F(4, 408) = 4.5624, p = 0.00129]$, mid-upper arm circumference $[F(5, 505) = 4.4335, p = 0.00058]$ and mid-upper arm muscle circumference $[F(5, 505) = 4.4723, p = 0.00054]$. Children with initial adequate nutritional status based on weight-for-age had a significantly higher mean height at all the study intervals compared to those with initial inadequate nutritional status (p values < 0.0001). Those with initial inadequate nutritional status (weight-for-age $\leq$ -1 SD) had a significantly higher mean height velocity compared to those who had initial adequate nutritional status ($P < 0.01$) in the MVP group.

The within-group changes in mean height and height velocity from one study interval to the next were significant for all the children (p values < 0.0001). Although the interaction between initial weight-for-age z-score and weight was not significant, $[F(5, 510) = 0.79482, p = 0.55370]$, the children in the MVP group with an initial weight-for-age $> $ -1 SD had a significantly higher mean weight at all the study intervals (P values < 0.01).

The mean mid-upper arm circumference measurements were significantly higher in the children in the MVP group with initial adequate nutritional status (weight-for-age $> $ -1 SD) at baseline and at the 6[th] and 12[th] month study intervals (p values < 05) compared to those in the same group with an initial weight-for-age z-score $< $ -1 SD. Mid-upper arm muscle circumference was significantly higher among the MVP children with initial adequate nutrition (weight-for-age z-scores $> $ -1 SD) at the 6[th] and 18[th] month study intervals compared to those who had inadequate initial nutritional status (weight-for-age $< $ -1 SD; P value < 0.05). Mid-upper arm muscle area was significantly higher in the 18[th] month compared to the 12[th] month among children in the MVP group with initial weight-for-age $< $ -1 SD ($P < 0.0001$).

3.17.2 Effect of initial nutritional status, based on weight-for-age z-scores, on the impact of the SFP on growth and body composition in the control group

In the control group, initial nutritional status on the basis of weight-for-age z-score had significant effects on the changes in mean subscapular skinfold measurements [$F(5, 425) = 2.3635$, $p = 0.03918$] and fat mass [$F(5, 425) = 2.6027$, $p = 0.02469$; TABLE 3.42].

In the 12[th] month study interval, the mean subscapular measurements were significantly higher than in the 6[th] month study interval for both the children with adequate and those with inadequate nutritional status ($P < 0.001$) in the control group. In the 12[th] month study intervals, the mean subscapular skinfold measurements were significantly higher (p values < 0.05) for the control children with initial adequate nutritional status (weight-for-age > -1 SD) compared to those with initial inadequate nutritional status (weight-for-age ≤ -1 SD). Unlike in the MVP group, the effects of initial nutritional status on height, height velocity, mid-upper arm circumference and mid-upper arm muscle area were not significant in the control group (TABLE 3.42).

TABLE 3.42: Initial nutritional status based on weight-for-age by growth, body composition, group and time

Variable	WAZ by study group	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
Height (cm)	MVP < -1 SD	112.5 (9.7) [108.4, 116.5]	116.8 (8.9) [113, 120.5][a]***	120.6 (9.5) [116.6, 124.6][a]***	124.7 (8.3) [121.2, 128.2][a]***	127.6 (7.7) [124.3, 130.8][a]***	130.7 (7.8) [127.4, 134][a]***	F(5, 510) = 9.3708, P < 0.000001***
	> -1 SD	122.7 (9.3)[b]** [120.6, 124.8]	125.6 (9.5)[b]** [123.5, 127.7][a]***	128.2 (8.9)[b]* [126.2, 130.2][a]***	131.3 (8.5) [129.4, 133.2][a]***	134.2 (8.3) [132.3, 136][a]***	138 (8.8)[b] [136, 139.9][a]***	
	Control < -1 SD	115.5 (7.6) [112, 118.9]	118.4 (7.7) [114.9, 121.9][a]***	120.8 (7.1) [117.6, 124.1][a]***	124.2 (6.7) [121.1, 127.2][a]***	126.2 (7) [123, 129.4][a]***	130.1 (6.5) [127.2, 133.1][a]***	F(5, 425) = 0.98263, p = 0.42794
	> -1 SD	122.8 (10.6) [120.2, 125.4]	125 (10.3) [122.5, 127.5][a]***	127.5 (9.9) [125, 129.9][a]***	130.4 (9.2) [128.1, 132.6][a]***	132.3 (9) [130.1, 134.5][a]***	136.7 (10.1) [134.2, 139.1][a]***	
HV(cm/ 6 months)	MVP < -1 SD		0.7 (0.5) [0.5, 0.9]	1.4 (0.7)[b]* [1.1, 1.6][a]***	2 (0.7)[b]** [1.8, 2.3][a]***	2.5 (0.8)[b]** [2.2, 2.9][a]***	3 (0.8)[b]* [2.7, 3.4][a]***	F(4, 408) = 4.5624, p = 0.00129*
	> -1 SD		0.5 (0.2) [0.4, 0.5]	0.9 (0.4) [0.8, 1][a]***	1.4 (0.5) [1.3, 1.5][a]***	1.9 (0.6) [1.8, 2][a]***	2.5 (0.5) [2.4, 2.7][a]***	
	Control < -1 SD		0.5 (0.4) [0.3, 0.6][a]***	0.9 (0.5) [0.7, 1.1][a]***	1.5 (0.5) [1.2, 1.7][a]***	1.8 (0.5) [1.6, 2][a]***	2.4 (0.5) [2.2, 2.7][a]***	F(4, 340) = 0.42050, p = 0.79384
	> -1 SD		0.4 (0.2) [0.3, 0.4][a]***	0.8 (0.3) [0.7, 0.9][a]***	1.3 (0.5) [1.1, 1.4][a]***	1.6 (0.5) [1.4, 1.7][a]***	2.3 (0.5) [2.2, 2.4][a]***	
Weight (kg)	MVP < -1 SD	18.7 (2.9) [17.5, 20]	20.7 (3.4) [19.3, 22.1][a]**	21.6 (3.7) [20, 23.1]	24 (4) [22.4, 25.7][a]***	23.9 (3.6) [22.4, 25.4]	26.1 (4) [24.4, 27.7]	F(5, 510) = 0.79482, p = 0.55370
	> -1 SD	23.9 (4.1)[b]** [22.9, 24.8]	25.4 (4.5)[b]** [24.4, 26.4][a]***	26.1 (4.6)[b]** [25.1, 27.1]	28.2 (4.7)[b]** [27.2, 29.3][a]***	28.4 (4.9)[b]** [27.3, 29.5]	30.7 (5.2)[b]** [29.6, 31.9][a]***	
		23.2 (4.5) [22.2, 24.1]	24.3 (4.5) [23.3, 25.3]	25.2 (4.6) [24.2, 26.2]	26.8 (4.8) [25.8, 27.9]	27.3 (4.8) [26.3, 28.3]	29.4 (5.4) [28.3, 30.6]	
	Control < -1 SD	20 (2.9) [18.6, 21.3]	21.1 (2.9) [19.8, 22.4]	21.9 (2.9) [20.6, 23.3]	23.4 (3.2) [22, 24.9][a]*	23.9 (3.2) [22.5, 25.3]	25.7 (3.3) [24.2, 27.2][a]*	F(5, 425) = 0.87478, p = 0.49789
	> -1 SD	24.2 (4.5)[b]* [23.1, 25.3]	25.3 (4.5)[b]* [24.2, 26.4][a]**	26.2 (4.5)[b]* [25.1, 27.4][a]***	27.9 (4.7)[b]* [26.8, 29.1][a]***	28.4 (4.7)[b]* [27.2, 29.5]	30.6 (5.5)[b]* [29.3, 32][a]***	

WAZ: weight-for-age z-score; MVP group [N = 24 for children with < -1 SD and N = 79 for children with WAZ ≥ -1 sd]; Control group [N = 20 for children with < -1 SD and N = 64 for children with WAZ ≥ -1 SD]); *P=< 0.05,** P < 0.001, *** P < 0.0001; HV: height velocity; [a] Data point is compared with the previous data point in the same study group. [b] Data point is compared with the same data point across the two categories of initial nutritional status

TABLE 3.42: Initial nutritional status based on weight-for-age by growth, body composition, group and time (Continued)

Variable	WAZ by study group	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
WV (kg/ 6 months)	MVP < -1 SD		0.3 (0.2) [0.2, 0.4]	0.5 (0.2) [0.4, 0.6]	0.9 (0.4) [0.7, 1]^{a***}	0.9 (0.4) [0.7, 1]	1.2 (0.5) [1, 1.4]^{a***}	$F_{(4, 408)}$ = 0.40838, p = 0.80263
	> -1 SD		0.3 (0.2) [0.2, 0.3]	0.4 (0.3) [0.3, 0.4]	0.7 (0.3) [0.7, 0.8]^{a***}	0.8 (0.3) [0.7, 0.8]	1.1 (0.5) [1, 1.3]^{a***}	
	Control < -1 SD		0.2 (0.2) [0.1, 0.3]	0.3 (0.3) [0.2, 0.4]	0.6 (0.3) [0.4, 0.7]^{a*}	0.7 (0.3) [0.5, 0.8]	1 (0.3) [0.8, 1.1]^{a**}	$F_{(4, 340)}$ = 0.71237, p = 0.58393
	> -1 SD		0.2 (0.2) [0.1, 0.2]	0.3 (0.2) [0.3, 0.4]^{a**}	0.6 (0.2) [0.6, 0.7]^{a***}	0.7 (0.3) [0.6, 0.8]	1.1 (0.4) [1, 1.2]^{a***}	
MUAC (cm)	MVP < -1 SD	16 (1) [15.6, 16.4]	16.4 (1) [16, 16.8]	16.2 (1.2) [15.7, 16.7]	17.3 (1.1) [16.8, 17.7]^{a***}	17.6 (1.1) [17.1, 18.1]	18 (1.3) [17.4, 18.5]	$F_{(5, 505)}$ = 4.4335, p = 0.00058**
	> -1 SD	17.6 (1.5)^{b**} [17.3, 18]	17.9 (1.6)^{b*} [17.5, 18.3]	17.8 (1.6)^{b} [17.5, 18.2]	18.4 (1.6) [18, 18.7]^{a**}	18.5 (1.4) [18.2, 18.8]	18.9 (1.5) [18.6, 19.3]^{a*}	
	Control < -1 SD	16.2 (1.2) [15.6, 16.8]	16.4 (1.1) [15.8, 16.9]	16.4 (1.2) [15.8, 17]	17 (1.1) [16.5, 17.5]	17.2 (1.2) [16.7, 17.7]	18.1 (2.3) [17.1, 19.2]^{a*}	$F_{(5, 410)}$ = 1.2160, p = 0.30066
	> -1 SD	17.5 (1.2)^{b*} [17.2, 17.8]	17.7 (1.2)^{b*} [17.5, 18]	17.7 (1.2)^{b*} [17.4, 18]	18.2 (1.1)^{b} [17.9, 18.5]^{a**}	18.3 (1.1) [18, 18.6]	19 (1.3) [18.7, 19.3]^{a**}	
Triceps (mm)	MVP < -1 SD	4.5 (1.1) [4, 4.9]	5 (1.1) [4.6, 5.5]	5.5 (1) [5.1, 5.9]	5.5 (0.8) [5.1, 5.8]	6 (1.3) [5.5, 6.5]	5.9 (1.3) [5.4, 6.5]	$F_{(5, 505)}$ = 0.19104, p = 0.96595
	> -1 SD	4.9 (1.7) [4.5, 5.3]	5.4 (1.8) [5, 5.8]^{a*}	5.9 (1.8) [5.5, 6.3]	6.1 (1.8) [5.7, 6.5]	6.5 (1.6) [6.2, 6.9]	6.2 (1.4) [5.9, 6.6]	
	Control < -1 SD	4.4 (1.5) [3.7, 5.1]	4.7 (1.8) [3.9, 5.5]	5.4 (1.8) [4.5, 6.2]	5.8 (1.7) [5, 6.6]	5.6 (1.4) [5, 6.2]	5.6 (1.5) [5, 6.3]	$F_{(5, 420)}$ = 0.63294, p = 0.67470
	> -1 SD	4.9 (1.2) [4.6, 5.2]	5.2 (1.2) [4.9, 5.5]	6.1 (1.6) [5.7, 6.5]^{a***}	6.3 (1.6) [5.9, 6.7]	6.5 (1.5) [6.1, 6.8]	6.5 (1.6) [6.1, 6.9]	

WAZ: weight-for-age z-score; MVP group [N = 24 for children with < -1 SD and N = 79 for children with WAZ ≥ -1 sd]; Control group [N = 20 for children with < -1 SD and N = 64 for children with WAZ ≥ -1 SD]);*P < 0.05, ** P < 0.001, *** P < 0.0001; WV: weight velocity; MUAC: mid-upper arm circumference; [a] Data point is compared with the previous data point in the same study group. [b] Data point is compared with the same data point across the two categories of initial nutritional status

TABLE 3.42: Initial nutritional status based on weight-for-age by growth, body composition, group and time (Continued)

Variable	WAZ by study group	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
Sub-scapular	MVP	3.7 (0.7)	4.2 (1)	5 (1.1)	4.7 (0.7)	4.5 (0.8)	4.6 (0.8)	$F_{(5, 505)} = 0.46874$, $p = 0.79961$
	< -1 SD	[3.4, 4]	[3.7, 4.6]	[4.5, 5.6]	[4.3, 5]	[4.1, 4.8]	[4.3, 5]	
	> -1 SD	4.4 (1.1)	4.5 (0.9)	5.1 (0.9)	4.9 (0.8)	4.9 (0.9)	5.2 (1)	
		[4.1, 4.7]	[4.3, 4.7]	[4.9, 5.4]	[4.7, 5.1]	[4.7, 5.1]	[4.9, 5.4]	
	Control	4 (1)	4.4 (1.3)	4.8 (0.8)	4.5 (0.7)	4.6 (0.8)	4.8 (0.9)	$F_{(5, 425)} = 2.3635$, $p = 0.03918$*
	< -1 SD	[3.6, 4.4]	[3.8, 5]	[4.4, 5.1][a]**	[4.3, 4.8]	[4.3, 5]	[4.4, 5.1]	
	> -1 SD	4.5 (1.3)	4.5 (1.5)	5.1 (1.2)	4.9 (0.9)	4.8 (1.1)	5.1 (1.1)	
		[4.2, 4.8]	[4.2, 4.9]	[4.9, 5.4][a]***	[4.7, 5.1]	[4.6, 5.1]	[4.8, 5.3]	
Body fat (%)	MVP	6.8 (1.7)	8 (2.5)	8.5 (1.9)	9.3 (2)	9.3 (2.4)	10.3 (9.2)	$F_{(5, 500)} = 0.18488$, $p = 0.96829$
	< -1 SD	[6.1, 7.5]	[7, 9.1]	[7.7, 9.4]	[8.5, 10.1]	[0.5, 8.2]	[0.5, 8]	
	> -1 SD	7.8 (3)	8.6 (3.3)	9.4 (3.3)	10.1 (2.8)	10.2 (2.7)	10.8 (10)	
		[7.2, 8.5]	[7.9, 9.4]	[8.7, 10.2]	[9.4, 10.7]	[0.3, 9.6]	[0.3, 9.4]	
	Control	6.4 (2.3)	7.2 (3.1)	8.6 (3)	9 (2.8)	8.5 (2.5)	9.7 (8.8)	$F_{(5, 420)} = 0.86894$, $p = 0.50188$
	< -1 SD	[5.3, 7.4]	[5.7, 8.7]	[7.2, 10]	[7.7, 10.3]	[0.6, 7.4]	[0.6, 7.5]	
	> -1 SD	7.8 (2.3)	8.3 (2.4)	9.9 (2.5)	10.2 (2.3)	10.3 (2.6)	10.9 (10.7)	
		[7.3, 8.4]	[7.7, 8.9]	[9.3, 10.5][a]***	[9.6, 10.8]	[0.3, 9.7]	[0.4, 10]	
Fat mass (kg)	MVP	1.3 (0.5)	1.6 (0.5)	1.9 (0.6)	2.1 (0.6)	2.2 (0.8)	2.5 (1.2)	$F_{(5, 510)} = 0.35369$, $p = 0.87989$
	< -1 SD	[1.1, 1.5]	[1.4, 1.9]	[1.7, 2.2]	[1.8, 2.3]	[1.9, 2.5]	[2, 3]	
	> -1 SD	1.9 (0.9)	2.2 (1.1)	2.5 (1.2)	2.7 (1.1)	2.8 (1.1)	3.3 (1.2)	
		[1.7, 2.1]	[1.9, 2.4][a]*	[2.2, 2.7]	[2.5, 3]	[2.6, 3.1]	[3, 3.5][a]**	
	Control	1.3 (0.4)	1.5 (0.6)	1.9 (0.7)	2.1 (0.7)	2 (0.7)	2.2 (0.8)	$F_{(5, 425)} = 2.6027$, $p = 0.02469$*
	< -1 SD	[1.1, 1.5]	[1.2, 1.8]	[1.6, 2.2]	[1.7, 2.4][a]	[1.7, 2.4]	[1.8, 2.6]	
	> -1 SD	1.9 (0.6)	2.1 (0.8)	2.6 (0.8)	2.7 (0.8)	2.8 (0.9)[b]*	3.2 (1.1)[b]**	
		[1.7, 2]	[1.9, 2.3]	[2.3, 2.8][a]***	[2.5, 2.8]	[2.6, 3]	[2.9, 3.5][a]***	

WAZ: Weight-for-age z-score; MVP group [N = 24 for children with < -1 SD and N = 79 for children with WAZ ≥ -1 sd]; Control group [N = 20 for children with < -1 SD and N = 64 for children with WAZ ≥ -1 SD]);*P=< 0.05, **P < 0.001, ***P < 0.0001; [a] Data point is compared with the previous data point in the same study group. [b] Data point is compared with the same data point across the two categories of initial nutritional status

TABLE 3.42: Initial nutritional status based on weight-for-age by growth, body composition, group and time (Continued)

Variable	WAZ by study group	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
		Mean (sd) [95% CI]						
MUAFA (mm)^2	MVP < -1 SD	339.6 (83.2) [304.5, 374.7]	393.5 (88.1) [356.3, 430.8]	422.2 (90.8) [383.9, 460.5]	450.1 (82.7) [415.1, 485]	502.6 (116.1) [453.6, 551.7]	508 (128.5) [453.7, 562.2]	F(5, 505) = 0.23532, p = 0.9469
	> -1 SD	414.5 (165) [377.6, 451.5]	468.2 (192.3) [425.2, 511.3]^{a*}	504.3 (192.6) [461.1, 547.4]	531.9 (181.4) [491.3, 572.5]	571.1 (163.6) [534.5, 607.7]	561.6 (140.3) [530.2, 593]	
	Control < -1 SD	338.3 (107.5) [288, 388.6]	370 (159.2) [295.5, 444.6]	417.5 (148) [348.2, 486.8]	468.3 (144.6) [400.6, 535.9]	460.3 (127.8) [400.5, 520.1]	485.1 (130.3) [424.1, 546.1]	F(5, 410) = 0.45102, p = 0.81254
	> -1 SD	411.3 (104.9) [385.1, 437.5]	439.3 (116.2) [410.3, 468.3]	513.1 (144.9) [476.9, 549.3]^{a**}	540.8 (148.4) [503.7, 577.9]	557.9 (144.1) [521.9, 593.9]	588.3 (166.7) [546.6, 629.9]	
MUAMA (mm)^2	MVP < -1 SD	1707.9 (230.5) [1 610.6, 1 805.2]	1 773.1 (241.5) [1 671.2, 1 875.1]	1 723.4 (215.8) [1 632.3, 1 814.6]	1 954.6 (254.6) [1 847.2, 2062.1]^{a***}	1 996.2 (259) [1 886.8, 2 105.5]	2 088.4 (262.4) [1 977.5, 2 199.2]	F(5, 505) = 4.4723, p = 0.00054 **
	> -1 SD	2 095.7 (352.2)^{b**} [2 016.8, 2 174.6]	2 122.8 (374.8)^{b*} [2 038.8, 2 206.7]	2 091.2 (355.6)^{b***} [2 011.5, 2 170.8]	2 190.9 (378.2) [2 106.1, 2275.6]^{a*}	2 181.4 (336.2) [2 106.1, 2 256.7]	2 328.5 (390.3) [2 241.1, 2 415.9]^{a***}	
	Control < -1 SD	1 776.2 (329.8) [1 621.8, 1 930.5]	1 781.7 (224.6) [1676.6, 1886.8]	1 762.3 (226.7) [1 656.2, 1868.4]	1 856.1 (234.7) [1 746.3, 1965.9]	1 920.2 (230.6) [1812.3, 2 028.1]	2 193.8 (731) [1 851.7, 2 536]^{a**}	F(5, 410) = 2.1272, p = 0.06134
	> -1 SD	2 058.2 (300.5)^{b*} [1 983.1, 2133.3]	2 095.9 (286)^{b*} [2 024.4, 2167.3]	2 049.8 (267.8)^{b*} [1 982.9, 2116.7]	2 123.9 (276.1) [2 054.9, 2192.8]	2 129.9 (270.9) [2 062.2, 2 197.6]	2 307.2 (293.8) [2 233.8, 2 380.5]^{a***}	
Fat-free mass (kg)	MVP < -1 SD	17.5 (2.6) [16.4, 18.6]	19 (3.2) [17.7, 20.4]^{a*}	19.6 (3.3) [18.3, 21]	21.1 (2.8) [19.9, 22.3]^{a*}	21.4 (2.6) [20.3, 22.5]	24.3 (3.3) [22.9, 25.7]^{a}	F(5, 510) = 1.2953, p = 0.26447
	> -1 SD	22 (3.7)^{b***} [21.1, 22.8]	23.2 (3.9)^{b**} [22.3, 24.1]^{a***}	23.6 (3.9)^{b**} [22.7, 24.4]	25 (3.6)^{b**} [24.2, 25.8]^{a***}	24.9 (3.8)^{b*} [24.1, 25.7]	27.8 (4.2) [26.9, 28.7]^{a***}	
	Control < -1 SD	18.7 (2.8) [17.4, 20]	19.6 (2.8) [18.3, 20.9]	20.1 (2.8) [18.8, 21.3]	21.4 (2.9) [20.1, 22.8]	21.7 (2.7) [20.5, 22.9]	23.8 (3) [22.4, 25.2]^{a**}	F(5, 425) = 0.51802, p = 0.76268
	> -1 SD	22.3 (4.2)^{b*} [21.3, 23.3]	23.2 (4.1)^{b*} [22.2, 24.2]^{a}	23.7 (4.1)^{b*} [22.7, 24.7]	24.7 (3.6) [23.8, 25.6]^{a}	24.9 (3.6) [24, 25.7]	27.6 (4.8)^{b*} [26.4, 28.7]^{a}	

WAZ: Weight-for-age z-score; MVP group [N = 24 for children with < -1 SD and N = 79 for children with WAZ ≥ -1 sd]; Control group [N = 20 for children with < -1 SD and N = 64 for children with WAZ ≥ -1 SD]); *P=<0.05, **P < 0.001, ***P < 0.0001; MUAFA: mid-upper arm fat area; MUAMA: mid-upper arm muscle area; ^{a}Data point is compared with the previous data point in the same study group. ^{b}Data point is compared with the same data point across the two categories of initial nutritional status

3.18   Effects of Initial Nutritional Status, Based on BMI-for-age Z-score, on the Impact of the SFP
        on Growth and Body Composition in the Combined Group

There was a significant effect of initial nutritional status, based on BMI-for-age z-score, on the mean weight [$F(5, 1090) = 3.0821$, $p = 0.00907$], mid-upper arm circumference [$F(5, 1070) = 3.4139$, $p = 0.00458$], subscapular skinfold measurement [$F(5, 1085) = 2.4647$, $p = 0.03126$] and upper arm muscle circumference [$F(5, 1070) = 2.8647$, $p=0.01411$] when comparing children with initial inadequate nutritional status (BMI-for-age z-scores $\leq$ -1 SD) and those with initial adequate nutritional status (BMI-for-age z-scores > 1 sd; TABLE 3.43). Children with an initial BMI-for-age $\leq$ -1 SD had significantly lower mean MUAC at baseline (P value = 0.01) compared to those with a BMI-for-age z-score > -1 SD. This difference was not observed for MUAC in other study intervals, indicating that children with a low BMI-for-age at baseline experienced an increase in MUAC measurement to almost equal that of those with a high BMI-for-age by the 30th month.

The effect of initial nutritional status, based on BMI-for-age z-score, on the mean fat mass was nearly significant [$F(5, 1090) = 2.0646$, $p = 0.06747$]. The mean fat mass was significant for all the children by the 6th month and the 12th month compared with the preceding data points (p values < 0.000 for the children with inadequate and those with adequate nutrition based on BMI-for-age). In addition, the mean fat mass was significantly higher by the 30th month time point compared to the 24th month one in those children with an adequate initial nutritional status based on BMI-for-age (p = 0.000). Repeated measures ANOVA was not carried out for the intervention and control groups separately because there were only 10 cases with initial inadequate nutritional status based on BMI-for-age in one of the study groups.

TABLE 3.43: Initial nutritional status based on BMI-for-age by growth, body composition and time in the combined groups

Variable	BMI-AZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
Height (cm)	≤ -1 SD	125.1 (11.5) [120.5, 129.6]	128.2 (11.3) [123.7, 132.6][a***]	131.1 (11.3) [126.6, 135.6][a***]	134.1 (10.8) [129.8, 138.4][a***]	136 (10.5) [131.9, 140.2][a*]	140.1 (11.2) [135.7, 144.5][a***]	$F(5, 1090) =$ 0.68277, $p = 0.63656$
	> -1 SD	122 (10.9) [120.5, 123.6]	124.8 (10.6) [123.3, 126.3][a***]	127.2 (10) [125.8, 128.7][a***]	130.2 (9.2) [128.9, 131.5][a***]	132.9 (9.4) [131.6, 134.3][a***]	136.8 (9.8) [135.4, 138.2][a***]	
HV (cm/6 months)	≤ -1 SD		0.5 (0.2) [0.4, 0.6]	1 (0.5) [0.8, 1.2][a***]	1.5 (0.6) [1.3, 1.7][a***]	1.8 (0.6) [1.6, 2.1][a*]	2.5 (0.5) [2.3, 2.7][a***]	$F(4, 872) =$ 0.50327, $p = 0.73336$
	> -1 SD		0.5 (0.3) [0.4, 0.5]	0.9 (0.5) [0.8, 0.9][a***]	1.4 (0.6) [1.3, 1.5][a***]	1.8 (0.7) [1.7, 1.9][a***]	2.5 (0.7) [2.4, 2.6][a***]	
Weight (kg)	≤ -1 SD	22.3 (5.4) [20.2, 24.4]	24.1 (5.7) [21.8, 26.4][a**]	24.9 (5.9) [22.6, 27.2]	27.7 (7) [24.9, 30.5][a***]	27.9 (7) [25.1, 30.6]	29.8 (7.2) [26.9, 32.6][a***]	$F(5, 1090) =$ 3.0821, $p = 0.00907*$
	> -1 SD	24 (4.9) [23.3, 24.6]	25.2 (5) [24.5, 25.9][a***]	26 (4.9) [25.3, 26.7][a***]	28 (5.2) [27.2, 28.7][a***]	28.3 (5.4) [27.5, 29]	30.5 (5.7) [29.7, 31.3][a***]	
WV (kg/6 months)	≤ -1 SD		0.3 (0.2) [0.2, 0.4]	0.4 (0.2) [0.3, 0.5]	0.9 (0.4)[b*] [0.7, 1.1][a***]	0.9 (0.4) [0.8, 1.1]	1.2 (0.4) [1.1, 1.4][a***]	$F(4, 872) =$ 1.8625, $p = 0.11499$
	> -1 SD		0.2 (0.3) [0.2, 0.3]	0.3 (0.3) [0.3, 0.4][a***]	0.7 (0.3) [0.6, 0.7][a**]	0.7 (0.3) [0.7, 0.8]	1.1 (0.5) [1, 1.2][a***]	
MUAC (cm)	≤ -1 SD	16.4 (1.4) [15.8, 17]	16.8 (1.6) [16.2, 17.5]	16.8 (1.5) [16.2, 17.4]	17.6 (1.5) [17, 18.2][a*]	17.7 (1.2) [17.2, 18.2]	18.6 (2.1) [17.7, 19.4][a*]	$F(5, 1070) =$ 3.4139, $p = 0.00458*$
	> -1 SD	17.6 (1.5)[b*] [17.4, 17.8]	17.8 (1.5) [17.5, 18]	17.7 (1.5) [17.5, 17.9]	18.3 (1.5) [18, 18.5][a***]	18.4 (1.4) [18.2, 18.6]	18.9 (1.5) [18.7, 19.1][a***]	
Triceps (mm)	≤ -1 SD	4.3 (0.9) [4, 4.7]	4.7 (1.2) [4.2, 5.1]	5.5 (1.1) [5, 5.9]	5.8 (1.3) [5.3, 6.4]	6.1 (1.5) [5.5, 6.7]	6.2 (1.6) [5.5, 6.8]	$F(5, 1080) =$ 0.63622, $p = 0.67213$
	> -1 SD	4.9 (1.4) [4.6, 5.1]	5.3 (1.6) [5.1, 5.5][a**]	6 (1.7) [5.7, 6.2][a***]	6.1 (1.7) [5.9, 6.4]	6.5 (1.5) [6.3, 6.7]	6.4 (1.6) [6.1, 6.6]	
Subscapular (mm)	≤ -1 SD	4 (1.1) [3.6, 4.4]	4.1 (1) [3.7, 4.5]	5.2 (1.2) [4.8, 5.7][a***]	4.9 (1.1) [4.5, 5.4]	4.7 (1) [4.3, 5.1]	5.1 (1.1) [4.7, 5.5]	$F(5, 1085) =$ 2.4647, $p = 0.03126*$
	> -1 SD	4.4 (1.2) [4.2, 4.5]	4.6 (1.3) [4.4, 4.7]	5.1 (1) [5, 5.3][a***]	4.9 (0.9) [4.8, 5]	4.9 (1) [4.7, 5]	5.1 (1.1) [5, 5.3]	

BMI-AZ: BMI-for-age z-score ≤ -1 SD (N = 27); BMI-for-age > -1 SD (N = 193); HV: height velocity; WV: weight velocity; MUAC: mid-upper arm circumference; *P=< 0.05, **P < 0.001, ***P < 0.0001; [a]Data point is compared with the previous data point in the same study group. [b]Data point is compared with the same data point across the two categories of initial nutritional status

TABLE 3.43: Initial nutritional status based on BMI-for-age by growth, body composition and time in the combined groups (Continued)

Variable	BMI-AZ	Baseline	6 months	12 months	18 months	24 months	30 months	Repeated measures ANOVA; F statistics
				Mean (sd) [95% CI]				
Body fat (%)	≤ -1 SD	6.9 (1.7) [6.2, 7.6]	7.5 (2.6) [6.5, 8.5]	9.1 (2.5) [8.1, 10.1]^a*	9.9 (2.5) [8.9, 11]	9.7 (3) [8.5, 10.9]	10 (2.9) [8.8, 11.2]	F(5, 1075) = 0.84143, p = 0.52032
	> -1 SD	7.7 (2.6) [7.3, 8.1]	8.4 (3) [8, 8.8]	9.5 (3) [9.1, 9.9]	10.1 (2.6) [9.7, 10.4]^a*	10.2 (2.7) [9.8, 10.5]	10.2 (2.9) [9.8, 10.6]	
Fat mass (kg)	≤ -1 SD	1.6 (0.6) [1.3, 1.8]	1.8 (0.8) [1.5, 2.1]^a***	2.3 (1) [1.9, 2.7]^a***	2.7 (1.4) [2.1,3.2]	2.7 (1) [2.2, 3.1]	3.3 (1.6) [2.6, 3.9]	F(5, 1090) = 2.0646, p = 0.06747
	> -1 SD	1.8 (0.8) [1.7, 2]	2.1 (1) [2, 2.3]^a***	2.5 (1.1) [2.3, 2.6]^a***	2.6 (1.1) [2.5,2.8]	2.8 (1.1) [2.6, 3]	3.2 (1.3) [3, 3.4]^a***	
MUAFA (mm)^2	≤ -1 SD	341.3 (77.4) [310.1, 372.6]	375.1 (100.7) [334.4, 415.7]	438 (112.3) [392.6, 483.3]	492.5 (141.2) [435.5,549.5]	511.1 (142.6) [453.5, 568.6]	540.8 (155.9) [477.8, 603.8]	F(5, 1070) = 0.78694, p = 0.55910
	> -1 SD	409.5 (136.4) [390, 429]	451.7 (162.5) [428.4, 474.9]^a**	504.1 (175.9) [478.9, 529.3]^a***	534 (180.8) [508.2,559.9]	565.1 (158.6) [542.4, 587.8]	571.7 (164.2) [548.2, 595.2]	
MUAMA (mm)^2	≤ -1 SD	1 825.1 (350.4)^b* [1 683.6, 1 966.7]	1 908.8 (407) [1 744.4, 2 073.2]	1 868.1 (356.3) [1 724.2, 2012]	2 011.6 (355.6) [1 868, 2155.3]	1 997.7 (256.5) [1 894.1, 2101.3]	2 258.2 (663) [1 990.4, 2526.1]^a***	F(5, 1070) = 2.8647, p = 0.01411*
	> -1 SD	2 079.9 (360.2) [2 028.3, 2 131.4]	2 092.1 (345.4) [2 042.7, 2 141.6]	2 053.9 (329.8) [2 006.7, 2 101.1]^a***	2 154.8 (350.7) [2 104.6, 2205]	2 164.3 (323.8) [2 118, 2210.6]	2310.6 (357.5) [2259.5, 2361.8]^a***	
Fat-free mass (kg)	≤ -1 SD	20.8 (4.9) [18.8, 22.7]	22.3 (5.2) [20.2, 24.3]^a*	22.6 (5.1) [20.6, 24.6]	24.1 (5.8) [21.8, 26.4]^a*	23.5 (4.3) [21.8, 25.2]	26.6 (6) [24.3, 29]^a***	F(5, 1090) = 1.0188, p = 0.40513
	> -1 SD	22.1 (4.4) [21.5, 22.7]	23.1 (4.5) [22.5, 23.7]^a***	23.5 (4.3) [22.9, 24.1]	24.6 (3.9) [24.1, 25.2]	24.8 (4.2) [24.2, 25.4]	27.6 (4.7) [27, 28.3]^a***	

BMI-AZ: BMI-for-age z-score ≤ -1 SD (N = 27); BMI-for-age > -1 SD (N = 193); MUAFA: mid-upper arm fat area; MUAMA: mid-upper arm muscle area; *P < 0.05, **P < 0.001, ***P < 0.0001; ^{a}Data point is compared with the previous data point in the same study group. ^{b}Data point is compared with the same data point across the two categories of initial nutritional status

3.19   Effects of Initial Nutritional Status, Based on Height-for-age Z-score, on the Impact of the School Feeding Programmes on Haemoglobin Concentration for all the Children Combined

TABLE 3.44 presents the results of the effects of initial nutritional status, based on HAZ, on the impact of the SFP on haemoglobin status. The results are presented first for all the children combined, and then in the separate groups. Repeated measures ANOVA comparing the mean Hb concentration over time with initial nutritional status showed a marginal level of significant interaction between the initial nutritional status based on height-for-age z-score, over the three measurement intervals [$F(2, 436) = 2.97$, $p = 0.055$; TABLE 3.44]. The Bonferroni post hoc test did not show any significant differences in mean Hb concentration at any of the measurement intervals between children with adequate and those with inadequate initial nutritional status based on HAZ. When the mean Hb concentration was considered in each of the two categories of initial nutritional status separately, significant increases were observed at all the three measurement intervals (p values < 0.001).

The results of repeated measures ANOVA for serum ferritin concentration did not show any significant interaction between initial nutritional status and time for all the children combined (TABLE 3.46). Post hoc tests did not show any significant differences in the mean serum ferritin concentration over time for all the children combined. When groups were considered separately, based on initial height-for-age, there was no interaction between initial nutritional status and serum ferritin concentration over time. Due to the limitation of the serum ferritin sample size, an analysis was not done to compare the effects of initial nutritional status on the basis of weight-for-age and BMI-for-age z-scores in each of the study groups.

3.19.1 Effects of initial nutritional status, based on height-for-age z-score, on the impact of the school feeding programmes on haemoglobin concentration by study group

When the effects were considered in each of the groups separately, there was a significant interaction between the initial nutritional status, based on height-for-age z-scores, and mean Hb concentration over the three measurement intervals [$F(2, 232) = 5.32$, $p = 0.00551$] for the MVP children (TABLE 3.44). Children in the MVP group with initial inadequate nutritional status (height-for-age < -1 SD) had a significantly higher ($P < 0.0001$) increase in mean Hb concentration from baseline (Hb concentration of 9.1 g/dl) to 6 months (Hb concentration of 11.8 g/dl) compared to those with adequate initial nutritional status (height-for-age > -1 SD), whose mean Hb concentration increased from 9.8 g/dl at baseline to 11.8 g/dl at 6 months. In the control group, the

interaction between change in mean Hb concentration over time and initial nutritional status based on height-for-age was not significant [$F_{(2, 200)} = 1.4$, $p = 0.25479$]. During the first 6 months of the study, when there was no school feeding programme in the control group, the mean Hb concentration did not change significantly among the children with an initial inadequate nutritional status based on height-for-age z-score ($p = 0.07$). However, those in this group with an initial adequate nutritional status showed a significant increase in mean Hb concentration from baseline to 6 months and to 24 months (p values = 0.03 and < 0.0001; Table 3.34).

3.20 Effects of Initial Nutritional Status, Based on Weight-for-age Z-score, on the Impact of the School Feeding Programmes on Haemoglobin Concentration for all Children Combined

Overall, the repeated measures ANOVA comparing the mean Hb concentration over time with initial nutritional status showed that there was a significant interaction between the initial nutritional status based on weight-for-age z-score and the mean Hb concentrations over the three measurement intervals [$F_{(2, 378)} = 3.97$, $p = 0.021$; TABLE 3.44]. The initial nutritional status had an effect on the changes in mean Hb concentration for all the children in the two study groups combined. Children with a weight-for-age z-score < -1 SD had a significantly higher increase in mean Hb concentration from baseline (9.2 g/dl) to 6 months (11.3 g/dl) compared to the increase among those with an initial weight-for-age > -1 SD (from 10.0 g/dl to 11.5 g/dl) ($p = 0.01$).

3.20.1 Effects of initial nutritional status, based on weight-for-age z-score, on the impact of the school feeding programmes on haemoglobin concentration by study group

When the repeated measures ANOVA was done for separate study groups, there was no significant interaction between initial nutritional status based on weight-for-age z-scores and changes in mean Hb concentration over time for the MVP group [$F_{(2, 204)} = 2.4343$, $p = 0.0902$] or for the controls [$F_{(2, 170)} = 1.9853$, $p = 0.14052$]. Furthermore, the Bonferroni post hoc comparisons showed significant changes in Hb concentration for each of the initial nutritional status categories for both the MVP and control groups and across all three data points (TABLE 3.45).

3.21 Effects of Initial Nutritional Status, Based on BMI-for-age Z-score, on the Impact of the School Feeding Programmes on Haemoglobin Concentration for all Children Combined

The interaction between initial nutritional status, based on BMI-for-age z-scores, and mean Hb concentration over time was significant [$F_{(2, 436)} = 5.251$, $p = 0.006$] for all the children combined

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(TABLE 3.45). The mean Hb concentration increased significantly from baseline to 6 months (ANOVA: P < 0.001). The mean Hb concentration was not significantly different at 24 months compared to 6 months for children with an initial inadequate nutritional status based on BMI-for-age z-scores, although the Hb concentration at 24 months was significantly higher (ANOVA: P < 0.001) than at 6 months for children with an initial adequate nutritional status. Repeated measures ANOVA was not carried out for the intervention and control groups separately because there were only 10 cases with initial inadequate nutritional status based on BMI-for-age in one of the study groups.

3.21.1 Effects of initial nutritional status on the impact of the school feeding programmes on serum ferritin concentration for all the children combined and for separate study groups

The results of repeated measures ANOVA for serum ferritin concentration did not show any significant interaction between initial nutritional status and time for all the children combined (TABLE 3.46). Post hoc tests did not show any significant differences in the mean serum ferritin concentration over time for all the children combined. When groups were considered separately based on initial height-for-age, there was no interaction between initial nutritional status and serum ferritin concentration over time. Due to the limitation of the serum ferritin sample size, analysis was not done to compare the effects of initial nutritional status on the basis of weight-for-age and BMI-for-age z-scores in each of the study groups.

TABLE 3.44: Initial nutritional status based on height-for-age by haemoglobin concentration, time and study group

	Haemoglobin (g/dl); Mean (sd) [CI]			Repeated measures ANOVA; F statistics	Bonferroni test: p[a]	Bonferroni test: p[b]	Bonferroni test: p[c]
	Baseline	6 months	24 months				
Height-for-age							
All	*Mean (sd) [95% CI]*						
≤ -1 SD (N = 93)	9.6 (1.7) [9.3, 10]	11.4 (1.5) [11.1, 11.7]	12.7 (0.5) [12.6, 12.8]	F(2, 436) = 2.97, p = 0.055	0.000	0.000	0.000
> -1 SD (N = 127)	10.2 (1.5) [9.9, 10.4]	11.7 (1.4) [11.5, 12]	12.7 (0.8) [12.6, 12.9]		0.000	0.000	0.000
MVP	9.6 (1.6) [9.3, 9.8]	11.8 (1) [11.6, 12]	12.8 (0.8) [12.6, 12.9]	F(2, 232) = 5.32, p = 0.00551*			
≤ -1 SD (N = 44)	9.1 (1.6)[d]* [8.6, 9.6]	11.8 (0.9) [11.5, 12.1]	12.8 (0.4) [12.7, 13]		0.000	0.000	0.0001
> -1 SD (N = 74)	9.8 (1.5) [9.5, 10.2]	11.8 (1) [11.6, 12]	12.7 (0.9) [12.5, 12.9]		0.000	0.000	0.000
Control	10.4 (1.5) [10.1, 10.7]	11.2 (1.8) [10.8, 11.5]	12.7 (0.7) [12.5, 12.8]	F(2, 200) = 1.4, p = 0.25479			
≤ -1 SD (N = 49)	10.1 (1.6) [9.7, 10.6]	10.9 (1.8) [10.4, 11.4]	12.6 (0.7) [12.4, 12.8]		0.07	0.000	0.000
> -1 SD (N = 53)	10.7 (1.3) [10.3, 11]	11.4 (1.9) [10.9, 12]	12.7 (0.8) [12.5, 12.9]		0.03	0.000	0.000

[a] P value compares baseline to 6 months; [b] p value compares baseline and 24 months; [c] p value compares 6 months and 24 months; [d] mean Hb concentration at baseline significantly higher for those with height-for-age > -1 SD; p value = 0.01

TABLE 3.45: Initial nutritional status based on weight-for-age and BMI-for-age by haemoglobin concentration, time and study group

Weight-for-age	Haemoglobin (g/dl); Mean (sd) [CI]			Repeated measures ANOVA; F statistics	Bonferroni test: p^a	Bonferroni test: p^b	Bonferroni test: p^c
	Baseline	6 months	24 months				
All	*Mean (sd) [95% CI]*						
≤ -1 SD	9.3 (1.4)^d*	11.4 (1.7)	12.7 (0.6)	F(2, 378) = 3.97,	0.00	0.00	0.000
(N = 45)	[8.8, 9.7]	[10.9, 11.9]	[12.6, 12.9]	p = 0.021*			
> -1 SD	10.0 (1.6)	11.6 (1.5)	12.7 (0.8)		0.00	0.00	0.00
(N = 146)	[9.8, 10.3]	[11.3, 11.8]	[12.6, 12.8]				
MVP	9.5 (1.6)	11.8 (1.0)	12.7 (0.8)	F(2, 204) = 2.4343,			
	[9.2, 9.8]	[11.6, 11.9]	[12.6, 12.9]	p = 0.09020			
≤ -1 SD	9.0 (1.3)	11.7 (0.7)	12.9 (0.4)		0.000	0.000	0.002
(N = 24)	[8.5, 9.6]	[11.4, 12]	[12.7, 13.1]				
> -1 SD	9.6 (1.6) [9.3, 10]	11.8 (1.1)	12.7 (0.9)		0.000	0.000	0.000
(N = 80)		[11.5, 12]	[12.5, 12.9]				
Control	10.3 (1.4)	11.1 (2)	12.6 (0.8)	F(2, 170) = 1.9853,			
	[9.9, 10.6]	[10.7, 11.6]	[12.5, 12.8]	p = 0.14052			
≤ -1 SD	9.5 (1.5)	11 (2.4)	12.5 (0.8)		0.006	0.000	0.003
(N = 21)	[8.8, 10.2]	[9.9, 12.1]	[12.2, 12.9]				
> -1 SD	10.5 (1.3)	11.2 (1.9)	12.6 (0.7)		0.03	0.000	0.000
(N = 66)	[10.2, 10.8]	[10.7, 11.6]	[12.5, 12.8]				
BMI-for-age							
All							
≤ -1 SD	9.5 (1.1)	12 (1.6)	12.9 (0.6)	F(2, 436) = 5.251,	0.00	0.000	0.066
(N = 27)	[9, 9.9]	[11.4, 12.7]	[12.7, 13.2]	p = 0.006**			
> -1 SD	10.0 (1.6)	11.5 (1.4)	12.7 (0.7)		0.000	0.000	0.000
(N = 193)	[9.8, 10.3]	[11.3, 11.7]	[12.6, 12.8]				

[a] P value compares baseline to 6 months; [b] p value compares baseline and 24 months; [c] p value compares 6 months and 24 months; [d] mean Hb concentration at baseline significantly higher among those with weight-for-age > -1 SD; p value = 0.01; * Statistically significant at P < 0.05

TABLE 3.46: Effect of initial nutritional status on serum ferritin concentration by study group and time

	Serum ferritin (µg/l); Mean (sd) [CI]			Repeated measures ANOVA; F statistics	Bonferroni test: p[a]	Bonferroni test: p[b]	Bonferroni test: p[c]
	Baseline	6 months	24 months				
Height-for-age z-score							
≤ -1 SD (N = 32)	40.8 (27.1) [33.0, 48.5]	34.2 (24.5) [25.3, 43.0]	37.5 (21.7) [29.7, 45.3]	F(2, 164) = 0.26, p = 0.77	1.0	1.0	1.0
> -1 SD (N = 52)	42.0 (27.4) [32.6, 51.4]	32.8 (22.6) [26.6, 39.1]	40.5 (29.6) [32.3, 48.8]		0.29	1.0	0.62
MVP (N = 43)	35.8 (35.8) [27.8, 43.8]	31.6 (23) [24.5, 38.7]	34.7 (24.4) [27.2, 42.2]	F(2, 82) = 0.26935, p = 0.76455			
≤ -1 SD(N = 15)	35.4 (35.4) [16.2, 54.6]	32.7 (29.1) [16.5, 48.8]	31.9 (21.6) [20, 43.9]		1.0	1.0	1.0
> -1 SD(N = 28)	36 (36) [27.9, 44]	31.1 (19.6) [23.5, 38.7]	36.2 (26) [26.1, 46.3]		1.0	1.0	1.0
Control (N = 41)	47.1 (47.1) [38.5, 55.7]	35.1 (23.6) [27.7, 42.6]	44.3 (28.5) [35.3, 53.3]	F(2, 78) = 0.10114, p = 0.90392			
≤ -1 SD (N = 17)	45.4 (45.4) [34.3, 56.5]	35.5 (20.4) [25, 46]	42.4 (21.3) [31.5, 53.4]		1.0	1.0	1.0
> -1 SD (N = 24)	48.2 (48.2) [35.2, 61.3]	34.9 (26) [23.9, 45.9]	45.6 (33.1) [31.6, 59.5]		1.0	0.39	1.0
Weight-for-age z-score[d]							
≤ -1 SD(N = 17)	39.8 (22.2) [33.8, 45.8]	27.9 (18.2) [18.5, 37.2]	38.8 (25.9) [25.4, 52.1]	F(2, 144) = 0.58, p = 0.56	0.5	1.0	1.0
> -1 SD(N = 57)	41.0 (33.8) [24.7, 57.3]	33.6 (22.8) [27.5, 39.6]	39.5 (28.4) [32, 47.1]		1.0	1.0	1.0
BMI-for-age z-score[d]							
≤ -1 SD(N = 10)	40.7 (26.7) [34.4, 47]	34.1 (14.1) [24, 44.2]	43.2 (18.9) [29.7, 56.7]	F(2, 164) = 0.27, p = 0.76	1.0	1.0	1.0
> -1 SD(N = 74)	44.8 (29.7) [25.8, 63.7]	33.2 (24.3) [27.6, 38.9]	38.9 (27.7) [32.4, 45.3]		0.35	1.0	1.0

[a] P compares baseline to 6 months; [b] p value compares baseline and 24 months; [c] p value compares 6 months and 24 months; [d] Data on within-study group comparisons for initial nutritional status based on weight-for-age and BMI-for-age z-scores not presented because numbers are less than 10 in some categories with less than 10 subjects.

An adjusted logistic regression model was used to determine the effect of initial nutritional status to the impact of the school feeding programme on the prevalence of anaemia and body iron stores for all children combined. In order to adjust the regression model, factors that were identified as significant determinants of anaemia and low body iron stores at baseline were added to the model. The factors included in the model were initial nutritional status based on height-for-age, weight-for-age and BMI-for-age ≤ -1 SD, study group, gender of household head, child's gender and mother's employment status. Children who had an inadequate initial nutritional status, as defined by height-for-age ≤ -1 at baseline, were three times (significantly) (p = 0.02) more likely to be anaemic by the 6[th] month compared to those who had an initial height-for-age z-score > -1 sd (TABLE 3.47). Although not statistically significant, children who had an inadequate initial nutritional status as defined by weight-for-age ≤ -1 at baseline were 3.4 and 11 times more likely to have low body iron stores by the 6[th] and 24[th] months of study, compared to children who had good nutritional status at baseline (weight-for-age > -1).

Other factors that determined the likelihood of being anaemic or having low body iron stores included study group and household headship. Children in households headed by mothers or grandparents were 3.5 times more likely to be anaemic by the 24[th] month compared to those headed by fathers. Children in the MVP group were 4 times and 10.4 times more likely to have low iron stores by the 6[th] and 24[th] months of the study compared to those in the control group (logistic regression; p = 0.06 and p = 0.02 respectively).

TABLE 3.47: Adjusted odds of anaemia and low iron stores by initial nutritional status, socio-demographic characteristics, and time

	Anaemia						Low iron stores					
	Baseline	logistic regression; p	6 months	logistic regression; p	24 months	logistic regression; p	Baseline	logistic regression; p	6 months	logistic regression; p	24 months	logistic regression; p
Height-for-age	*Odds ratio [95% CI]*		*Odds ratio [95% CI]*		*Odds ratio [95% CI]*		*Odds ratio [95% CI]*		*Odds ratio [95% CI]*		*Odds ratio [95% CI]*	
≤ -1 SD	2.2 [1.0, 5.0]	0.06	3 [1.2, 7.4]	0.02*	0.4 [0.1, 2]	0.26	1 [0.2, 5.3]	0.97	0.8 [0.2, 4.3]	0.84	0.5 [0.1, 4.0]	0.54
> -1 SD[rc]		1.0		1.0		1.0				1.0		1.0
Weight-for-age												
≤ -1 SD	1.2 [0.4, 3.5]	0.71	0.8 [0.3, 2.6]	0.74	1 [0.2, 6.1]	0.97	1.8 [0.2, 16.5]	0.6	3.4 [0.4, 27.3]	0.26	11.1 [0.9, 139.8]	0.06
> -1 SD[rc]		1.0		1.0		1.0				1.0		1.0
BMI-for-age												
≤ -1 SD	3.2 [0.8, 12.7]	0.11	0.4 [0.1, 2.0]	0.28	[a]		0.8 [0, 15.3]	0.88	0.3 [0.5, 8.0]	0.45	0.2 [0.5, 4.0]	0.36
>-1 SD[rc]		1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Gender												
Male	0.8 [0.4, 1.5]	0.46	0.9 [0.4, 2.0]	0.82	1.7 [0.5, 6.0]	0.39	2.6 [0.6, 11.9]	0.2	2.9 [0.7, 11.6]	0.14	4.1 [0.8, 21.9]	0.1
Female[rc]		1.0		1.0		1.0		1.0		1.0		1.0
Study group												
MVP	2.6 [1.3, 5.0]	0.01*	0.1 [0, 0.3]	0.000**	0.4 [0.1, 1.5]	0.17	4.7 [1, 22.5]	0.05	4 [1, 17.1]	0.06	10.4 [1.5, 7.2]	0.02*
Control[rc]	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Household head												
Mother and others	1.8 [0.8, 3.8]	0.16	1.1 [0.4, 3.0]	0.78	3.5 [0.9, 12.8]	0.06	0.8 [0.2, 3.9]	0.79	1.5 [0.3, 6.4]	0.61	2.7 [0.5, 15.4]	0.25
Father[rc]		1.0		1.0		1.0		1.0		1.0		1.0
Mother's employment status												
None Employed	1.1 [0.5, 2.3]	0.87	1.0 [0.4, 2.3]	0.92	0.6 [0.2, 2.0]	0.37	0.5 [0.1, 2.4]	0.38	0.6 [0.1, 2.5]	0.44	0.7 [0.1, 3.8]	0.65
Self-employed[rc]		1.0		1.0		1.0		1.0		1.0		1.0

[a] Model not fit for BMI-for-age z-score by anaemia; [rc] Reference category *P values significant at p value < 0.05; **P values significant at p value < 0.01

3.22   Effects of Initial Nutritional Status on the Impact of the School Feeding Programme on Serum Retinol Concentration and Vitamin A Status for all the Children Combined and by Group

Repeated measures ANOVA showed no significant interaction between serum retinol concentration and initial nutritional status based on height-for-age and weight-for-age z-scores for all the children combined. Repeated measures ANOVA showed a significant interaction between the change in mean serum retinol concentration by baseline BMI-for-age z-score over time [$F_{(2, 184)} = 4.4036$, $p = 0.013$; TABLE 3.48].

Among children with a BMI-for-age $> $-1 SD at baseline, serum retinol concentration increased significantly ($p = 0.002$) from baseline to the sixth month, but this was offset by a significant decrease ($p = 0.01$) from the 6[th] month to the 24[th] month. In contrast, children with a BMI-for-age z-score $< $-1 SD at baseline the mean serum retinol concentration decreased slightly from baseline to six months, and then increased significantly ($p = 0.01$) from the 6[th] to the 24[th] months of the study. No significant differences were observed when the effect of initial nutritional status was assessed comparing the MVP and control groups. Due to the limitation of the serum retinol sample size, analysis was not done to compare the effects of initial nutritional status on the basis of weight-for-age and BMI-for-age z-scores in each of the study groups.

An adjusted logistic regression model was used to determine the effect of initial nutritional status on the impact of the school feeding programme on vitamin A status. In order to adjust the regression model, factors that were identified as significant determinants of inadequate vitamin A status at baseline were included in the model. Children who had a weight-for-age $\leq$ -1 SD at baseline were 2.6 and 2.1 times more likely to have inadequate vitamin A status by the sixth and 24[th] months of the study, compared to those with a weight-for-age $> $-1 SD. This was not statistically significant. Initial nutritional status, based on height-for-age and BMI-for-age, did not have a significant effect on the inadequate vitamin A status by the 6[th] and 24[th] months of the study (TABLE 3.49).

TABLE 3.48: Serum retinol concentration by initial nutritional status by group and time

	Baseline	6 months	24 months	Repeated measures ANOVA; F statistics	Bonfe rroni test: p[a]	Bonfe rroni test: p[b]	Bonfe rroni test: p[c]
Height-for-age z-score	Serum retinol (µmol/L) Mean (sd) [CI]						
≤ -1 SD (N = 36)	0.75(0.28) [0.66,0.85]	0.73(0.61) [0.52,0.94]	0.78(0.2) [0.71,0.85]	F(2, 184) = 0.63016,	1.0	1.0	1.0
> -1 SD (N = 58)	0.72(0.19) [0.67,0.77]	0.64(0.21) [0.58,0.69]	0.79(0.4) [0.68,0.9]	p = 0.53	1.0	1.0	0.13
MVP (N = 51)	0.68(0.2) [0.61,0.74]	0.71(0.54) [0.54,0.88]	0.79(0.34) [0.68,0.9]	F(2, 98) = 0.79859,			
≤ -1 SD (N = 19)	0.69(0.27) [0.55,0.83]	0.81(0.84) [0.38,1.24]	0.83(0.22) [0.72,0.94]	p = 0.45287	1.0	1.0	1.0
> -1 SD (N = 32)	0.67(0.14) [0.61,0.73]	0.64(0.16) [0.58,0.7]	0.76(0.4) [0.6,0.93]		0.6	1.0	0.7
Control (N = 43)	0.78(0.24) [0.71,0.85]	0.64(0.26) [0.57,0.71]	0.79(0.34) [0.69,0.88]	F(2, 82) = 0.42226,			
≤ -1 SD (N = 17)	0.81(0.29) [0.67,0.95]	0.66(0.29) [0.52,0.8]	0.74(0.17) [0.65,0.82]	p = 0.65698	1.0	1.0	1.0
> -1 SD (N = 26)	0.76(0.21) [0.68,0.84]	0.63(0.24) [0.54,0.72]	0.81(0.41) [0.67,0.96]		1.0	1.0	1.0
Weight-for-age z-score							
≤ -1 (N = 19)	0.7(0.26) [0.58,0.83]	0.81(0.8) [0.43,1.19]	0.84(0.46) [0.62,1.06]	F(2, 164) = 1.4359,	1.0	1.0	1.0
> -1 (N = 65)	0.73(0.21) [0.67,0.78]	0.62(0.22) [0.57,0.68]	0.77(0.32) [0.69,0.85]	p = 0.24	1.0	1.0	0.21
BMI-for-age z-score							
≤ -1 (N = 10)	0.81(0.29) [0.61,1.02]	1.05(1.06)*[e] [0.29,1.8]	0.8(0.3) [0.58,1.01]	F(2, 184) = 4.4036,	1.0	1.0	1.0
> -1 (N = 84)	0.72(0.22) [0.67,0.77]	0.62(0.22) [0.58,0.67]	0.79(0.35) [0.71,0.86]	p = 0.013*	0.7	1.0	0.015 *

[a] P compares baseline to six months; [b] p value compares baseline and 24 months; [c] p value compares six months and 24 months, [d] Data on within-study group comparisons for initial nutritional status based on weight-for-age and BMI-for-age z-scores not presented because numbers are less than 10 in some categories with less than 10 subjects. [e] Serum retinol concentration significantly higher among children with BMI-for-age ≤ -1 SD (n = 10; p = 0.002) in the 6th month study interval. *Statistically significant at P < 0.05

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TABLE 3.49: Adjusted odds of inadequate vitamin A status[a] by initial nutritional status, socio-demographic characteristics, and time

	Baseline	logistic regression; p	6 months	logistic regression; p	24 months	logistic regression; p
			Odd Ratio [95% CI]			
Height-for-age < -1						
≤ -1 SD	0.9 [0.3, 3.0]	0.92	1.0 [0.3, 3.3]	0.98	0.6 [0.2, 1.8]	0.36
> -1 SD	1.0		1.0		1.0	
Weight-for-age						
≤ -1 SD	3.8 [0.7, 21.5]	0.13	2.6 [0.4, 17.7]	0.32	2.1 [0.4, 10.8]	0.36
> -1 SD	1.0		1.0		1.0	
BMI-for-age						
≤ -1 SD	0.1 [0, 0.9]	0.04*	0.2 [0, 2.1]	0.19	0.4 [0, 3.1]	0.36
> -1 SD	1.0		1.0		1.0	
Gender						
Male	0.6 [0.2, 1.5]	0.25	0.8 [0.3, 2.2]	0.7	0.7 [0.3, 1.8]	0.44
Female	1.0		1.0		1.0	
Study group						
MVP	0.5 [0.2, 1.4]	0.19	1 [0.4, 2.7]	0.97	1.3 [0.5, 3.4]	0.54
Control	1.0		1.0		1.0	
Household head						
Mother and others	1 [0.3, 2.8]	0.93	0.6 [0.2, 1.7]	0.31	1.2 [0.4, 3.4]	0.73
Father	1.0		1.0		1.0	
Mother's employment status						
None	1.7 [0.5, 5.1]	0.37	1.2 [0.4, 3.6]	0.78	0.7 [0.2, 2]	0.52
Employed	1.0		1.0		1.0	

*P values significant at p value < 0.05; [a] Inadequate vitamin A status as determined by serum retinol < 0.70 µmol/L

3.23   Summary of Findings in Section C

An analysis of the impact of school feeding programme based on initial nutritional status showed significant effects of initial nutritional status, based on height-for-age z-scores, on the mean height, height velocity and weight. It was notable that, by the 24[th] month, the children with an initial inadequate nutritional status (height-for-age ≤ -1 SD) had gained significantly more mean height velocity compared to those with an initial adequate nutritional status (height-for-age z-score > -1 SD). Fat mass and fat-free mass were significantly higher among children who had an initial adequate nutritional status, based on height-for-age z-score, by the 18[th] and 30[th] months of the study. Fat-free mass was significantly higher among children who had an initial adequate nutritional status based on height-for-age z-score in all the study intervals. In the MVP group, the initial nutritional status on the basis of height-for-age ≤ -1 SD had an effect on the impact of the school feeding programme on height, height velocity, weight and weight velocity. Those with an initial height-for-age ≤ -1 SD in the MVP group had a significant higher height velocity by the 24[th] month

compared to those with an initial height-for-age z-score > -1 SD in this group. Among the control children there was a significant effect of the initial height-for-age z-scores on the impact of the school feeding programme on the mean height and height velocity, although the Bonferroni analysis did not show any significant differences between groups.

The effects of initial nutritional status on the impact of the school feeding programme based on weight-for-age among all the children in the MVP group showed that the children with an initial lower weight-for-age ≤ -1 SD showed less gains in height, height velocity, mid-upper arm circumference and mid-upper arm muscle area compared to their counterparts with an initial adequate nutritional status (weight-for-age > -1 SD) at 30 months. In the control group, the effect of initial weight-for-age was observed for the changes in the mean subscapular skinfold fat mass and measurement. The effects of initial nutritional status based on BMI-for-age was significant for MUAC in all the children. Those with an initial BMI-for-age < -1 SD had lower MUAC measurements at baseline, but did not differ at the rest of the study intervals. The effects of initial nutritional status based on BMI-for-age were not compared between the two study interventions because of few subjects with a low BMI-for-age < -1.

There was a marginal level of significant interaction between the initial nutritional status based on height-for-age z-score with Hb concentration over time. Children in the MVP group with initial inadequate nutritional status based on height-for-age < -1 SD and weight-for-age z-score < -1 SD had a higher improvement of the mean Hb concentration from baseline during the first 6 months of the study compared to those with adequate initial nutritional status.

There were no significant interactions between serum ferritin concentration and initial nutritional status and time. The factors that determined the likelihood of being anaemic or having low body iron stores included study group and household headship. Children in households headed by mothers or grandparents were 3 times more likely to be anaemic by the 24[th] month time point compared to those headed by fathers.

Children with initial adequate nutritional status based on BMI-for-age > -1 SD had a significant gain in serum retinol concentration from baseline to the 6[th] month time point, followed by a significant decrease from the 6[th] month to the 24[th] month time points. In contrast, children with inadequate nutritional status based on BMI-for-age z-score < -1 SD had a slight decrease in serum retinol concentration from baseline to the six months time point, and then a significant increase from the 6[th] to the 24[th] months time points of the study.

4   CHAPTER FOUR: DISCUSSION

4.1   Introduction

This was a longitudinal controlled intervention study designed to assess the effects of initial nutritional status on the responses to two different school feeding programmes in the Millennium Villages Project in Siaya District, rural Kenya. A minimal drop-out rate was observed in this study, as the sampled children remained in the same schools during the 30 months of follow-up. Understanding the effect of initial nutritional status on the responses to school feeding programmes offers important guidance for a programmatic approach to designing well-targeted interventions that meet the requirements of children with compromised nutritional status. The participation of the local community in the management of the project and the implementation of the programme provides an appropriate model for the sustainability and continuity of school feeding programmes.

4.1.1   Overall summary of project

This study was conducted among school children attending schools in the Mmillennium Villages Project in Siaya, Kenya. The intervention group was children attending schools in the village project and the controls were children attending school outside of the MVP area up to the 6[th] month of the study. The control area was included in the MVP as a scale-up programme 6 months into the study and children were given the basic school meal throughout the study. This unexpected change in design did not cause any ethical dilemma because the control children were in different schools. There was not inter-school transfers observed during the period of study. The school feeding intervention provided one-quarter of the daily EER while the control school meal provided 21% of daily EER The intervention school meal included animal source foods (small whole fish and beef) which was not included in the school feeding among the controls.

Children were followed up for 30 months during which dietary intake and socio-demographic data was collected once at the baseline, anthropometric assesments were taken every six months and blood sampling was done three times at baseline, 6 months and 24 months. No food wastage or lack of meal attendance was observed during the meal times and the menus were acceptable to the children. The children had been involved in selecting the menu items at the planning stage and thus the compliance with the diets. The portion sizes were also served as per the pre-determined amounts and in respective classes. This maintained an appropriate portion size for the age of the children and avoided wastage. The implementation of the school feeding programmewas well coordinated between the MV Project, the school management committees, the school feeding teacher in charge and the Principle investigator. This ensured adherence to the protocol in regard to food distribution. The regular meetings between all the mentioned stakeholders ensured timely resolution of possible

challenges such as delay in transportation and storage of materials. This close coordination made the implementation of the school feeding programmea success.

4.2   Summary of the Baseline Observations

Majority of the children in the study lived in male-headed households and most of the mothers had attained primary level of education. The socioeconomic characteristics of the two study groups were comparable at baseline, except for household headship, as the percent of households headed by mothers and grandparents was higher in the control group than in the MVP group. In general, the dietary intake of the children lacked variety, as it comprised predominantly staple starches, with a limited intake of milk, grains, legumes and total fats. In addition, a high prevalence of inadequate nutrient intake was observed among the children in all the groups at baseline. No significant differences were observed between study groups in regard to the levels of nutrient intake and in the prevalence of inadequate nutrient intake at baseline. Stunting and linear growth deficit were the main forms of undernutrition among the children. One in every six children was stunted at baseline, with linear growth deficit affecting almost half of the children. This was observed to be more common in males and younger children. Wasting occurred more among the females and older children. No significant differences were observed in regard to the prevalence of undernutrition between the two study groups. The children's age and maternal employment status were significant determinants of linear growth deficit. The boys were leaner than the girls. No significant differences were observed between the two study groups with regard to body composition measurements.

The mean full blood count parameters were significantly different between the groups at baseline. Anaemia was of severe public health significance in this population at baseline – above the WHO cut-off of 40% prevalence – with a significantly higher prevalence among the MVP group than the controls and higher among younger children than older ones. The presence of elevated CRP levels was observed in twelve percent of the all the children in the study, with a greater magnitude in the control group, the boys and the younger children. The factors associated with anaemia and body iron stores at baseline were; household headship, children's age, sex and undernutrition status.

The mean serum retinol concentration was higher in the MVP group than in the control group. One child in the control group was vitamin A deficient (serum retinol < 0.35 µmol/L). There was evidence of an inadequate vitamin A status as measured by serum retinol < 0.70 µmol/L in more than one third of the children at baseline. This was more common in the control group. Employment status of the household head, gender and the children's age were the factors associated with vitamin

A status. Multiple micronutrient deficiencies were found in this population, with 8% of the children having iron deficiency combined with inadequate vitamin A status.

4.2.1   Dietary Intake

The first objective of this study was to determine the dietary intake of the school children in the MVP and control groups at baseline, using a six-month Quantitative Food Frequency Questionnaire (QFFQ). The food frequency questionnaire contained a comprehensive list of 96 food items developed in consultation with the community, and can be assumed to cover all the foods consumed by the children at the time of the study. However, it did not reflect the seasonal variability of the food items, because the foods that were not in season at the time of the survey may have been omitted from the questionnaire as a result of recall bias. Parental involvement has been recommended when investigating children's dietary intake, especially of fruit and vegetables, because parents are believed to offer more objective information on the accessibility of foods at home.[305] This is especially so in environments where children do not have alternative sources of food apart from what is eaten at home. The main respondent for the QFFQ in the current study was the mother, or the immediate caregiver in the absence of the mother. This was necessitated by the fact that school children are known to under-report dietary intake.[306-309] Underreporting by school children is more common when using semi-quantitative QFFQ, which assume that participants consume a standard portion size.[307] Overestimation of dietary intake has also been observed in self-reported validation studies among school-age children.[310,311]

The findings at baseline revealed inadequate nutrient intake among all the school children. There was a high probability of inadequate intake of total energy, total fat, folate, zinc, selenium and calcium among all the children. The mean energy intake was inadequate compared with the energy requirements of children who are eight years old with moderate physical activity (8 079 kJ/1 931 kcals for boys and 7 573 kJ/1 810 kcals for girls).[37] The main energy source for the children was from carbohydrates, which was observed to be slightly above the RDA for the two groups of children. Similarly, protein intake was reported as adequate in relation to RDA. However, the quality of the proteins was not examined in this study.

The usual fat intake contributed less than 25% to the total energy intake. When interpreting the inadequacy of fat intake in this study, it is important to note that the dietary fat intake was estimated on the basis of the total amount of fat used for a common household meal, rather than an individual's direct fat consumption. This was necessitated by a lack of consumption of direct fats

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and oils by the individual children, such as the consumption of margarine and other bread spreads, which was not reported by any child in this rural population. Although food frequency questionnaires are known to underestimate fat intake,[312] there was no reported intake of high-fat foods by the children. Dietary fat intake in a study among rural Chinese children aged 2 to 15 years was reported to be as low as 16.4% of total energy intake.[313] Fats provide a dense energy source, while providing essential fatty acids and aiding in the absorption of fat-soluble vitamins, as well as enhancing the texture, flavour and aroma of foods.[314] Children with a low fat intake are at risk of an inadequate intake of n-3 fatty acids, which are essential for the growth of brain cells.[70] Randomised controlled trials with n-3 fatty acid supplementation among school children have shown a beneficial effect on verbal learning and memory.[315] The usual diet was mainly plant based, with limited animal-source foods. Previous studies in Kenya have reported predominantly plant-based diets among school children.[43,50]

The prevalence of multiple micronutrient deficiencies in developing countries is associated with an inadequate intake of animal-source foods.[50] Animal-source foods are nutrient dense, providing protein, energy, fats, iron and other micronutrients with a high biological value.[50] Despite the predominance of plant-source foods in the current study, the mean intake of vitamin A, folate, vitamin C, zinc, iron and selenium was noted to be equal to or above the EAR for children in the two groups. The baseline dietary intake data was collected in the month of June, which is usually the end of the long rains, and therefore the green leafy vegetables were freely available from household subsistence farming in the area. The vegetables are normally eaten as accompaniment to the staple maize meal *ugali*. The varieties of indigenous vegetables consumed by the children in both the MVP and control groups were cowpea leaves (*Vigna sinensis*), corchorus species (known locally as *murenda*), *Crotaria* species (known locally as *mitoo*), pumpkin leaves *(cucurbits)* and *Amaranthus* species. African leafy vegetables (ALV) are rich sources of β-carotene, vitamin C, folic acid, riboflavin, iron and calcium.[316]

There is a possibility of overestimating the adequacy of micronutrient intake from our study using the DRIs which were developed for the North American population. The DRI for example assumes 18% bioavailability of iron and a diet containing haem sources of iron. The diet reported in our study was mainly plant based with a limited availability of haem sources of iron. Micronutrients from plant sources have poor bioavailability, especially β-carotene, folic acid and iron, as a result of phytates that hinder absorption. A plant-based diet with little animal-source foods and high amounts of starchy foods and legumes may inhibit the bioavailability of minerals, especially iron. Although the children had a high intake of vegetables and starchy foods, their intake of grains and legumes

was very limited, pointing to the possibility of less phytate activity, which would hinder the bioavailability of iron. The high intake of vitamin C was likely to have increased the absorption of iron from non-haem sources.

The probability of inadequate nutrient intake was high for folate, calcium, selenium and zinc in the study groups, with no significant differences between the intervention and control group. The limited intake of legumes, grains, nuts and seeds observed among the children resulted in a high probability of inadequate intake of folate, selenium and zinc, which are found in those foods if not obtained from meat sources. In addition, the absorption of zinc, iron and calcium is hindered by phytic acid, which is found in legumes, cereals, seeds and nuts.[317,318] This occurs through the formation of insoluble complexes that chelate with the minerals,[319] forming insoluble complexes that cannot be digested or absorbed due to the lack of an intestinal phytase enzyme.[317]

The habitual intake of a diet based on unrefined maize, which is the staple diet in the study area, increases phytate levels in the diet, thus increasing the probability of an inadequate intake of zinc and of calcium deficiency among the children. The possibility of estimating dietary zinc intake in our study was limited by the scantiness of data on the zinc content of local foods, which means that it is likely that zinc intake was under-estimated. Phytate data is not included in the Kenyan food composition tables that were used to compile nutrient intake, therefore an estimation of available zinc intake could not be done using the developed algorithms. Phytate values are also required in order to calculate available iron intake[320,321] and the bioavailability of calcium from habitual diets.[52,322] The high intake of vegetables reported among the school children in the current study was unlikely to interfere with zinc absorption, as studies have suggested that high amounts of dietary fibre do not inhibit zinc absorption, as speculated earlier.[323] An insufficient intake of zinc, vitamin A and calcium can result in impaired growth in children and in growth failure.[324] Folic acid deficiency and vitamin B_{12} deficiency cause megaloblastic anaemia[325]

The number of studies examining these vitamin deficiencies in school children in Kenya is very limited. No deficiency of folate was found in studies by Siekmann et al.[326] in Embu, Kenya. However, a high deficiency of vitamin B_{12} (67.9%) was reported in combination with a low intake of animal sources that contributed less than 1% of total energy intake.[326] Calcium is one of the main micronutrients of concern for growth in children,[82,327] as it plays a key role in the normal growth and development of the skeleton.[328] Adequate amounts of calcium are required to achieve optimal peak bone mass and maintain bone health. In our study population there was a low dietary intake of milk and milk products, which are the main sources of calcium. The reported frequency of the

intake of milk and milk products was limited to milk taken in tea and other beverages, as there is limited access to milk and milk products in this population. This has implications for bone mineral accretion in the children, given the benefits of an adequate calcium intake with regard to several disorders such as osteoporosis[329-331], hypertension,[332,333,333,334] colon cancer,[335,336] and kidney stones, in addition to implications for bone mass[337] and fractures.[338] The peak bone mass is accumulated during the first two decades of life, meaning that calcium intake in childhood and adolescence is critical for optimising peak bone mass.[8,339] Pre-teen children require a sufficient intake of calcium to support their accelerated growth spurt, as well as to lay the foundations for their peak bone mass, which will have an impact on bone density in later years.[340] Chan et al.[341] found that the rate of bone mineralisation increased in girls aged 11 years who took dairy products contributing to 1 200 mg of calcium daily for 12 months.

4.2.2   Prevalence of undernutrition

Chronic undernutrition, defined as height-for-age z scores < -2 standard deviation, affected one in every four children in the study population. Further, about half of the children had a linear growth deficit, measured as a height-for-age z-score < -1 standard deviation. The prevalence of chronic undernutrition and linear growth deficit was not significantly different between the groups. However, the level of chronic undernutrition was higher among males and older children (9 to 13 years). Six percent of the children were underweight, defined as a weight-for-age z-score < -2 sd, while just 3% had wasting defined as BMI-for-age z-scores < -2 sd. Slightly below one third (30%) of the children had inadequate nutritional status, defined as weight-for-age < 1 sd, while one in every four of the children had inadequate nutritional status, defined as a BMI-for-age z-score < -1 SD.

There were no differences between the groups in the level of undernutrition, based on weight-for-age and BMI-for-age at baseline. Slightly more children were overweight (BMI-for-age z-scores > 1 sd) in the control group compared to the MVP group, as were more males than females and younger children than older ones. The observed levels of undernutrition in our study point to the continuum of undernutrition carried forward from the persistently high levels of stunting and underweight among children younger than five in Kenya.[116,342] Although the greatest degree of stunting occurs during the first two years of life,[107] our findings are in agreement with those of other studies,[54,59,343] which suggest that growth stunting continues through the school-age years. Given that growth rate is much slower in middle childhood than in the earlier years, school-age children in resource-constrained environments may experience insufficient or delayed pre-pubertal catch-up growth.[344] The higher proportion of the older children with linear growth deficit and low BMI-for-

age compared to the younger ones may be linked to a growth cohort among the older children aged 9-13 years. Similar findings have been reported in Zanzibar[54] school children, where the prevalence of stunting was higher when children were 13 year of age compared to when they were 7 years of age. In interpreting the findings of our study, one has to consider that some of the known factors that influence linear growth failure in young children, such as birth weight[77,345,346] and maternal stature,[347] were not included in the data under consideration.

Chronic undernutrition is caused by the chronic poor-quality diet that dominates dietary intake in many low-income countries,[348] as well as infections and repeated morbidity that result in the depletion of body nutrient stores. The persistent undernutrition in Kenyan children is an indication of the consequences of persistent food insecurity in the country, resulting in low dietary intake or poor-quality diets, with an estimated 3.8 million people being in need of food aid.[7] Despite the fact that increasing agricultural production is one of the elements of the economic pillar for the achievement of Kenya's Vision 2030, the country is continually unable to produce enough maize to feed its population.[349] Overall food production of maize, which is the staple food, is still below what is required.[349] Furthermore, as observed in the results of this study, the probability of inadequate energy intake among the study subjects was as high as 70% among those in the MVP group and 62% among those in the control group.

The Sauri area, where the MVP is located, was classified as a hunger hotspot, with the production of maize limited by small land holdings, poor soils and parasitic weeds.[276] The Sauri Baseline Report further states that households suffer food shortages for three to seven months of the year.[277] In addition, 67% of the population was below the poverty line, which is defined as living on less than $1 per day.[288] The findings of the current study demonstrate this further, with a majority of the households earning a reported total household income of less than USD 12 per month. Although no data was available for the control group with regard to poverty levels and maize production, the control group was drawn from within 15 kilometres of the Sauri MVP, therefore the data with regard to maize production and household food shortage could be assumed to be similar to that reported for the Sauri area. In addition, the socio-demographic characteristics of the control group, as per our baseline findings, were similar to that of the intervention group, except for household headship, where the percentage of father-headed households was higher in the control group.
Wamani and collegues[350] reported a higher rate of undernutrition among boys than girls in a meta-analysis of 16 demographic and health surveys from sub-Saharan African countries. Other studies report similar findings in Africa.[351] In agreement with the Wamani[350] report, our study found a higher prevalence of undernutrition among boys than girls. However, other studies have reported no

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gender differences in anthropometric measurement outcomes in young children.[345,352] Neonatal epidemiological studies of pre-term infants predict morbidity and mortality to be consistently higher in males than females at an early age.[353]

The pattern of boys having poorer nutrition outcomes than girls is probably also related to cultural and behavioural factors, such as caregiving practices, which have been shown to influence children's nutritional outcomes.[354] No literature was found that explains any cultural beliefs, attitudes or practices that expose boys to undernutrition. It is common in rural African communities for girls to be involved more in kitchen and food-preparation chores,[355] thus they are more likely to spend more time than the boys with the mother or caregiver, thereby receiving more attention. On the other hand, male children may be spending more time out of the home, carrying out other family chores such as herding cattle, or playing with other male children, which would increase their energy expenditure.

Similar to our findings, other studies have reported age differences in the prevalence of stunting among school children in developing countries. In Mozambique,[356] Kenya,[50] Chad[59] and India, older children between the ages of 9 and 15 years have been found to have higher rates of undernutrition compared to younger ones.[19] These findings suggest a delayed pre-pubertal growth spurt, which was associated with food insecurity among adolescent girls in Ethiopia.[357]

The biological significance of mild to moderate undernutrition has been emphasised in studies among preschool-age children. Mild to moderate deficits in weight-for-age are associated with a higher risk of morbidity and mortality among young children in developing countries.[358-360] A similar association has been described among school children in Uganda, where mild to moderately undernourished children were more likely to have visceral leishmaniasis-malaria co-infection compared to their well-nourished counterparts.[361]

An association has been reported between maternal education and undernutrition among Kenyan pre-school-age children.[342] Children whose mothers had attained no education had higher odds of being underweight, stunted and wasted.[116,342] Maternal education is also associated with positive economic advantages, such as increased income, access to water and sanitation services, as well as access to health care,[362] as well as overweight and obesity for the mothers and children.[132] The positive effect of maternal education on income and employment may lead to increased access to food, which will improve the dietary intake of the children and result in better nutrition, as well as increasing the risk of overweight and obesity.

Our study found an association between lack of maternal employment and high odds of the children having linear growth failure, which is consistent with the findings of Srivastava et al.,[19] who reported maternal education and work status as important determinants of nutritional status of school-age slum children in India. The improvement of the nutritional status of school-age children with maternal education has been reported by other authors,[79,363] and is consistent with similar patterns observed in children under the age of five years in many developing countries.[364]

In line with the findings of other studies,[19,365,366] we did not find an association between maternal characteristics and wasting or underweight, as defined by BMI-for-age and weight-for-age < -1 SD. These findings can be explained by the fact that maternal characteristics may have limited effect on acute undernutrition, caused by illnesses such as diarrhoea or the short-term lack of food resulting from seasonal fluctuations in household food security.

4.2.3   Prevalence of overnutrition

The findings of this study document a noteworthy level of overweight among school children in the study area. These signs of overnutrition in the country were observed by Semproli and colleagues in the same region of Nyanza in 2003.[344] These findings are an important suggestion of the rising public health concern of overweight and obesity in the country. This rising trend of overnutrition has been reported among Kenyan children aged 36 to 59 months on the basis of Demographic and Health Survey (DHS) data.[132]

De Onis and colleagues reported an increasing trend of childhood overweight and obesity globally, with an estimated rate of 8.5% in Africa in 2010.[12] This phenomenon is increasingly common in communities in low-middle-income countries undergoing nutrition transition.[367] By providing estimates for overweight in rural Kenyan school children, our findings highlight the importance of developing preventative strategies for diseases associated with overweight and obesity in children and in the adult population. Both undernutrition and overnutrition are important public health issues of concern at the individual and national level, as they raise the human and financial costs through the increased demand for health care.[368]

Overweight and obesity among pre-adolescent and adolescent children present an increased risk for metabolic disease. Excessive body weight was found to be an important determinant of hypertension in school children from the Sychelles.[369] An association has also been reported between elevated blood pressure and BMI among school children aged 8 to 16 years in the Ashanti

region of Ghana.[370] Furthermore, urban South African children aged 6 to 13 years who had higher BMI were also found to have higher blood pressure.[371]

The association of low physical activity and higher economic status with overweight in school children has been reported in developed countries[372] and in the urban areas of low-income countries. Overweight in rural school children, who presumably have high levels of physical activity, requires further investigations to discover the determinants. Our findings that boys were more overweight than girls at baseline is contrary to the findings of a study by Arstrong et al[131] that reported a higher prevalence of overweight and obesity among girls than boys in South Africa. Another study among Danish[373] school children reported a higher prevalence of overweight and obesity among the boys than girls that was mainly resulting from a higher energy intake by the boys. Our study reported a higher prevalence of overweight among boys compared to girls at baseline. Boys are generally heavier than girls up to the age of eight years, following which the girls grow to be heavier due to a gain in body fat associated with pubertal changes.[29] In this growing population of school children, our study found a higher proportion of overweight among the boys and among the younger children.

4.2.4   Body composition

Few published anthropometric studies of Kenyan school children provide information on body composition. The Embu study by Neumann and colleagues[50] reported general leanness among school children, with triceps, subscapular, arm fat and arm muscle area below the 10[th] percentile of the NHANES reference data.[50] The findings of our study show a general leanness, with below the fifth percentile of NHANES for triceps and upper arm fat area. However, the children in the current study had a higher muscle mass than those in the Embu study, with a mid-upper arm muscle area within the 50[th] percentile of NHANES.

The findings of our study are in contrast with those from a study among school children in Bahrain, which reported a substantial subcutaneous fat deposition and low muscle mass.[145] Less lean body mass has been associated by Friedman and collegues[3745] with stunting in school children in a longitudinal study in rural Western Kenya. In their study, Friedman et al. found that progression in stunting could be explained by upper arm muscle area z-scores, rather than by lean body mass. In addition, deficits in fat-free mass rather than fat mass were reportedly related to growth failure in eight-year-old children with HIV.[375] The onset of puberty is marked by body composition changes such as increase in muscle mass and fat deposition.[376] Our study reported a higher fat and fat-free

mass concentration among the older children aged 9 to 13 years that was related to the effect of onset of puberty.

4.2.5   Haemoglobin concentration, prevalence of anaemia body iron stores and by serum ferritin

This study found that more than two thirds of the school children had anaemia at baseline, making it a severe public health problem based on the cut-off of ≥ 40%.[377] Therefore, the prevention of anaemia in this population was of paramount importance in order to avert the known consequences of anaemia, especially to achieve improved cognitive potential in these school children. This prevalence of anaemia was much higher in the MVP children than in the children in the control group. The mean serum ferritin concentration was also lower in the MVP children than in the controls. The prevalence of anaemia reported in our study was higher than what has been reported in this age group by other studies in the same province,[72] and among adolescent girls aged 12 to 18 years.[378] A higher prevalence of anaemia than found in our study has been reported in other sub-Saharan countries, such as among children aged 7 to 12 years in Nigeria (82.4%).[159,222]

The multiple causes of anaemia involve a complex interaction between nutrition, infectious diseases and genetic haemoglobin disorders.[379] The presence of inhibitors of the absorption of non-haem iron, such as phytates found in whole grains, legumes and cereals, further contributes to causing anaemia. In our study, the intake of dietary iron was reported at the same level as the EAR for age. It is important to bear in mind that this study used the DRIs developed for the North American population which assumes a diet with haem sources of iron and a high bioavailability of 18%.[380] This creates a possibility of overestimating adequacy of iron intake in this study. The results might possibly have been different if other reference standards had been applied. For example, the WHO/FAO/UN vitamin and minerals requirements in human nutrition report which provides a guideline of 5% and 10% bioavailability for iron in developing countries.[381]

The dietary intake of children in our study showed a considerably higher frequency of intake of haem iron sources (meat, fish and poultry), of 14%, compared to the frequency of intake of non-haem sources such as legumes (4%) and grains (0.5%). The dietary intake of vitamin C was noted to be adequate, resulting largely from an intake of guavas and mangoes, which were in season at the time of baseline data collection. The intake of sufficient amounts of rich vitamin C dietary sources by the children could have contributed to the much lower occurrence of depleted body iron stores, as indicated by serum ferritin levels, as vitamin C is known to enhance iron absorption.[382] The trivariate analysis showed that only 8% of the children had iron deficiency anaemia, which points to other factors in the study population contributing to their anaemia. Other micronutrient deficiencies,

such as of vitamin A, folate and B$_{12}$, may play a role in the aetiology of anaemia. We did not investigate the presence of megaloblastic anaemia, but did observe a probability of inadequate dietary folate intake among the children.

Three out of ten children had anaemia occurring with inadequate serum retinol concentration. Vitamin A deficiency and iron deficiency were identified as the most significant predictors for anaemia in a study involving 845 school children in Tanzania.[222] Furthermore, the occurrence of anaemia, iron deficiency and vitamin A deficiency is interrelated,[383-385] and threatens the health and nutrition outcomes of children in the school-age years. Haematological alterations have been reported in vitamin A-deficient children,[386] and increased intake of vitamin A through a single-dose supplementation was reported to improve the haemoglobin concentration in preschool children[387] and improve haematological status in pregnant women.[388] The prevalence of anaemia in our study was higher among younger children, in line with the results of other studies,[343] and may be explained by the fact that the younger children had experienced higher iron demand recently, during early childhood.

Infectious diseases contribute to anaemia by impairing the absorption and metabolism of iron and other haematopoietic nutrients, or by increasing nutrient losses.[389] The contribution of acute infection could not be ruled out in our study considering that the mean CRP concentration was slightly above the normal range, with an 11% prevalence of elevated CRP levels. Worm infestations, which are common in the tropics, have been associated with depleted serum ferritin concentration, thus causing anaemia.[233] Blood loss resulting from heavy worm infestation leads to loss of nutrients and anaemia. The baseline data in this study was collected shortly before a deworming programme that was organised and carried out by the MVP in the study area. Deworming was carried out 3 months after baseline data collection in the control area. The contribution of helminthic infestation to the high prevalence of anaemia observed in among the children can therefore not be ruled out in this population.

Although our study did not investigate the presence of *Plasmodium falciparum* paracitaemia, malaria is an important cause of anaemia in sub-Saharan Africa[390] and in our study area, which is a known endemic malaria region. One of the documented mechanisms of the pathogenesis of malarial anaemia is that malaria causes anaemia through the increased destruction of the red blood cells and decreased red blood cell production.[391] In our study, the mean red blood cell count was within normal ranges, and therefore this mechanism may have been less of a cause of the high prevalence

of anaemia observed. Nutritional status as indicated by weight-for-age, height-for-age and BMI-for-age z-scores was a significant predictor of anaemia in our study.

Children with mild to moderate undernutrition were more likely to be anaemic at baseline. Stunting has been associated with anaemia in a study among school children by Stoltzfus et al.[233] in Zanzibar, Tanzania, and in pre-school-age children in the East African region.[392-395] The specific aetiology of anaemia and growth failure is not clear. One hypothesis that has been suggested is the malaria-anaemia pathway, which suggests that nutritional inadequacies that cause stunting also impair host immunity, resulting in an increasing degree to which malaria decreases haemoglobin concentration, thus increasing the demand for iron required for erythroblasts.[389] Furthermore, anaemia, stunting and malaria infection have been identified as risk factors for poor motor development outcomes in young Zanzibari children.[395]

Balarajan et al.[389] described a complex interplay of political, ecological, social and biological factors as important determinants of anaemia in middle- and low-income countries. The socioeconomic factors described by Balarajan et al.[389] are household wealth, which affects the ability of household members to access diverse and fortified food sources, iron supplementation and deworming, adequate knowledge and education about anaemia, clean water, sanitation and insecticide-treated bed nets. The household socioeconomic factors listed above influence an individual's nutrient intake and absorption, as well as exposure and response to infections.

Female-headed households are assumed to be poorer than male-headed households and thus less able to invest in the education and health of children.[396] Furthermore, female-headed households have been reported to own less than half of the farming equipment owned by male-headed households,[397] and female entrepreneurs in Kenya have less access to supplier credit than their male counterparts.[398] Also, poor, vulnerable female-headed households may lack adequate access to health care,[399,400] thus delaying the treatment of diseases and the seeking of preventive primary health-care services. In this study, an association emerged between household headship and the occurrence of anaemia in children. Those from households headed by mothers or grandparents had a higher likelihood of being anaemic.

Female and grandparent household headship in this population could be an indicator of the death or absence of the male household head, which has a strong impact on the nutrition outcomes of the children. Studies of young children in India identified family wealth and food insecurity as determinants of anaemia.[401] The findings of our study show a lower prevalence of depleted body

iron stores than that of 19.8% reported by Leenstra et al.[378] in adolescent girls aged 12 to 18 years in the same province, and that found in an earlier study by Olsen et al.[402] conducted among school children in a neighbouring province in Western Kenya.

The lower prevalence of depleted body iron stores in the current study may be as a result of the apparently adequate mean iron intake reported by the children in the two study groups. Low serum ferritin levels were associated with sex of the child at baseline, although this relationship was not sustained at the multivariate level of analysis, which controlled for household and maternal socio-demographic characteristics, as well as anthropometric indicators and the child's age. Longstanding consumption of diets with low iron bioavailability has been reported to cause low body iron stores and iron deficiency among school children in Morocco.[221] A high consumption of plant-based foods, such as grains, legumes and vegetables, with few animal sources of food, results in the low bioavailability of iron.[219,221] The diets of children in the MVP group and in the control group were noted to consist predominantly of staple starches, such as green bananas, maize and rice, which contain little non-haem iron that is not readily absorbed.[219] However, a high intake of vitamin C was also observed, and this is known to enhance iron absorption.[382]

4.2.6   Vitamin A status and serum retinol concentration

The mean serum retinol concentration at baseline in this study was similar to that reported in the Embu studies,[259] while the inadequate vitamin A status (< 0.70 µmol/L) in our study was similar to that reported in South Africa[403] among children 6 to 11 years old (39.1%), but lower than that reported in the Embu study (68.6%),[259] in an Ethiopian study among children aged 6 to 9 years old (51%),[404] and in a study in Burkina Faso among children aged 6 to 11 years old (40%).[405] However, our results show a higher prevalence of inadequate vitamin A status than that reported in Tanzania (20%),[197] Bangladesh (20%)[406] and Kazakhstan (20%).[407] Given the absence of an established classification of public health significance of vitamin A status in the school-age population, the WHO classification[189] of public health significance in younger children (aged 6 to 71 months) was used. Based on this classification, low vitamin A status (< 0.70 µmol/L) was a severe public health problem among the children in the MVP and the control areas at baseline (> 20% with serum retinol (< 0.70 µmol/L). These findings indicate a persistent sub-clinical vitamin A deficiency of public health importance among school children across Kenya. Serum retinol reflects depleted liver stores only when they are severely depleted (< 0.35µmol/L) or extremely high (1.047 µmol/L).[289]

Vitamin A deficiency is known to occur mainly as a result of the insufficient intake of vitamin A, which is needed for growth and development and the body's physiological functions.[198] The low reported intake of animal sources of food in this population points to a lack of nutrient-dense diets, which can contribute to meeting the high nutritional requirements of the pre-adolescent children studied. Previous studies in a rural setting in the Eastern Province of Kenya reported a usual intake of predominantly plant-based diets among school children.[49,150] Low intake of dietary animal-source diets has also been reported among school children in Uganda.[408] The intake of animal-source foods has been shown to improve dietary quality, micronutrient status, growth and cognitive function in Kenyan school children.[50] The bioavailability of β-carotene, found in plant sources of vitamin A – especially vegetables, is as low as 3 to 6% for green leafy vegetables and 19 to 34% for carrots. The main source of vitamin A in the study population was green leafy vegetables, which may explain why there was a considerable level of inadequate vitamin A status although the mean vitamin A dietary intake was above the EAR.

The consumption of green leafy vegetables is common in the rural villages of western Kenya, as they form the staple meal eaten with *ugali* (maize meal), which implies that green leafy vegetables are the main source of vitamin A for the population, as also observed in our study, which showed that the intake of vegetables was second to that of staple starches. In our study, the frequency of consumption, in the previous six months, of vegetables such as kale, pumpkin leaves and indigenous green vegetables, as well as of carrots, was 19%, while the consumption of fruit such as mango, ripe pawpaw and citrus fruits was 15%, compared to the consumption of meat sources at 14%. Serum retinol concentration is also affected by the synthesis of retinol-binding protein in the liver, infections, nutritional status and the existing level of other nutrients such as zinc and iron.[409]

The levels of subclinical vitamin A deficiency observed in our study can be explained further by the low fat intake, as insufficient intake of dietary fat (under about 5 to 10 g daily) obstructs the proper absorption of retinol and carotenoids, increasing the risk of vitamin A deficiency.[410] Intestinal absorption of vitamin A and carotenoids is facilitated by dietary fat ingested with vitamin A-rich sources.[411,412] The digestion of retinyl esters and the conversion of provitamin A in the intestines is stimulated by the presence of dietary fat in the intestines.[413] This is accomplished through the stimulation of pancreatic enzyme secretion of bile salts, which serve to form mixed micelles of lipids, and provide products of lipid digestion mechanisms involved in intestinal metabolism of provitamin A.[414] In addition, fat ingestion provides the lipid components for intestinal chylomicron assembly, which in turn promotes the absorption of carotenoids and vitamin A.[414]

Elevated CRP is associated with low serum retinol, indicating a decline in serum retinol during the acute-phase response to infections.[415] Serum retinol concentrations have been shown to decrease during malarial infection.[416] This reduction can be explained as the direct consequence of inflammation caused by the *Plasmodium falciparum* infection.[415,417] This is a factor to consider when interpreting the presence of sub-clinical vitamin A deficiency among children in the study, which was conducted in a malaria endemic area. A decline in serum retinol levels results from an increased release of interleukin-6, which subsequently causes a reduction in the serum levels of retinol-binding proteins as result of the inflammatory response of *Plasmodium falciparum*, thus impeding the transport of vitamin A from the liver.[416]

The presence of sub-clinical vitamin A deficiency in this population at baseline warranted appropriate food-based approaches to increasing carotenoid intake through the diversified school feeding programme. This is so especially because vitamin A deficiency is known to increase susceptibility to malaria through impaired antibody response to malarial antigens requiring T-helper type 2 cell mediators.[418] SanJoaquin and Molyneux[418] argue that providing vitamin A to populations at risk of malaria is beneficial in reducing all-cause mortality in malaria endemic areas. A study among children aged 6 to 60 months in Papua New Guinea, involving supplementation with high-dose vitamin A every three months for 13 months, reported a reduction in the number of malaria episodes in the supplemented group.[419]

Our study found sub-clinical vitamin A deficiency to be significantly higher among the control children and the females. The high prevalence of inadequate vitamin A status based on low serum retinol concentration in the control group may have been confounded by the higher level of elevated CRP levels observed, which was more than that in the MVP group. The difference observed in vitamin A stores among the girls can be explained by growth indicators (weight-for-age, height-for-age and BMI-for-age), which diminished the differences in vitamin A status seen at the bivariate level of analysis between girls and boys when included in the logistic regression model.

The relationship observed between inadequate vitamin A status and the employment status of the household head, as well as household income, was an interesting finding, pointing to the contribution of household socioeconomic to the vitamin A status of household members. This relationship was maintained even after controlling for baseline nutritional status, age, sex and maternal education. A similar relationship was reported in a study of pre-school-age children in Zambia.[420] These findings are related to the fact that a household with a higher socio-demographic status would be in a better position to afford animal-source foods, which have higher contents of

vitamin A. The negative finding, that children with a higher BMI-for-age z-score at baseline were more likely to develop low vitamin A status, should be interpreted with caution, as the number of children with a low BMI-for-age was only 10.

4.3   Impact of the School Feeding Programme on Nutritional Status and Body Composition

Food-based approaches are known to offer more protection and sustainability than single or multiple micronutrient supplementations for school children.[7] In our study, children in the intervention group were provided with school lunch on school days and followed up for a period of 30 months. The school feeding programme in the MVP group was made up of *githeri* (maize and beans mixture) for three days of the week, beef with *ugali* (made from maize meal) and cooked kale on two days of the week, or *ugali* and whole fish *omena* with cooked cabbage on two days of the week during the second school term, when fishing of *omena* was allowed in Lake Victoria. Children in the control group were provided with *githeri* (maize and bean mixture) from six months throughout the three school terms. The *githeri* meal provided 387 kcals (1 619 kJ) on average in both in the intervention and control groups, while the *ugali omena* and *ugali* bean meals provided 467 kcal (1 954 kJ) on average for the intervention children. The *ugali omena* meal provided 10.2 g of fat, the meat *ugali* meal provided 9.1 g of fat, while the *githeri* meal provided 4.6 g of fat. Given the low dietary fat intake of these children at baseline (< 25% of energy), the additional fat probably provided a necessary dietary change and increased energy density and satiety.

In the MVP group, the intervention was associated with greater height gain and height velocity and decreased odds of having linear growth retardation from the 18th to the 30th month of follow-up. This association was maintained after controlling for socio-demographic characteristics. Children gained height and weight as expected, but the gains were greater among the MVP children consuming a diversified diet with meat-source foods, especially by the 18th and 24th months, when the MVP children had a significantly higher growth velocity. Studies among school children have reported mixed results for growth and body composition outcomes between different interventions and controls from both lower- and higher-income countries.[133,263]

Some studies have reported positive effects of school feeding on children's growth and anthropometric indices, while others have found no effect. Kristjansson and colleagues[133] conducted a systematic review and meta-analysis involving a total of 18 studies, including studies from Kenya, Jamaica, India, Indonesia and China, with both randomised controlled trials (RCT) and controlled before and after (CBA). The meta-analysis showed an overall small, non-significant change in height between children who did or did not have school meals at school in the RCTs,[50,268,421]

whereas a significant increase in height was observed in the CBA trials.[133] This review by Kristjansson et al.,[133] suggested that methodological issues may have caused the differences observed in the outcomes. In the same review, children participating in school feeding programmes were found to gain on average 1.43 cm in height more than the controls over one year.

There was a small yet significant effect of school feeding on height-for-age z-scores following meta-analysis of two RCTs.[133] Kristjansson and collegues[133] found a stronger and more consistent effect of school feeding on weight gain in the randomised controlled trials and the CBA; the weight gain ranged from 0.25 to 0.75 kg a year. Furthermore, Simeon[422] found no significant weight gain in 7 year-old school children in Jamaica provided with 100 ml of milk and a slice of cake or meat-filled pastry for one semester. Mixed results from the impact of school feeding programmes on growth may be due to the settings, methodological differences and objectives of the interventions reported in various studies. The main differences have to do with the programme design, the quality and quantity of food given to the children, the length of any particular intervention and the modalities of school feeding.[262] Despite the mixed results on the impact of school feeding programmes on growth, the significant gain in height velocity we observed in our study in the intervention group emphasis the need to provide children with high-quality protein that also provides multiple micronutrients and can increase the bioavailability of iron and zinc.

It has been debated whether stunted children can achieve catch-up growth after the age of three years, Although findings from lower-income countries have reported no significant increases in height gain, as reviewed by Kristjansson et al.,[133] our findings are similar to those of Powell et al.,[268] who found that children provided with a breakfast snack gained more height and BMI than the controls. Powell et al. found that the gains in growth parameters among the children in the breakfast intervention group were more in the younger ones, who were in grades 1 and 2. The main differences in anthropometric indicators reported in our study were more noticeable in the 18th, 24th and 30th months of the interventions. By this time the children were aged between 10 and 11 years and in class 3 and 4, suggesting the occurrence of a pre-adolescent growth spurt. Our findings also show an improvement in height-for-age z-score in both groups of children, an increase in fat-free mass among the children in the diversified diet group (MVP), and significant gains in percentage body fat in the control group. These findings indicate that the additional energy from the diversified school feeding programme was not used to increase body fat in children in the MVP.

Our findings on positive changes in fat-free mass for children receiving meat and fish as part of the school lunch concurs with the findings in studies by Neumann and colleagues in the Embu District

of Kenya, which reported a gain in mid-upper arm muscle area and mid-upper arm circumference measuarements.[66,264] The effects of a school feeding programme on school children are often affected by meal replacement, as children receiving lunch from school may reduce their dietary intake at home – as suggested in a Ghanaian study that evaluated a school breakfast programme.[423] On the contrary, other researchers have reported no evidence of substitution of school lunch with home dietary intake.[256,268,424,425] Although evidence from the literature is non-conclusive on whether school meals partially substitute home caloric intake, the energy intake from the school lunch in the Millennium Villages Programme contributed to about 25% of the estimated energy requirement of the children and therefore it is unlikely to substitute home lunch. Although this aspect was not investigated in this study, there was an increasing trend of overweight between baseline and the subsequent intervals of data collection especially among the control children. While the reasons for this trend are not clear, it is noteworthy that this trend is similar to that reported among school children in Chile,[127] and Pakistan.[118]

4.4   Impact of the School Feeding Programme on Anaemia and Body Iron Stores

Low haemoglobin is the accepted indicator for anaemia and it is the basis on which appropriate interventions and programmes for reducing anaemia in populations are evaluated.[72] Our study reported a higher prevalence of anaemia among the children in the MVP group than in the controls at baseline, but changed to a significantly lower anaemia prevalence in the MVP group at six months. Several studies[50,257,262] have observed increased biochemical parameters following food-based interventions among school children. In Bogota, Columbia, a breakfast snack providing 30% of energy, 50% of iron and 40% of calcium needs resulted in increases in serum vitamin B_{12} concentration and in haemoglobin and plasma ferritin concentration after three months.[257] The findings of Arsenault et al. in Bogota, Colombia[262] are similar to those of Neumann et al.[50] in the Embu study in Kenya, who found a significant increase in B_{12} concentration upon food supplementation with animal-sources foods. However, the study in Embu did not find any significant gains in body iron stores.

In our study, the mean Hb concentration was not significantly different at the 24^{th} month interval, showing that Hb concentration increased in the control group from 6 to 24 months to an amount almost equal to the mean Hb concentration in the MVP group. The *githeri* meal provided to the control group from six months provided 6.5 mg of iron on average, and this was mainly non-haem iron. Considering the bioavailability of non-haem iron sources, it is unlikely to have been the only cause of the increased Hb concentration in this group between six and 24 months. This increase may have been caused by the general health-related MV intervention introduced in the control

group from month six, similar to the interventions in the MVP area, such as the construction and equipping of health facilities for the treatment of malaria and deworming, and the improved water and sanitation facilities.

There are a number of other factors that may have contributed to decreased anaemia in the MVP group beyond the benefit accrued from dietary intake. The MV Project involved free testing and treatment for malaria at the Sauri health facility. The Project also provided medical supplies and personnel for the Sauri government health dispensary, which resulted in prompt treatment of infections. In addition, food production by the MVP households increased from 1.9 to 5.0 tons per hectare during the first months of the intervention, following the provision of fertiliser and seeds to the farmers.[276] Other broad-based interventions in the MVP included improved access to water and sanitation facilities as well as improved roads network. These broad based interventions were also done in the control area after the first 6 months of the study following the scale-up of the MVP activities to the control areas.

The school feeding programme in the MVP provided 7.5 mg of iron on average, which is about 75% of the RDA and above the EAR for the ages of 6 to 13 years. The diversified diet offered to the MVP children provided an increased intake of other micronutrients, such as vitamin B_{12}, in addition to the benefit of haem iron provision through the meat provided twice a week to children in the MVP group. The control group did not receive any school meals during this six-month period and therefore their dietary iron intake remained as what was provided at home.

Although dietary diversification is the most sustainable approach[325] to correcting iron deficiency in populations, the inclusion of beef on one day of the week and *omena* fish on two days of the week did not demonstrate an improvement in body iron stores in this population. Other researchers have found similar outcomes, where supplementation or food-based approaches have not increased ferritin concentration in school-aged children. Serum ferritin concentration decreased in a group of school children that were supplemented with multiple micronutrients in a breakfast health drink for fourteen months.[426] Although the effect of malaria was not studied in the current study, malaria infection has been reported to decrease the concentration of serum iron, plasma vitamin B_{12} and retinol, independent of the effects of CRP.[326]

The effect of malaria infection in this population may have affected body iron stores, as this is a malaria endemic region and data was collected during the rainy seasons, when malaria transmission is likely to occur. However, the extent to which malaria and other infections contributed to a lack of

improved iron stores was limited, considering the provision of free medical care at the health facility in the MVP. A decrease in serum ferritin levels can be an adaptive response, reflecting increased utilisation of iron for tissue growth, and this process may be particularly important during adolescence.

It is important to note that the blood sampling for serum ferritin analysis in this study was conducted during the second week after school opening in January 2007 and May 2008, following two months of school holiday in December and three weeks of holiday in April respectively. No school feeding was provided during the holiday and therefore the serum ferritin during the sixth-month measurement interval could be reflective of the depletion of nutrition stores during the school holidays, rather than a lack of improvement resulting from the school feeding programmes. This is possible, considering that the half-life of serum ferritin is 27 to 30 hours.[427] The decline in serum ferritin concentration by the sixth month was higher in the control group compared to the MVP group. Thus the effect of the long school holiday was felt more in the control group. By the 24[th] month there was a restoration in serum ferritin concentration among the children in the two groups to a level almost equal to the baseline amounts. The 24[th] month interval was in May 2008, which followed a shorter school holiday of three weeks and also a season with more household food availability, coming towards the end of the long rains.

4.5   Impact of the school feeding programme impact on vitamin A status

There was a decline in serum retinol concentration during the 6[th] month study interval in the MVP group, while the serum retinol concentration increased slightly among the controls during the same study interval. The mean serum retinol concentration increased for both groups of children by the 24[th] month interval. During the 6[th] month interval, 7.8% of the children in the MVP group had vitamin A deficiency (serum retinol $< 0.35\mu mol/L$), compared to 4.2% of the children in the control group. In addition, the prevalence of inadequate vitamin A status (serum retinol between 0.35 and 0.70 $\mu mol/L$) was slightly higher among the control group than the MVP group, while the prevalence of inadequate vitamin A status was slightly higher by the 24[th] month among the MVP group compared to the controls.

The decline in vitamin A status among the school children may be attributable to the lack of adequate intake of vitamin A-rich foods during the school holidays, as well as the low intake of dietary fat that is required for the absorption of dietary vitamin A. Blood sampling for serum retinol analysis was done within two weeks of school opening, at a time that may not have been enough to replenish serum levels of vitamin A following the school holidays, considering the vitamin A half-

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life of 13.5 hours.[428] During our study, the 2007 December holidays coincided with a period of lower rainfall than expected as a result of *la-nina* conditions, as reported by the Kenya Meteorological Department.[429] The short rains in the western part of the country were below normal ranges (< 75% of the usual amount of rainfall expected during the short rain period), resulting in low food production. The short rainy season mainly enables the growing of vegetables and other short-season foods, such as beans that are harvested within three months. Failure in the rains therefore resulted in poor vegetable and fruit output on the farms, with a possible reduction in consumption. Since dark green leafy vegetables are season dependent,[430] a depressed rainfall period of three months would result in a lower intake of vitamin A.

African leafy vegetables (ALV), which are commonly grown and consumed in the western part of the country, contribute up to one third of total vitamin A consumed in rural households in Western Kenya[431] were also not in supply following the failure in the short rains. Besides the inclusion of kale in the diversified school meal in the MVP, no other food item was targeted at increasing vitamin A intake in the children. Vitamin A deficiency among children aged 3 to 11 years is considered very likely to improve with an increased consumption of vitamin A.[289] There was an increase in serum retinol concentration and improved vitamin A status by the 24[th] month of study in both groups of children. A number of other extraneous factors besides dietary intake may affect serum retinol levels. Liver disease decreases serum or plasma levels as a result of decreased synthesis and secretion of RBP, stress, zinc deficiency through its role in the synthesis of RBP, as well as low-fat diets.[289] Low dietary zinc intake and marginal zinc deficiency are said to cause poor absorption of β-carotene, mainly because zinc is required for the synthesis of chylomicrons.[432]

In addition to zinc, dietary iron intake may be of importance in the bioconversion of pro-vitamin A carotenoids, because the monooxygenase enzyme that converts pro-vitamin A carotenoids to vitamin A in the intestinal mucosal cells is iron dependent.[433] Although the interactions of the three micronutrients is not clearly defined, it is of interest to note given that zinc, iron and vitamin A deficiencies are seen to co-exist in populations.[434] The conversion of β-carotene to retinol is estimated at 16.7% from a mixed diet, and approximately 50% of the absorbed β-carotene is converted to retinol in human intestinal cells.[380] The absorption of plant sources of pro-vitamin carotenoids is also affected by the chemical structure, plant matrix, amount of dietary fat in a meal, amount of pro-vitamin carotenoid in the meal, interactions with other carotenoids, and micronutrients such as zinc in the diet.[435]

The bioavailability of carotenoids from plant-source foods is generally low because carotenoids are not easily released from plants during digestion. This low bioavailability of vitamin A increases the risk of vitamin A deficiency in populations depending on pro-vitamin dark green leafy vegetables as their main source of vitamin A. The interaction among carotenoids within the intestinal lumen and mucosa may affect the absorption and bioconversion of pro-vitamin A in the carotenoid form.[435] The incorporation of carotenoids into the intestinal micelles and chylomicrons may be affected by competition between different carotenoids, thus impairing absorption.[435] Studies that included a rich vitamin A source such as fleshy yellow sweet potatoes have reported substantial increases in total vitamin A intake from β-carotene in children aged one to five years and in pregnant women.[322,436]

Retinol concentrations are also decreased by acute and underlying chronic infections.[437] Given the seasonality of disease patterns, shifts in serum retinol distribution may occur that are unrelated to the availability of vitamin A-rich foods. Disease status that affect the secretion of gastric and pancreatic enzymes as well as bile salts affects intestinal permeability and decreases the absorption of pro-vitamin A carotenoids.[435] Malaria infection has been associated with severe retinol deficiency (< 0.35 µmol/L) in children younger than six months in Western Kenya.[438] Although infection with malaria was not investigated in this study, children who were found to have severe anaemia were referred to the health facility for treatment.

It has been argued that vitamin A deficiency enhances susceptibility to malaria. One of the proposed mechanisms for this interaction is that the antibody response to malarial antigens that require Th2-mediated response is impaired during vitamin A deficiency.[439] This argument is further connected to the role of vitamin A as a regulator for gene activities through its active metabolites such as 9-cis retinoic acid.[440] The 9-cis retinoic acid metabolite is known to increase phagocytosis of *Plasmodium falciparum*-parasitised erythrocytes, thus resulting in increased severity of malaria.[441] On the other hand, malaria infection contributes to vitamin A deficiency by decreasing food intake, malabsorption, direct urinary retinol losses during severe infection, and impaired plasma-carrying capacity resulting from the acute phase response that produces fever.[418]

4.6   The Effect of the Initial Nutritional Status on the Responses to the School Feeding
        Programmes on Growth and Body Composition

The initial nutritional status was based on growth indicators, namely height-for-age, weight-for-age
and BMI-for-age z-scores. When all the children were combined, those with an initial inadequate
nutritional status based on height-for-age z-score < -1 SD had a significantly lower height and
weight compared to those with an initial adequate height-for-age z-score in all six study intervals. In
addition, those with inadequate initial nutrition based on height-for-age had a significantly
increased height velocity by the 24th month compared to those with an adequate nutritional status
based on a height-for-age z-score < -1 SD. The children gained more fat mass and fat-free mass if
their initial height-for-age z-score was adequate at baseline. When considered in separate study
groups, children in the MVP group with an initial height-for-age z-score > -1 SD gained
significantly more height throughout the study. Of interest to note is that those with an initial
inadequate nutritional status (height-for-age z-score < -1 SD) in the MVP had a higher height
velocity compared to those with an initial height-for-age z-score > -1 SD by the 24th month.

In the same MVP group, children with better nourishment (height-for-age z-score > -1 SD) had a
significantly higher mean weight by the 24th and 30th months and a higher weight velocity by the
24th month. In the control group the only indicator that was significantly affected by the initial
nutritional status based on height-for-age > -1 SD was height, which remained significantly greater
among the well-nourished children throughout the six study intervals. The observation of better
height and weight outcomes amongst children who had initial nutritional status could be an
indication of catch-up growth though. This indicator of catch-up growth among the children has
been demonstrated by studies among school children in developing countries.[107] Predictors of
recovery from growth faltering have been identified as maternal education, which influences child
feeding and care practices,[111] while a higher initial height-for-age z-score assessment increases the
likelihood of catch-up growth.[109]

Inadequate initial nutritional status based on a weight-for-age z-score < -1 SD had an effect on the
mean height, MUAC and mid-upper arm muscle area for all the children combined. Those with an
initial inadequate nutritional status on the basis of a weight-for-age z-score < -1 SD had a
significantly higher mean height velocity in the 18th, 24th and 30th month of study. This
improvement in linear growth among children who were earlier under-nourished is an indication
that the nutrients provided by the school meals contributed to catch-up in height among the
children. To a certain extent, children with initial mild and moderate underweight benefited more

from the school feeding programme with regard to height gain, fat-free muscle and fat mass. In the MVP group, children with initial mild to moderate underweight had greater height velocity through the study intervals. On the other hand, children in the MVP group with an initial adequate weight-for-age z-score > -1 SD gained significantly more fat-free mass by the 6th, 12th and 18th months. Specifically, those with a weight-for-age z-score > -1 SD in the MVP group had bigger mid-upper arm muscle circumference by the 6th and 18th month and bigger mid-upper arm muscle area by the 18th month.

A comparison of the two study groups with regard to initial nutritional status based on BMI-for-age was not done, as there were only 10 cases with a BMI-for-age < -1 SD and there was a lack of statistical power to run repeated ANOVA models per study group. Overall there was a steady increase in MUAC among children with an inadequate initial BMI-for-age of < -1 SD, from a significant different mean at baseline to almost an equal mean to those with a BMI-for-age > -1 SD by the 30th month of study.

The height velocity among children who had chronic undernutrition at the beginning of the school feeding programme in the MVP group is suggestive of an additional benefit of the diversified diet on growth. Those in the MVP group with an initial height-for-age < -1 SD had increasing height velocity from 0.6cm to 2.8cm/6months between the 6th and the 30th month. This was a higher rate than that of those in the MVP group with a height-for-age > -1 SD, whose gain in height velocity ranged from 0.5cm at 6 months to 2.5cm/month at 30 months. The study also found a higher height velocity among those in the MVP group with an initial inadequate nutritional status based on a weight-for-age < -1 SD, from 0.7 cm at 6 months to 3.0 cm/6 months by the 30th month, compared to an increase from 0.5 cm at 6 months to 2.5 cm/6 months at 30 months for those in the MVP group with a weight-for-age > -1 SD z-score.

Although the children with linear growth failure in the control group did not show significant gains in height or weight velocity during the period of observation, no further decline in growth indicators was observed in this group. This implies a protective effect of the meals provided in the control group, given that previous studies among school children in Kenya and in other developing countries who remained in the same environment showed that the children continued to accrue growth deficits in height compared to international reference standards.[107,374] While the evidence is inconclusive on the ability of catch-up growth for stunted children after the age of three years, a study of Filipino children by Adair[2] exhibited catch-up growth following an improved environment

during the school years. Furthermore, adoption studies also suggest a potential for catch-up growth during school years.[442]

Our findings are in line with those of Powell et al.,[268] who reported improved growth in stunted rural Jamaican school children following a breakfast snack. Similarly, Stoltzfus and colleagues reported an improved growth in late school age following a three-yearly deworming exercise in Zanzibar.[452]

Fat-free mass remained significantly lower throughout our study among children who had a height-for-age $\leq$ -1 SD at baseline compared to those who had a height-for-age > -1 SD. Fat-free mass, rather than fat mass, has been related to stature in studies among school-age children. Friedman et al[374] associated the upper arm muscle area z-score with progression of stunting through the school-age period among school children in Kenya. A relationship between deficiencies in fat-free mass rather than fat mass and growth failure was demonstrated by Arpadi et al. among HIV-infected children.[375] These findings, as well as ours, point to the importance of specific body composition, particularly lean body mass, in relation to linear growth.

4.7   The Effect of the Initial Nutritional Status on the Responses to the School Feeding Programmes on Haemoglobin Concentration, Prevalence of Anaemia and Body Iron Stores

Children with mild to moderate stunting at baseline had a bigger increase in mean haemoglobin concentration after 6 and 24 months of the school feeding programme in the MVP group. This effect did not occur in the control group at six months, but did occur by the 24[th] month. This can be explained by the fact that school feeding was introduced in the control group in the 6[th] month, which indicates that the effect occurred upon introduction of the feeding programme. That this effect of mild to moderate stunting on haemoglobin concentration was seen in both groups of children at different intervals indicates that the effect of initial linear growth failure on haemoglobin concentration was not limited to the children in the MVP group who were receiving a diversified diet.

Although the increase in Hb concentration was higher in the children with an initial linear growth deficit in the MVP group, a logistic regression model fitting in the socio-demographic and maternal characteristics showed that, in the combined group, children with an initial growth deficit had greater odds of being anaemic by the six-month study interval, but not at 24 months. However, this same regression model showed that the children in the MVP group had lower odds of anaemia at 6

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months compared to the controls. Thus the effect of initial linear growth deficit may have influenced the haemoglobin concentration and anaemia prevalence in the control group to a greater extent than in the MVP group.

Interventions for the treatment or prevention of anaemia in a school-age population should be well targeted to meet the specific needs of stunted children, since anaemia and undernutrition often share common causes, with growth placing demands on iron levels, which means that the two health outcomes are intrinsically related.[343] Anaemia coinciding with stunting in the same person results in an impairment of key body functions, such as growth, immune response and psychomotor development. Branca and Ferrari[443] suggest that when these symptoms occur in an individual person, this is an exhibition of 'stunting syndrome'. Our results support the suggestions by Branca and Ferrari that providing dietary diversification in the family and ensuring micronutrient density are important in preventing stunting syndrome throughout childhood and adolescence.[443]

Contrary to the effect of initial linear growth deficit, the effect of initial BMI-for-age z-score and weight-for-age z-score followed a different pattern. The effect of initial BMI-for-age z-score on the changes in haemoglobin concentration was observed among children in the control group between baseline and six months. In this group, haemoglobin concentration increased more among those with an initial low BMI-for-age z-score. These findings were unexpected, given that children in the control group did not receive school feeding from baseline to the six-month interval.

The change in haemoglobin concentration may have been related to age or socio-demographics, such as household headship, which was significantly different between the groups at baseline. When these other characteristics were included in a regression model, the observed differences diminished, although the regression model fitted haemoglobin and anaemia prevalence in the combined group and not in the study groups separately. The observed pattern of the effect of stunting compared to that of wasting and underweight on haemoglobin concentration following the school feeding programmes is an indication that interventions that target chronic growth deficit in this population are also beneficial in addressing anaemia and vice versa. Our study did not show significant effects of initial nutritional status on changes in serum ferritin concentration during the follow-up periods.

4.8   The Effect of the Initial Nutritional Status on the Responses to the School Feeding Programmes on vitamin A status.

Initial mild to moderate underweight increased the odds of inadequate vitamin A status in the 6th and 18[th] months of the study. Underweight children were at a higher risk of subclinical vitamin A deficiency at all the three measurement intervals in the combined data analysis, indicating that the school feeding interventions did not provide the extra vitamin A needs of underweight children. However, these results are to be interpreted with caution, as there were fewer than 20 children with mild to moderate underweight in this analysis.

4.9   Reflection on the Research

The longitudinal nature of this study, allowing for follow-up of children over a long period, provides an opportunity to explore the causal relationships and effects of the school feeding intervention.

4.9.1   Strengths of the study

A locally based and community supported school feeding programmeis a sustainable method of meeting the nutritional challenges among school children. This study demonstrates the success of community engagement and partnership in the management of a school feeding programmethat has shown positive outcomes on growth, health and micronutrient status of the children. The use of locally available foods to increase dietary variety and quality in partnership with local communities is important for community ownership, partnerships and sustainability. The use of local foods for school feeding programmehas the potential to increase local production considering the availability of a ready market. Although the analysing of the cost effectiveness of the school feeding intervention was not part of the objectives of this study, the use of locally available foods is less costly considering the proximity to local markets thus cutting down on the cost of transportation.

As described by Greenhalgh et al.,[266] our study was implemented in a well- schools' structure with support from the schools' administration, teachers and parents as well as cooperation from the pupils. This was made possible by the involvement of the entire school community at the start of the programmethrough the menu identification process and the strong partnership in implementation on the programme. The school feeding subcommittee drawn from the members of the community, parents, teachers and a representative of the MVP implementation office had weekly meetings to plan the critical details of food distribution chain and discuss any challenges that may have arisen and find solutions to those challenges. The Principle investigator was in consultation with this subcommittee and attended the planning meetings regularly.

Our feeding intervention was allocated by the school and therefore it was important to examine the outcomes in different schools without the ethical concern of giving children different diets in the same school. As recommended by Kristjansson[133], our study was able to provide disaggregated data by the parents socio-economic status, children's baseline nutritional status, sex and age. The important role of household sociodemographic characteristics especially maternal employment as a determinant of growth for the school children is well demonstrated in our findings. The study provides new knowledge on the effect of initial nutritional status on outcomes of growth, anaemia and vitamin A status following school feeding interventions in the country.

This study forms an appropriate baseline for further investigations in this area, particularly on the possible outcome of targeting children with poor initial nutritional status in relation to micronutrient status following school feeding. The study further lays emphasis on the considerations that need to be put into place to meet specific nutrition needs of children already with compromised nutrition in the design of school feeding programme. There is a need to target feeding to children with already compromised nutritional status while aiming at improved growth and being concurrently careful not to increase the risk of overnutrition. This study also offers an example of the successful efforts going towards the attainment of MDGs particularly on provision of basic education (goal 2), eradication of extreme poverty and hunger (Goal 1; target 2) and developing a global partnership for development.

4.10   Limitations of the study

- The extension of the MV Project into the control areas 6 months into the study was not anticipated at baseline. However, an analysis of the data was done at the six-month interval to highlight any differences in outcomes at that study point.
- The cost of the laboratory analysis of micronutrient status was limiting, leading to the analysis of only in a subsample of children. This limits the generalisability of the findings with regard to micronutrient status. It is recommended that further studies be conducted on a national scale to include larger samples for micronutrient analysis among this age group in order to establish the micronutrient status of school children in the county.
- No data was collected on dietary intake during the intervention, which limited the ability of the study to assess changes in dietary and nutrient intake following the intervention. Data on dietary intake following the intervention was not collected, as it was difficult to follow up the children during the school holidays as many of them were absent from home, having gone to visit relatives during the school holidays.

5 CHAPTER FIVE: CONCLUSION AND RECOMMENDATIONS

5.1   Conclusions

1. Dietary intake by school children in the Millennium Villages Project and in the control schools was predominantly plant-source foods at baseline, accompanied by a high probability of inadequate energy, fat, zinc, folate and selenium intake. The dietary intake of vitamin A and C in both groups was adequate as per the EAR for the age and sex of the children.

2. Undernutrition is a persistent problem among school children in Kenya, with linear growth failure being the most common form of growth retardation. At the same time, the emergence of overweight in this age group is of concern, as it is an indication of the double burden of under- and overnutrition that characterises nutrition transition and poses a challenge when designing programmes to meet the needs of children across the two nutritional status spectra. Child's age and maternal employment status were significant determinants of linear growth deficit. Linear growth deficit and wasting were more prevalent among older children, while lack of maternal employment was a predictor of linear growth failure.

3. Children were generally lean, with body composition measurements at the third percentile for triceps skinfold thickness and within the 25^{th} percentile for subscapular skinfold thickness compared to the NHANES reference points for boys and girls.

4. Anaemia was a severe public health problem among the children in the MVP and the control groups at baseline, while one in every ten children had iron deficiency anaemia. Stunting, wasting and underweight were significant determinants of anaemia in the multivariate analysis. The likelihood of being anaemic increased with the child's age. Depleted body iron stores were observed more commonly among the boys.

5. Inadequate vitamin A status is common among Kenyan school children, with a prevalence of more than one third observed in the current study population, making it a severe public health problem. Household socioeconomic status, characterised by unemployment of the household head, total household income less than KES 1, 000 per month and being from the control group, were predictors of inadequate vitamin A status among school children in the MVP and control areas. Concurrency of anaemia, depleted body iron stores and inadequate vitamin A status was observed among the children.

6. The school feeding programme that included meat and *omena* fish in the school lunch improved the growth and lean body mass of the children in the MVP group and was the most significant predictor of reduced prevalence of anaemia at the end of the intervention.

7. There was no effect of the school feeding programme in the MVP and control groups on body iron stores and vitamin A status, possibly as a result of the depletion of nutrient status during the school holidays and the limited amounts provided by the diet.

8. The initial nutritional status affected the responses to the school feeding programme, as demonstrated by accelerated growth among MVP children with initial growth stunting. Including meat and fish (*omena*) in the school meal demonstrated improved growth potential for children who had initial poor nutritional status.

5.2   Policy Recommendations

1. Participatory and community-initiated and implemented school feeding programmes can be successful in meeting the physical growth needs of school children and addressing micronutrient deficiencies and should go hand in hand with the education for all agenda in order to optimise the time children have in school for the achievement of MDG 2 and the implementation of the new Constitution, as per articles 53 to 59 of the new Kenyan Constitution of 2010.

2. In view of the modest improvement in growth and body composition and the reduction in anaemia observed in this study, especially in the MVP group with a diversified diet, cost-effective community-based initiatives and approaches should be promoted in order to meet the nutrition and health needs of school children in the country. This is a recommendation that the Ministry of Education together with the County education directorate should take into serious consideration when making national and county strategic development plans in education sector.

3. Dietary diversification, including animal-source foods in school feeding programmes, is recommended for improved outcomes in growth and a reduction of anaemia among school children. At present, school feeding programmes in Kenya are run mainly by the World Food Program and concentrated in the arid and semi-arid regions of the country. Efforts should be made to implement a multifaceted approach that includes mobilising communities to initiate school feeding programmes in all regions of the country to support the government-provided free primary education programmes.

5.3   Recommendations for Further Research

1. Baseline data is lacking on the nationwide distribution of multiple micronutrient deficiencies among school children thus creating an urgent need for a survey to establish the distribution so as to better understand the extent of the problem in the country. This will allow for proper health and nutrition planning and policy decisions that affect the children in the country as this is a critical age and population group making up about one third of the population in the Kenya.

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2. The emergence of overnutrition among rural Kenyan school children that co-exists with undernutrition warrants for further studies to investigate specific dietary patterns habitual physical activity and physical fitness among school children to clearly define the determinants of the dual burden.

3. Further research on the education outcomes in relevance to initial nutritional status with a targeted school feeding programmeis required to assess the effect of the initial nutritional status on education indicators following targeted interventions.

4. A detailed cost effectiveness analysis for community based school feeding programmewill provide clearer details on the requirements for a sustainable community based targeted school feeding programme.

5. A targeted school feeding design model should be tested that involves the community on key decisions on how to provide additional dietary requirement to the children with inadequate nutritional status incorporated within a broader intervention for all the school population in a given area. This will require a careful consideration of how to target the children with inadequate nutrition within a broad framework of a school feeding programme for all the children and being able to address the specific needs of those with inadequate nutrition while avoiding social stigmatisation and maintaining a cost sustainable community –involved programme.

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APPENDICES

Appendix 1: Model for Determining Sample Size

POWER ANALYSIS FOR THE DIFFERENCE BETWEEN TWO BERNOULLI PROPORTIONS				
CHOOSE ALPHA%	5%	Z(alpha/2) =	1.959964	
CHOOSE BETA%	10%	Z(beta) =	1.281552	
GIVE YOUR GUESSES ABOUT p1 AND p2, MAYBE FROM A PILOT STUDY AND CHOOSE THE RATIO k = n2/n1.				
p1 =	0.5		q1=	0.5
p2 =	0.19[a]		q2=	0.71
k = n2/n1 =	1			
SAMPLE SIZE n1	**118**			
ARC-SINE METHOD:	Paulson and Wallis (1947)	AS	CALCU-LATIONS	
n1 =	112			
n2 =	112			
CONTINUITY CORRECTED ARCSINE METHOD		NAS	Z=	4.5841 95
n1 =	125		C=	4.2037 98
n2 =	125		DELTA =	0.2167 23
SIMPLE NORMAL USING HETEROGENEITY		NH		
n1 =	109			
n2 =	109			
DESU AND RAGHAVARAO METHOD		DR		
n1 =	**118**			0.211 575
n2 =	**118**			
ASYMPTOTIC NORMAL METHOD:	Rosner (1990), Fleiss (1981)	AN	b =	0.21 6 0
n1 =	60		pbar =	0.395
n2 =	60		qbar =	0.605
ASYMPTOTIC NORMAL METHOD WITH YATES CORRECTION		ANY		
n1 =	78	NCC		
n2 =	78			
MODIFIED ASYMPTOTIC NORMAL METHOD WITH CORRECTION		ANF		
n1 =	69	MON		
n2 =	69			
SHORTCUT METHOD TO THE ANF METHOD		SN		
n1 =	69			
n2 =	69			
CHI-SQUARE WITH CONTINUITY CORRECTION		CHI		
n1 =	69			
n2 =	69			

Source: model provided by the Centre for Statistical Consultation, Stellenbosch University; [a]Proportion of stunting among school children in rural Kenya[50]

Appendix 2: Market Survey Tool

Millennium Villages Project: Yala Division; Market Survey Tool for the Study on:

Effects of initial nutritional status on the responses in young children to a school feeding programme

This tool is to be used to survey the availability of foods at the Yala and the Luanda markets

Date of survey

Market name	Name of food items
Name of field assistant	
Market section	
Grains, legumes, seeds and nuts	
Vegetables	
Fruits	
Roots and tubers	
Meats, fish, chicken and meat products	
Others	

Appendix 3: Menu Plans and Nutrient Content of School Feeding Programme

Millennium Villages Project: School Feeding Programme: Nutrient Content

MENU OPTION	NUTRIENT ANALYSIS									
NURSERY SCHOOL (4–6 yrs)										
	FOOD TYPE	QUANTITY (G)	Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
1 (42 days/ term)	Maize	100	119.0	25.4	2.7	1.2	0.0	1.2	2.0	0.2	0.0
	Beans	100	127.0	22.8	8.7	0.5	0.0	2.9	28.0	130.0	1.1
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
	Milk (1/4 cup)	30	21.8	1.7	1.0	1.2	1.5	0.5	54.2	1.8	0.1
	Sugar	3	11.3	3.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Maize meal porridge	125	75.0	31.6	1.7	0.6	0.0	2.7	3.9	10.0	0.8
NUTRIENT CONTENT			372.4	84.6	14.1	5.5	1.6	7.4	88.3	142.2	2.0
2 (14 days/ term)	Ugali	200	220.0	50.6	2.6	0.9	0.0	4.3	6.3	16.0	1.2
	Omena	30	69.0	3.9	8.4	4.8	0.0	0.2	13.5	1.8	0.1
	Vegetables (kale)	20	10.4	2.0	0.8	0.1	30.0	0.4	20.0	2.6	0.0
	Fruit (small mango/banana/orange)[1]	30	18.0	4.5	0.2	0.1	20.0	0.1	3.6	4.2	0.0
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
	Milk (1/4cup)	30	21.8	1.7	1.0	1.2	1.5	0.5	54.2	0.0	0.0
	Sugar	3	11.3	3.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Maize meal porridge	125	75.0	31.6	1.7	0.6	0.0	2.7	3.9	10.0	0.8
NUTRIENT CONTENT			443.8	97.4	14.7	9.6	51.6	8.1	101.6	34.8	2.1
2 (14 days/ term)	Ugali	200	220.0	50.6	2.6	0.9	0.0	4.3	6.3	16.0	1.2
	Meat	20	65.0	0.0	5.0	3.7	0.8	0.4	0.8	1.2	0.8
	Vegetables (kale)	20	10.4	2.0	0.8	0.1	30.0	0.4	20.0	2.6	0.0
	Fruit (mango/banana/orange)[1]	30	18.0	4.5	0.2	0.1	20.0	0.1	3.6	4.2	0.0

Millennium Villages Project: School Feeding Program: Nutrient Content (continued)

MENU OPTION			NUTRIENT ANALYSIS								
NURSERY SCHOOL (4–6 yrs)											
	FOOD TYPE	QUANTITY (G)	Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
	Milk (1/4 cup)	30	21.8	1.7	1.0	1.2	1.5	0.5	54.2	0.0	0.0
	Sugar	3	11.3	3.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Maize meal porridge	125	75.0	31.6	1.7	0.6	0.0	2.7	3.9	10.0	0.8
NUTRIENT CONTENT			439.8	93.5	11.2	8.5	52.4	8.4	88.9	34.2	2.8
STANDARD 1-3 (7–10 yrs)											
MEAL OPTION	FOOD TYPE	QUANTITY (G) /CHILD	Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
	Maize	150	178.5	38.1	4.1	1.8	0.0	1.8	3.0	195.0	1.7
	Beans	150	190.5	34.2	13.1	0.8	0.0	4.4	42.0	0.0	0.0
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
NUTRIENT CONTENT			387.4	72.4	17.1	4.6	0.1	6.2	45.2	195.2	1.7
	Ugali	300	330.0	75.9	4.0	1.4	0.0	6.4	9.4	24.0	1.8
	Omena	40	92.0	5.2	11.2	6.4	0.0	0.2	18.0	2.4	0.2
2 (14 days/term)	Vegetables (kale) [1]	30	15.6	3.0	1.1	0.1	45.0	0.6	30.0	3.9	0.1
Fish	Fruit (mango/banana/orange) [1]	30	18.0	4.5	0.2	0.1	20.0	0.1	3.6	4.2	0.0
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
	Salt										

Millennium Villages Project: School Feeding Program: Nutrient Content (continued)

MENU OPTION			NUTRIENT ANALYSIS								
NURSERY SCHOOL (4–6 yrs)											
	FOOD TYPE	QUANTITY (G)	Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
	NUTRIENT CONTENT		474.0	88.6	16.5	9.9	65.1	7.3	61.2	34.7	2.0
	Ugali	300	330.0	75.9	4.0	1.4	0.0	6.4	9.4	24.0	1.8
	Meat	30	97.5	0.0	7.5	5.6	1.2	0.6	1.2	1.8	1.2
2 (14 days/term)	Vegetables (kale)	30	15.6	3.0	1.1	0.1	45.0	0.6	30.0	3.9	0.1
Beef	Fruit (mango/banana/orange)1	30	18.0	4.5	0.2	0.1	20.0	0.1	3.6	4.2	0.0
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
	NUTRIENT CONTENT		479.5	83.5	12.8	9.1	66.3	7.7	44.4	34.1	3.1
STANDARD 4-8 (11–15yrs)											
MEAL OPTION	FOOD TYPE	QUANTITY (G)/CHILD	Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
	Maize	200	238.0	50.8	5.4	2.4	0.0	2.4	4.0	16.0	1.2
	Beans	200	254.0	45.6	17.4	1.0	0.0	5.9	56.0	260.0	2.2
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
	NUTRIENT CONTENT		510.4	96.5	22.8	5.4	0.1	8.3	60.2	276.2	3.4
			Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
	Ugali	350	385.0	88.6	4.6	1.6	0.0	7.5	11.0	28.0	2.1
	Omena	50	115.0	6.4	14.0	8.0	0.0	0.3	22.5	3.0	0.2
2 (28 days/term)	Vegetables (kale)	35	18.2	3.5	1.3	0.1	52.5	0.6	35.0	4.6	0.1
	Fruit (mango/banana/orange)1	35	21.0	5.3	0.2	0.1	23.3	0.1	4.2	4.9	0.0
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0

Millennium Villages Project: School Feeding Program: Nutrient Content (continued)

MENU OPTION	NUTRIENT ANALYSIS									
NURSERY SCHOOL (4–6 yrs)										
	FOOD TYPE	QUANTITY (G)	Energy (kcals)	Carbohydrate (g)	Protein (g)	Fat (g)	Vitamin A (ug)	Iron (mg)	Calcium (mg)	Folate (ug)	Zinc (mg)
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
NUTRIENT CONTENT			557.6	103.8	20.2	11.7	76.0	8.5	72.9	40.6	2.4
	Ugali	350	385.0	88.6	4.6	1.6	0.0	7.5	11.0	28.0	2.1
	Meat	35	113.8	0.0	8.7	6.5	0.3	0.7	1.4	2.1	1.4
2 (28 days/ term)	Vegetables (kale)	35	18.2	3.5	1.3	0.1	52.5	0.6	35.0	4.6	0.1
	Fruit (mango/banana/orange) [1]	35	21.0	5.3	0.2	0.1	23.3	0.1	4.2	4.9	0.0
	Fat	2	18.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0
	Onions	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	Tomatoes	1	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.2	0.0
NUTRIENT CONTENT			556.4	97.4	14.9	10.3	76.2	9.0	51.8	39.7	3.6

[1] This is the average amount of nutrients in the three fruits combined. the amounts were determined in each fruit and then averaged

Appendix 4: Socio-Demographic Questionnaire

EFFECTS OF INITIAL NUTRITIONAL STATUS ON THE RESPONSES TO A SCHOOL FEEDING PROGRAMME IN YOUNG CHILDREN STUDY QUESTIONNAIRE

The following questions are designed to collect information concerning the effects of initial nutritional status on the responses to a school feeding programme among school children aged 6 to 13 years in the Millennium Villages Project, in Yala division, Siaya, Kenya. Your co-operation and patience in answering these questions accurately will be highly appreciated.

A. Demography and socioeconomic information

Child's name_____ ID. no_____ School_____

Class_____   Date_____

			Village Write the name	**Main Respondent**
Village name				
Name of homestead		ID number (Where applicable:	**Relation of household head to child** 1=Father	1=Father 2=Mother
			2=Mother	3=Grandfather
Name of household head		Household ID:	3=Grandfather	4=Grandmother
			4=Grandmother	5=Brother (specify age……)
			5=Brother	6=Sister (specify age……)
Relation of household head to the child			6=Sister	7=Hired caretaker (Specify age……)
			7=Other relative (Specify……….)	7=Other relative (Specify……….)
Employment status of household (HH) head			8=Other non-relative (Specify……..)	8=Other non-relative (Specify……..)
Ethnicity of HH head			**Employment status of household head**	**Household type**
Religion of HH head			1=Unemployed	1= Male headed/single wife
			2=Self-employed	2=Male headed/polygamous
Denomination			3=Temporary employment (specify….)	3=Female headed widowed-single
Main respondent			4=Permanent employment (specify…….)	4=Female headed widowed-polygamous
Household type				5=Female headed husband away
Mother's level of education			**Ethnicity** 1=Luo	6=Male headed, divorced or single, widower
Mother's marital status			2=Luhya	7=Female headed, divorced or

| Mother's employment status | | | 3=Kisii
4=Kikuyu
5=Other
(Specify………….)

Religion
1=Christian
2=Muslim
3=Other (Specify……)

Denomination
1=Catholic
2=CCA (Church of Christ in Africa)
3=ACK
4=Roho ga Diera (Spirit and Truth)
5=Africa Israel Nineve
6=Nomiya Church
7=Jehovah's Witness
8=SDA
9=Africa Inland Church
10=Redeemed Gospel
11=Outreach
12=PEFA
13=CHRISCO
14=Other
Specify………….. | single
8=Child headed (aged 16 or under) – Orphan
9=Other (Specify……………)

Mother's level of education
1=Standard 1-4
2=Standard 5-8
3=Not completed secondary education
4=Completed secondary education
5=College education

Marital status of the mother
1=Single
2=Monogamously married
3=Polygamously married
4=Widowed
5=Separated/Divorced
6=Minor (aged 16 or under)

Mother's employment status
1=Housewife
2=Self-employed
3=Temporary employment (specify….)
4=Permanent employment (specify…….) |
| Total household income per month. (including salaries, wages, business, and all other sources) | | | | **Total household income per month (KES)**
1=less than 1 000
2=1 000 – 5 000
3= 6 000 – 10 000
4=11 000 – 20 000
5= 21 000 – 40 000
6=41 000 – 50 000
7=More than 51 000 |

B. Dietary intake data

Food frequency questionnaire

Information for this section should be collected from the mother or caregiver of the child.

In order for us to understand better if the children in the primary schools are having an adequate diet, I am going to ask you about foods and drinks the child has taken in the last six months before the month of study.

I will now go through a list of foods and drinks with you and I would like you to tell me:

1.   If the child eats these particular foods,

2.   How much of the food the child eat at a time, and

3.   How many times a day the child eats it and if he or she does not eat it every day, how many times a week or a month it is eaten?

FOOD FREQUENCY QUESTIONNAIRE RECORD TABLE

			Quantity	Amount usually eaten	Frequency of intake			
Food type	Code	Method of preparation	H/h meas-ures	H/h meas-ure	Per day	Per week	Per month	Never
Staple starches								
Ugali	100	Whole maize meal	Bowl					
	101	Refined maize meal	Bowl					
	102	Sorghum meal	Bowl					
	103	Millet meal	Bowl					
Boiled/roasted green maize	104	Boiled	Piece					
	105	Roast	Piece					
Rice	106	Boiled	Cup					
	107	Cooked in fat/oil	Cup					
Nyoyo	108	Nyoyo fried with cooking fat/oil	Cup					
	109	Nyoyo not fried	Cup					
Wheat chapatti	110	Average diameter 20 cm	Whole					
Cooked bananas	111	Boiled green Bananas	Cup					
	112	Green bananas fried with fat/oil	Bowl					
Irish potatoes	113	Boiled Irish potatoes	Cup					
	114	Fried Irish potatoes	Bowl					
Boiled cassava	115		Bowl					
Sweet	116	Sweet	Bowl					

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Food type	Code	Method of preparation	Quantity	Amount usually eaten	Frequency of intake			
			H/h meas-ures	H/h meas-ure	Per day	Per week	Per month	Never
potatoes		potatoes boiled with skin						
	117	Sweet potatoes boiled without skin	Bowl					
Nduma (arrow roots)	118	Nduma boiled	Cup					
	119	Nduma fried	Bowl					
Porridge	120	Mixed grain porridge	Cup					
	121	Millet porridge	Cup					
	122	Sorghum porridge	Cup					
	123	Maize meal porridge (whole meal)	Cup					
	124	Maize meal porridge (refined maize meal)	Cup					
Bread	125	Small size (400 g)	Slice					
	126	Large size (800 g)	Slice					
Cereals and legumes								
Cowpeas	127	Boiled cowpeas	Cup					
	128	Fried stew	Cup					
Beans	129	Boiled beans	Cup					
	130		Cup					
Green grams ndengu	131	Boiled ndengu	Cup					
	132	Fried stew	Cup					
Lentils	133	Boiled	Cup					
	134	Fried stew	Cup					
Meats and eggs								
Beef	135	Beef stew fried	Cup					
	136	Beef boiled	Cup					
Chicken with bone	137	Chicken fried	Cup					

Food type	Code	Method of preparation	Quantity H/h meas-ures	Amount usually eaten H/h meas-ure	Frequency of intake Per day	Per week	Per month	Never
	138	Chicken stew	Cup					
Fish	139	Tilapia fish fried (medium size)	Whole					
	140	Nile perch fish fried (medium size)	Piece					
	141	Obambla fried	Bowl					
	142	Obambla boiled	Bowl					
	143	Fulu fried	Cup					
	144	Fulu boiled	Cup					
	145	Omena fried	Cup					
	146	Omena boiled	Cup					
Offal (matumbo)	147	Cooked	Cup					
Liver (all types)	148	Cooked	Cup					
Egg	149	Egg boiled + egg yolk	Whole					
	150	Egg fried + egg yolk	Whole					
Vegetables								
Vegetables fried (with cooking fat/oil)	151	Cowpea leaves	Cup					
	152	Pumpkin leaves	Cup					
	153	Bean leaves	Cup					
	154	Omboga/ ododo/osuga/ dek-akeyo/ mrenda osuga/mito (miro)	Cup					
	155	Mushroom	Cup					
	156	Cabbage	Cup					
	157	Kale (sukuma wiki)	Cup					
	158	Spinach	Cup					
Vegetables boiled without	159	Cowpea leaves	Cup					

Food type	Code	Method of preparation	Quantity H/h meas-ures	Amount usually eaten H/h meas-ure	Frequency of intake Per day	Per week	Per month	Never
fat								
	160	Pumpkin leaves	Cup					
	161	Bean leaves	Cup					
	162	Omboga/ ododo/osuga/ dek-akeyo/ mrenda osuga/mito (miro)	Cup					
	163	Mushroom	Cup					
	164	Cabbage	Cup					
	165	Kale (sukuma wiki)	Cup					
	166	Spinach	Cup					
Onion	167	Amount used in household food preparation	Table-spoon					
Carrots	168	Amount used in household food preparation	Table-spoon					
Tomato	169	Amount used in household food preparation	Table-spoon					
Pumpkin	170	Pumpkin fried	Cup					
	171	Pumpkin boiled	Cup					
Fruit								
Avocado	172	Ripe small	Whole					
	173	Ripe large	Whole					
Ripe banana	174	Ripe small (sweet variety)	Whole					
	175	Ripe large (matoke size)	Whole					
Mango	176	Ripe (local variety)	Whole					
Orange	177	Ripe	Whole					
Lemon	178	Ripe	Whole					
Pawpaw	179	Ripe	Cup					
Guava	180	Ripe	Whole					

Food type	Code	Method of preparation	Quantity H/h measures	Amount usually eaten H/h measure	Frequency of intake Per day	Per week	Per month	Never
Pineapple	181	Ripe	Cup					
Passion fruit	182	Ripe	Whole					
Milk and milk products								
Whole milk	183	Taken alone (not in tea/ beverages)	Cup					
	184	Milk used to cook vegetables	Cup					
	185	Milk in beverages (tea, cocoa)	Cup					
	186	Milk in porridge						
Milk fat	187	Cream from milk used in cooking vegetables	Tea-spoon					
Nuts and seeds								
Ground nuts	188	Roasted and salted	Tea-spoon					
Simsim	189	Roasted and salted made into 'balls' common locally	Piece					
Fats and oils								
Fat	190	All brands of solid vegetable cooking fats (kimbo, kasuku, etc.)	Tea-spoon					
	191	All brands of liquid vegetable cooking oils (elianto)	Tea-spoon					
	192	Margarine	Tea-spoon					
	193	Fat made from milk or meat fat	Tea-spoon					
Sugars								

Food type	Code	Method of preparation	Quantity H/h meas-ures	Amount usually eaten H/h meas-ure	Frequency of intake Per day	Per week	Per month	Never
Honey	194	Raw	Tea-spoon					
Sugar	195		Tea-spoon					
Sugar cane	196	In 30 cm pieces as found in local market	Piece					
Others (specify)								

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C. Blood samples: Collection check form

Child's ID number						
Today's date						
Sex (circle one)	Male	Female				
School (circle one)	Bar Turo	Nyamninia	Bar Sauri		Muhanda	Migosi
Class (circle one)	ECD	Class 1	Class 2		Class 3	
Sample collected (tick)	Technician name					
Blood						

D. Anthropometry data entry

Child's ID number							
Today's date							
Sex (circle one)	Male	Female					
School (circle one)	Bar Sauri	Nyamninia	Bar Turo		Migosi	Muhanda	
Class (circle one)	ECD	Class 1	Class 2		Class 3		
	Measure 1		**Measure 2**		**Measure 3**		**Measurer Initials**
Height (cm)							
Weight (pounds)							
Arm length (cm)							
Mid-point of upper arm (cm)							
Mid-upper arm circumference (cm)							
Skinfolds: (mm)							
Triceps							
Subscapular							

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Appendix 5: Serum Sample Storage Tracking Sheets

SERUM SAMPLE STORAGE TRACKING SHEET (PENINAH MASIBO- July – 06)
NAME:_____ DATES_____
BOX NO._____

1	2	3	4	5	6	7	8	9
10	11	12	13	14	15	16	17	18
19	20	21	22	23	24	25	26	27
28	29	30	31	32	33	34	35	36
37	38	39	40	41	42	43	44	45
46	47	48	49	50	51	52	53	54
55	56	57	58	59	60	61	62	63
64	65	66	67	68	69	70	71	72
73	74	75	76	77	78	79	80	81

COLUMBIA UNIVERSITY
INSTITUTIONAL REVIEW BOARD

IRB#:AAAC5773   Approval Date: 10/11/06

IRB Initials: ALK   Expiration Date: 10/10/07

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Appendix 6: Serum Sample Transportation Permit From the Ministry of Medical Services (Kenya)

MINISTRY OF MEDICAL SERVICES
OFFICE OF THE DIRECTOR OF MEDICAL SERVICES

Telegrams: "MINHEALTH". Nairobi
Telephone: Nairobi 2717077
Fax: 2713234
When replying please quote

AFYA HOUSE
CATHEDRAL ROAD
P.O. Box 30016
NAIROBI

Ref No. MMS/ADM/3/8 Vol I

Date: 5th March 2009

Peninah Kinya Masibo
P. O. Box 4606-30100
ELDORET

RE: CLEARANCE FOR RESEARCH MATERIAL TRANSPORTATION TO THE REPUBLIC OF SOUTH AFRICA

Permission is hereby granted to transport serum samples on 16th March 2009 to Tygerberg Campus, Stellenbosch University, South Africa for analysis of micronutrient status.
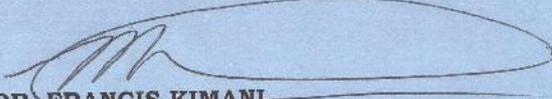
DR. FRANCIS KIMANI
DIRECTOR OF MEDICAL SERVICES

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Appendix 7: Sample Results for Blood and Serum Laboratory Analysis

Sample results for full haemoglobin

US ARMY MED RESEARCH UNIT
P.O BOX, 54, KISUMU

WALTER REED KISUMU
TEL; 057-2022942

CLINICAL TRIAL CENTER
FAX; 057-2022903

Sample ID: 1535 Patient Name:
Run Date/Time: 21/05/2008 04:27:11 PM Patient ID: KC2262 Collect Date/Time:
Seq#: 3014 Gender: Male DOB: Physician:
OPR: BCI Age: Location:
Flagging Set: Child
Comments:

|				Range					Range	Flags and Messages
WBC	4.7	L	10^3/µL	6.0 / 14.0	NE	46.9		%	40.0 / 65.0	**Diffplot and Histogram Flags**
					LY	41.8	H	%	25.0 / 40.0	ATL
RBC	4.70		10^6/µL	4.00 / 5.20	MO	8.9		%	3.0 / 10.0	
HGB	12.4		g/dL	11.5 / 15.5	EO	1.7		%	1.0 / 5.0	
HCT	37.6		%	35.0 / 45.0	BA	0.7	H	%	0.0 / 0.5	
MCV	80		fL	77 / 95						
CH	26.5		pg	23.0 / 31.0						
MCHC	33.2	H	g/dL	28.0 / 33.0	NE#	2.18		10^3/µL	2.00 / 6.00	
RDW	13.2		%	11.0 / 16.0	LY#	1.94	L	10^3/µL	5.00 / 8.50	
					MO#	0.41	L	10^3/µL	0.70 / 1.50	
PLT	197		10^3/µL	150 / 400	EO#	0.08	L	10^3/µL	0.30 / 0.80	
MPV	8.7		fL	6.0 / 10.0	BA#	0.03		10^3/µL	0.02 / 0.10	

Vol

RBC

50 100 200 300 fL

PLT

2 10 20 30 fL

WBC/BASO

100 200 300 400 fL

Abs
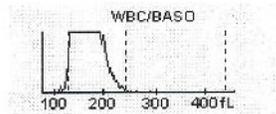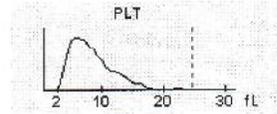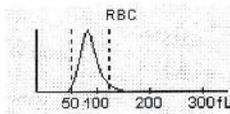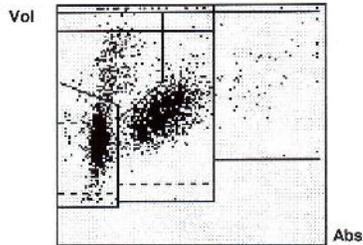
Microscopic Examination

Neutrophils	_____	Metamyelocytes	_____	Anisocytosis	_____	Retics	_____
Bands	_____	Myelocytes	_____	Hypochromia	_____	Sed. Rate	_____
Lymphocytes	_____	Promyelocytes	_____	Polychromasia	_____		
Monocytes	_____	Blast	_____	Poikilocytosis	_____		
Eosinophils	_____	Atyp. Lymph	_____	Microcytosis	_____		
Basophils	_____	NRBC s	_____	Macrocytosis	_____		

Comment : _____ Requested by : _____

_____ Reviewed by : Do 21mo1J0

Out of Action Range XXX Out of Patient Range XXX Printed 21/05/2008 04:27:16 PM

Serum ferritin and retinol, laboratory test report

Division of Human Nutrition, Stellenbosch University

Committed to Research and Service Excellence

PENINAH K MASIBO
MOI UNIVERSITY
SCHOOL OF PUBLIC HEALTH
DEPARTMENT OF EPIDEMIOLOGY AND NUTRITION
P.O BOX 4606
30100
ELDORET
KENYA

10 October 2009

Dear Peninah

Test reports for The Millennium Villages Project

I attach the original reports PK001/2009, PK002/2009 and PK003/2009 for your records. We would also appreciate your feedback on the services of the AMRG and kindly ask you to complete the included questionare.

Best regards
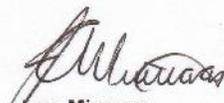
Jaco Minnaar
Laboratory Manager

Fakulteit Gesondheidswetenskappe • Faculty of Health Sciences

Verbind tot Optimale Gesondheid • Committed to Optimal Health
Division of Human Nutrition • Department of Interdisciplinary Health Sciences
PO Box 19063 • Tygerberg 7505 • South Africa
Tel.: +27 21 938 9259 • Faks/Fax: +27 21 933 2991
Webblad / Web page: www.sun.ac.za/nutrition; www.sun.ac.za/nicus

Sample results for serum ferritin and retinol, laboratory report

Document Title: Client Report Format II
Revision Number: 004
Document Number: QF020

Revised by: Alethia van Rensburg
Revision Date: August 2009

Page 2 of 3

Lab No	Sample No	Retinol [a] (µg/dl)	Ferritin [#] (µg/l)	CRP [#] (mg/l)
PK/236/09	HE 58	13.6	52	13.9
PK/237/09	HC 175	18.71	72	4.4
PK/238/09	HC 200	20.87	80	7.5
PK/239/09	HC 209	10.32	25	0.2
PK/240/09	HE 59	27.45	43	< 0.2
PK/241/09	HC 179	24.99	34	< 0.2
PK/242/09	HC 184	16.57	26	1.4
PK/243/09	HC 180	13.91	13	1.2
PK/244/09	HC 186	13.75	40	0.8
PK/245/09	SAC 10040	33.44	42	< 0.2
PK/246/09	SAC 10048	32.28	28	< 0.2
PK/247/09	SAC 10042	23.55	25	< 0.2
PK/248/09	SAC 10044	14.23	48	10
PK/249/09	SAC 10054	16.23	37	0.8
PK/250/09	SAECD 002	26.2	19	0.3
PK/251/09	SAECD 022	22.41	13	0.9
PK/252/09	SAECD 019	21.61	37	2.2
PK/253/09	SAECD 004	11.28	11	1.9
PK/254/09	SAECD 030	15.73	3	0.4
PK/255/09	SAECD 032	16.68	12	4.2
PK/256/09	SAECD 011	26.79	41	0.7
PK/257/09	SAECD 10053	12.05	36	0.4
PK/258/09	SAC 10041	23.91	59	0.4
PK/259/09	SAECD 012	14.03	20	1.3
PK/260/09	MC 243	10.88	53	4.6
PK/261/09	MC 121	14.94	83	2.5
PK/262/09	MEC 6	29.74	56	0.4
PK/263/09	MC 129	16.41	49	1.9
PK/264/09	MEC 7	23.87	22	< 0.2
PK/265/09	MC 372	23.33	25	0.3
PK/266/09	MC 247	24.37	62	1.2
PK/267/09	MC 242	20.87	65	0.6
PK/268/09	MC 120	17.32	58	0.3

Source: The Millenium Villages Project
Instrument used (if applicable): Waters Alliance HPLC
BN ProSpec Nepholometer

sanas
Pharmaceutical Laboratory

P0002   Technical signatory sign: ЭѠ

Date issued:2009/09/01

Tests marked with a # in this report are not included in the SANAS Schedule of Accreditation for this laboratory

"Opinions and Interpretations expressed herein are outside the scope of SANAS accreditation.

For further enquiries: Jaco Minnaar (Lab.Manager) @ tel: (021) 938-6312 or 5272 or email:
jaminnaar@sun.ac.za

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Appendix 8: Lange Skinfold Callipers Service Document

CREATIVE HEALTH PRODUCTS
SERVICE CENTER DIVISION
Service Report

Date 12-14-06

20258

Inv # SNP 1670

Customer # 32287   PO# XA717N   Ship Via UPS

Name/Bill To: COLUMBIA UNIVERSITY   Ship To:
            630 WEST 168th St.
            PHE15 - 1512
            NEW YORK, NY 10032

Phone Number Home 212-305-4808   Phone Number Office
        (YONETTE BEATIN)

Card # _____   Check # _____   Amount _____
Exp. Date _____ App. Code _____   Cardholder Name _____

TYPE OF PRODUCT_____ MAKE AND MODEL _____ SERIAL NO._____
OTHER ITEMS INCLUDED WITH PRODUCT (CASE, MANUAL, ETC.)_____
PROBLEMS FOUND JAWS LOOSE ON GEARS (CAUSED BY ALLOWING JAWS TO SNAP SHUT. LENS LOOSE

SERVICE PERFORMED TIGHTENED JAWS ON GEARS AND ALIGNED. LUBRICATED CALIBRATED AND REGLUED LENS

COMMENTS DO NOT ALLOW JAWS TO SNAP SHUT. CLOSE GENTLY

Warranty Info: Date of Purchase_____ Copy of Invoice or Other Record: Yes_____ No_____

Quantity	Part #	Description	Price	Total

GAGE   READING

10 mm   10.0
20   19.9
30   29.9
40   39.7
50   49.8

Michigan Sales Tax on Parts
Service/Repair Labor   50.00
Shipping   7.50
Total   57.50

SERVICE CENTER
CREATIVE HEALTH PRODUCTS
7621 E. Joy Road, Ann Arbor, MI 48105
800-742-4478 9:00 AM - 4:30 PM EST

20258

Appendix 9: Serum Samples Shipment Consignment

THE MILLENNIUM VILLAGES PROJECT

CUSTOMS INVOICE

Date: March 18, 2009

Shipper:

Peninah K Masibo
ICRAF- Millennium Villages Project
P. O. Box 2389,
40100
Tel 0734 756633 or 0721 952175

Consignee:

Division of Human Nutrition,

Room 3083, 3rd floor. Clinical Building

University of Stellenbosch, Franzie van Zyl Avenue

Tygerberg, 7505, SOUTH AFRICA

T: 09 27 21 938 9205
F: 09 27 21 933 2991

Contents: NON-INFECTIOUS HUMAN SERUM SAMPLES
AS BIOLOGICAL SUBSTANCE, CATEGORY B, UN3373,
Packed in compliance with IATA PACKING Instruction 650.
PACKED ON DRY ICE UN1845, PGIII, PKI904 QTY 10KGS
Quantity: __280*2VIALS X 0.05 ML

For Laboratory Testing Only.
For drug level evaluation in a clinical study.
Human material containing no animal material
And not of tissue culture origin.
Human material that was neither inoculated with nor exposed to
Agents of agricultural concern, including zoonotic agents.

No Commercial Value - For Customs Purposes Only $ 10

DR. PENINAH MASIBO

THE MILLENNIUM VILLAGES PROJECT (MVP), MILIMANI, BUSIA ROAD – BLOCK 10/35
C/O ICRAF –KISUMU, P.O. Box 2389 - 40100, KISUMU
Tel: 254 (0)57 – 2023191/2023194/2023204   FAX : 2023211 Celtel: +254 0736 200026 Safaricom: +254 0726 988866

Appendix 10: Ethical Approvals

INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471//2/3

MOI UNIVERSITY
FACULTY OF HEALTH SCIENCES
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

Reference: IREC/2006/17
Approval Number: 000199

4th August, 2006

Ms. Peninah Masibo,
Moi University
School of Public Health,
Department of Epidemiology & Nutrition,
P.O. Box 4606,
ELDORET

Dear Ms. Masibo,

RE: FORMAL APPROVAL

The Institutional Research and Ethics Committee has reviewed your research proposal titled:

"Effects of Initial Nutritional Status on the Responses to a School Feeding Program in Young Children".

The proposal has been granted a Formal Approval Number: **FAN: IREC 000199** on 3rd August, 2006 and you are therefore permitted to commence your investigations.

You are required to submit progress report(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change (s) or amendment (s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Yours Sincerely,

PROF. J. K. ROTICH
FOR: PROF. D. NGARE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc: Director – MTRH
     Dean   – SOM
     Dean   – SPH

APPROVED
04 AUG 2006
P.O. Box 4606 ELDORET

UNIVERSITEIT·STELLENBOSCH·UNIVERSITY
jou kennisvennoot • your knowledge partner

13 April 2006

Ms PK Masibo
Discipline of Human Nutrition
Dept of Interdisciplinary Health Sciences

Dear Ms Masibo

RESEARCH PROJECT: "EFFECTS OF INITIAL NUTRITIONAL STATUS ON THE RESPONSES TO A SCHOOL FEEDING PROGRAM IN YOUNG CHILDREN"

PROJECT NUMBER : N06/01/002

At a meeting of the Committee for Human Research that was held on 7 February 2006 the above project was approved on condition that further information that was required, be submitted.

This information was supplied and the project was finally approved on 12 April 2006 **for a period of one year from this date.** This project is therefore now registered and you can proceed with the work. Please quote the above-mentioned project number in all further correspondence.

Please note that a progress report (obtainable on the website of our Division) should be submitted to the Committee before the year has expired. The Committee will then consider the continuation of the project for a further year (if necessary).

Patients participating in a research project in Tygerberg Hospital will not be treated free of charge as the Provincial Government of the Western Cape does not support research financially.

Due to heavy workload the nursing corps of the Tygerberg Hospital cannot offer comprehensive nursing care in research projects. It may therefore be expected of a research worker to arrange for private nursing care.

Yours faithfully

CJ VAN TONDER
RESEARCH DEVELOPMENT AND SUPPORT (TYGERBERG)
Tel: +27 21 938 9207 / E-mail: cjvt@sun.ac.za

CJVT/ev

Copy to: Prof D Labadarios

C:\DOCUMENTS AND SETTINGS\EVS\CACIE.SKOWY DOCUMENTS\KH4\PROJEKTE\2006\N06-01-002-001.DOC

Fakulteit Gesondheidswetenskappe · Faculty of Health Sciences

Verbind tot Optimale Gesondheid · Committed to Optimal Health
Afdeling Navorsingsontwikkeling en -steun · Research Development and Support Division
Posbus/PO Box 19063 · Tygerberg 7505 · Suid-Afrika/South Africa
Tel: +27 21 938 9677 · Faks/Fax: +27 21 931 3352
E-pos/E-mail: rdsdinfo@sun.ac.za

Columbia University

Certification of Training

This is to certify that

Peninah Masibo (pm2266)

Has successfully completed Columbia University's course entitled

"Morningside Human Subjects Training Course"

by examination on 04/03/2006.
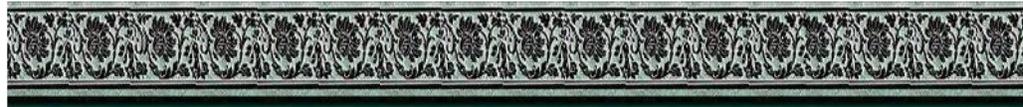
Appendix 11: Informed Consent: English Swahili and Dholuo Translations and Translation

Certificate

English version

Participant Information Leaflet and Consent Form For Use By Parents/Legal Guardians

Study Title: Effects of Initial Nutritional Status on the Responses to a School Feeding Programme among School Children Aged 6 to 13 Years in the Millennium Villages Project, Siaya, Kenya

Principal Investigator: Peninah Kinya Masibo

Address: Department of Epidemiology and Nutrition; School of Public Health, Moi University, Moi University, P.O Box 4606, 30100, Eldoret, Kenya

Contact Number: 0734 756633

Introduction:

Your child is being invited to take part in a study assessing the nutritional status of school children in the millennium village's project. Please take some time to read the information presented here, which will explain the details of this study project. Please ask the study staff any questions about any part that you do not fully understand. Your child's participation is entirely voluntary and you are free to decline to participate. If you say no, this will not affect you or your child negatively in any way whatsoever. You are also free to withdraw him/her from the study at any point, even if you do initially agree to let him/her take part.

- The purpose of the study is to measure how well the school children are growing by taking their body measurements of height, weight, arm circumference and skinfold thicknesses (amount of fat under the skin), and their food intake. The study will also determine the amount of minerals and vitamins in your child's body.

The study will involve the following procedures:

a. The study will involve a total of 146 children in ecd, class 1, 2 and 3 in Bar Sauri, Nyamninia and Bar Turo primary schools. These schools have been selected randomly to represent all the schools in the clusters of the village project. The selection of the children to participate is done by random sampling and any of the school children can be selected. The names of all the children in the 3 classes have been typed into a computer and a command given to select a random sample of names.

b. The project staff will ask the mother or caretaker of the child about the foods and drink the child has eaten/drank and how often the child eats the foods and this information will be recorded in a questionnaire.

c. The child's body measurements of weight and height and the Upper Arm will be taken. These measurements will be repeated every term and will help to check the growth pattern of the child. The body measurements will not cause any pain.

d. In order to check the amount of vitamins and minerals the child has, five (5) mls of blood will be taken from the child's arm. It may be painful when the blood sample is being collected but the pain will quickly go away. This blood will be used to check for the amount of iron and vitamin A stores in the child's body.

e. The information collected will be treated as confidential and the names of your child will not be used in any way against him/her. The information will help us to identify the nutritional needs of school children in the clusters area of the millennium villages project. Your child will be treated for anemia if the tests carried out find them to be having a lack of iron. There will be no costs involved for you if your child takes part in this study.

f. You will be given a copy of this information and consent form for your own records.

Acceptance by the Child

I (*Name of the Child*)……………………………………………………. have been invited to take part in the above study and the project staff and my parents have explained the details of the measurements to me and I understand what they have said to me.

By writing my name below, I voluntary agree to take part in this project. I confirm that I have not been forced either by my parents or the staff to take part.

Name of child Independent witness

(To be written by the child if possible)

Declaration by parent/legal guardian

By signing below, I *(name of parent/legal guardian)* ……………………………………...……. agree to allow my child (name of child) …………………………………… who is ………. years old, to take part in the study entitled: Health and nutritional status of school children in the millennium development villages project (clusters group).

Signed at (*place*) ....…........……………. On (*date*) …………....………..

....
Signature of parent/legal guardian   Signature of witness

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Declaration by Enumerator

I *(name)* …………………………………………….……… declare that: I explained the information in this document to …………………………………..,I encouraged him/her to ask questions and took adequate time to answer them and I am satisfied that he/she adequately understand all aspects of the research, as discussed above.

Signed at (*place*) ....….......……………... On (*date*) …………....………... .

....
Signature of Enumerator   Signature of witness

COLUMBIA UNIVERSITY	
INSTITUTIONAL REVIEW BOARD	
IRB#:AAAC5773	Approval Date: 10/11/06
IRB Initials: _ALK_	Expiration Date: 10/10/07

Consent form Swahili version

Ukurasa wa taarifa na fomu ya kibali ya kutumiwa na wazazi au walezi halali

Mada ya mradi wa utafiti: jinzi lishe la watoto linavyo lingana la hali ya kulishwa kwa watoto shuleni

Mpelelezi mkuu: Peninah Kinya Masibo

                     Idara ya epidemiolojia na lishe

                     Shule ya Afya kwa umma, Chuo kikuu cha moi

                     Saduku la posta 4606, 30100. Eldoret, Kenya

Nambali ya mawasiliano: 0734 756633

Kuanzishwa

Mtoto wako anaalikwa kushiriki katika mradi wa utafiti. Tafadhali chukua muda wako usome habari iliyowakilishwa hapa ambayo inaeleza mengi kuhusu mradi huu. Tafadhali uliza wasaidizi au wachunguzi maswali yoyote kuhusu sehemu yeyote ya mradi huu ambayo hauilewi barabara. Ni mhimu sana wewe kurithika ili ufahamU kwa wazi utafiti huu unahusu nini na jInsi mwanao anaweza kuhusishwa. Pia kushiriki kwa mtoto wako ni kwa hiari na una uhuru wa kukataa kushiriki. Iwapo utasema la, hii haitaathiri wewe au mwanao kwa njia yoyote. Una uhuru wa kumtoa mtoto wako kutoka kwa uchunguzi katika sehemu yeyote au wakati wowote, hata iwapo mwanzoni utakubali kumwachilia kushiriki. Uchunguzi huu umethibitishwa na kamati za utafiti ya kibinadamu za vyuo vikuu via Stellenbosch (Afrika Kusini), Columbia (Amerika), na Moi (Kenya).

Kusudi la utafiti

1. Kusudi la utafiti huu ni kuchunguza hali ya malisho na ukuzi wa watoto kwa kupima urefu, wepesi na unono kwa mkono na sehemu zinginezo mwilini. Utafiti huu pia utapereza kiasi cha chakula motto ananchokila akiwa nymbani na kupelelza kiasi cha madini iliyo kwa mwili wa motto wako.

Uchunguzi utahusisha mpangilio ufuatao:

a. Uchunguzi utahusisha watoto mia mbili therathini na sita (236) wa shule ya msingi kutoka shule za msingi za Bar Sauri, Nyamninia Bar Turo, Kanyuto, Migosi na Muhanda. Shule hizi zimechaguliwa bila taratibu yeyote kwa ziwakilishe vijiji vinavyo husika kwa murandi wa vijiji wa millennium. Watoto wanoahusika pia wamechagulia bila taratibu yeyote. Majina ya watoto wote katika madarasa tatu waliandikwa kwa kompyuta na ile amri ya kuchagua sampuli ikatumiwa kuchagua majina ya watoto watakao hu.

b. Wafanyi kazi kutoka kwa mradi watatembelea nyumbani kwako na kumuuliza mama au mlezi wa mtoto maswali kuhusu chakula na vinywaji mtoto amekuwa akila na kunywa na kiwango cha chakula na vinywaji hivyo.

c. Mtoto atapimwa uzani, urefu na sehemu ya juu za mkono. Hivi vipimo vitarudiwa kwa mara moja kila mhula ili kuchunguza namna ya ukuaji wa mtoto. Vipimo hivi havitasababisha maumivu yoyote.

d. Ili kuchunguza kiasi cha vitamini na madini mengine katika mwili wa mtoto, milimita tano za damu zitachukuliwa kwa kutoka kwa mkono wa mtoto. Damu hii itatolewa na nurse ambeye anajua kutoa damu kwa watot. Mtoto huenda atahisi uchungu wakati anapotolewa damu lakini uchungu huu utapungua haraka. Damu hii itatumika kuchunguza uwepo wa madini ya iron na vitamini A mwilini

e. Taarifa zilizokusanywa yatashughulikiwa kama siri na majina ya mtoto wako si kutumika kwa njia yoyote dhidi yake / zake. Habari yatatusaidia kutambua mahitaji ya lishe ya watoto wa shule katika eneo la makundi ya mradi wa milenia ya vijiji. Mtoto wako kutibiwa kwa upungufu wa damu kama vipimo kufanyika mnawajua kuwa ni kuwa na ukosefu wa chuma. Hakutakuwa na gharama za kushiriki kwa ajili yako kama mtoto wako inachukua sehemu katika utafiti huu.

Idhini ya mtoto

Mimi (Jina la mtoto)……………………………………………………… Mimi wamealikwa kushiriki katika utafiti wa juu na wafanyakazi wa mradi na wazazi wangu kuwa alielezea maelezo ya vipimo na mimi na mimi kuelewa nini wao akaniambia. Kwa kuandika jina langu hapa chini, mimi hiari unakubali kushiriki katika mradi huu. Mimi kuthibitisha kwamba mimi si kulazimishwa ama kwa wazazi wangu au wafanyakazi kushiriki

....
Jina la mtoto Jina la Shahidi
(Liandikwe na mtoto ikiwezekana)

Ilani ya mzazi au mlezi halali

Kwa kutia sahihi hapa chini, mimi (jina la mzazi au mlezi halali) .... nina kubali kumruhusu mwanangu (jina la mtoto)......................................mwa miaka..........(umri ya mtoto) kuhusika katika mradi huu na uchunguzi huu kuhusu: hali ya kulisha watoto shuleni.

Mahali ya sahihi ....tarehe...............................

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....

Sahihi ya mzazi au mlezi halali   Sahihi ya shahidi

Ilani ya mpelelezi

Mimi (jina)...ninajulisha kuwa

Nimeelezea taarifa kuhusu muradi huu kwa...

Nimemhimiza kuuliza maswali na nikachukua muda ufaao kwa kujibu maswali hayo

Nimerithika kuwa ameelewa barabara vipengele vyote vya utafiti kama vilivyojadiliwa hapo juu

Mahali ya sahihi ....tarehe...............................

....

Sahihi ya mplelezi   Sahihi ya shahidi

COLUMBIA UNIVERSITY	
INSTITUTIONAL REVIEW BOARD	
IRB#:AAAC5773	Approval Date: 10/11/06
IRB Initials: ALK	Expiration Date: 10/10/07

Informed consent (Dholuo version)

It oboke moting'o wenche kod kalatas mar yie mar janyuol kata jarit nyathi

Thuon wach mar nonro: ranyisi motelo korkan tekre chi chiemo e del ma luwore gi chenro moket ne miyo nyithindo matindo chiemo e school

Mpelelezi mkuu: Peninah Kinya Masibo

> Idara ya epidemiolojia na lishe
>
> Shule ya Afya kwa umma, Chuo kikuu cha moi
>
> Saduku la posta 4606, 30100. Eldoret, Kenya

Nambali ya mawasiliano: 0734 756633

Chakrouk

Nyathini Ogwel Mondo obed achiel e nonro mar dongruok. Yie Ikaw thuolo mari Mondo Isom andika man mwloka. Mabiro lero tiend nonro ni mar dongruok.

Wakwayi gimuolo ni yie ioenj jogo manie nonroni psenjo moro amora moluwore kod nonroni to ok iwinj tiende maber. En gima dwarore ni iwinj wachni maler to kod gimomiyo nyathini ikwayo mondo obed achiel e nonroni. Bende ngeni bedo nyathini e nonroni en kuom chiwruok ma bende en thuolo mar wuokie ka ok oneno ka en thuolo. Ka ikwer ni ok ibed achiel kuom nonroni to onge kum moro kata rach moro ma wanyalo nenoe. Bende in thuolo mar golo nyathini e nonroni e sa asaya machinyi odwaro kata obedo ni ne wachoro kode kendo e iyie chutho kata nus.

Tiend nonroni:

- Tiend nonro ni en ni mondo warang ngima motundre kod chiemo maber kuoni nyithindogi kapok chenro mar chiemo ochaki e school, bange ibiro nono kaka keto chenro mar chiemo otudore kod somo maber mar nyithindo e class. Duoko maber e nonroni ibiro tiyogodo kuom keto chenro maber mar chiemo sauri kendo timo kamano kuonde duto ma project mar millenium timore e Africa.

Nonroni luwo kama

(a) Nonroni ibiro timo gi nyithindo 136 e skunde mag primary kaka, Bar Sauri, Nyamninia, Kod Bar Turo. Nyithindo duto e skundegi ibiro miyo chienib Odiechieng to manok ema ibiro kawo raqtind Pek mar dendgi. Yiero mar nyithindo mibiro pimo ibiro timo a poya mamiyo nyathi ka nyathi bedo gi thuolo mar bedo ni onyalo donjo e nonro ni.

(b) Jatim nonro kod ji ariyo moko mo konyre go biro timo limbe e dalani kend gibiro pejo min nyathi ka nyathi kata jaritne kuom chiemo kod math kod echiemo ma nyathi yudo to kod kind chiemo ka chiemo.

(c) Nyithindogi ibiro pimo pek gi, bor gi kod lach mar badgi ma malo. Pimgi ibiro kaw kinde ka kinde moyaw school mondo okawgo nonro mar dongogi. Mani ok nyalkelo rem moro amora e ngimagi.

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(d) Mondo Otim nonro mar kit chiemo manie dend jogo mimiyo nonroni, ibiro kaw rembgi matin madirom 5ml Rem matin sama igolo remo lal niyo remo mogolno ibiro nono ka dibedie kute mag malaria kata tin mar remo e del. Nyathini ibiro miyo chupe ariyo modhi kete lachne kod losruok maduong.

(e) Osenyisa ni an gi thuolo mar penjo duko mar pim ma otim kuom nyathi kendo ni duoko mar nonro ni duto ibiro ler e baraza ka nonro orumo.

(f) Ibiro miyi copy mar wechegi manyiso ni iyie donjo e nonroni mamari iwuon

Yie mar Nyathi e nonroni

An (Nying nyathi) ………………………………. Oseluonga mondo abed achiel kuom joma biro bedo e nonro mar dongruok mosewue malokano. Jalmochung ne weche mag chiemo maloso del to kod jonyuolna osenyisa/opimona matut tiend nonroni kendo a winjo.

Kuom ndiiko nyinga piny, aseyie mondo a bed achiel kuom jogo ma ibiro timo godo nonroni ma dongunok. Daher lero ni ok osechuno kata gi janyuolna kata jogo mochung ne nonroni mondo abedie nonroni.

………………………………………….   ………………………………….

Nying nyathi   Janeno mochung kende

(Ondik kende kaber)

Ng'ongruok Mar janyuol/Jarit mochung machik oyieg'o

Kuom keto seyi, an (nying janyuol kata jarit) …………………………….ayie

Mondo nyathina …………………………………………………………..ma en

Jahigni ……………………………mondo obed e nonro Mar "ranyisi motelo korkan tekre chi chiemo e del ma luwore gi chenro moket ne miyo nyithindo matindo chiemo e school"

Nyathina inyalo kwayo mondo owuogi enonro kapok ochopo e gikone ka jononro oneno ni owinjore kuom nyathina kata kadipo nyathina ok luw dwaro moket ni nonroni.

Seyi (kanye)…………………………………..Tarik…………………….2006

………………………………………….   …………………………..

Seyi mar janyuol/   Seyi mar janeno

Ngong'ruok mar ja nonro

An (nying)…………………………………………………….. I wacho: Aselero weche

mondik ka ni …………………………………………..Asejiwo jali/nyani mondo openj penjo

kendo akawo kindena mar duoko penjone. An kod chir ni koro en kod winjo makare kaluwore gi

nonro kaka osewuo malo kanyo. Ne atiyo/ok atiyo kod jalok dhok (ka jalok dhok otigo to jalok

nyaka ket seyi ka.

   Seyi (kanye)………………………………….. Tarik……………………

   ………………………………………….   …………………………………..

   Signature mar janonro   Signature mar   Janeno

COLUMBIA UNIVERSITY	
INSTITUTIONAL REVIEW BOARD	
IRB#:AAAC5773	Approval Date: 10/11/06
IRB Initials: _ALK_	Expiration Date: 10/10/07

Informed consent Swahili translation certificate

REPUBLIC OF KENYA

IN THE MATTER OF RESEARCH PROJECT BY PENINA KINYA MASIBO IN BAR SAURI VILLAGE

IN THE MATTER OF TRANSLATION OF PARTICIPANT INFORMATION LEAFLET AND CONSENT FORM FOR USE BY PARENTS/LEGAL GUARDIANS FROM ENGLISH TO SWAHILI
AND
IN THE MATTER OF OATHS AND STATUTORY DECLARATIONS ACT CAP 15 LAWS OF KENYA

A F F I D A V I T

I, **BARZIL MWAKULOMBA** of Post Office Box 3770-30100, Eldoret and a resident within the purlieus of Eldoret town within Uasin Gishu District, Rift Valley province, do hereby make this solemn Oath and state as follows:-

1. THAT I am the deponent herein, a Kenyan citizen and a holder of a Kenyan Identity Card No. 22647532.

2. THAT I am fully conversant with Swahili language both Oral and written; after having been born and raised in the Coastal region of the Republic of Kenya whereby Swahili dialect is widely spoken.

3. THAT I am also fully conversant with oral and written English Language hence am able to fully translate any written document from English to Swahili.

4. THAT I translated the above stated participant information leaflet and Consent form for use by parents/legal Guardians from English written language to written Swahili language.

5. THAT to the best of my understanding of both English dialect and Swahili dialect I have translated the aforestated document.

6. THAT what is deponed to herein above is true to the best of knowledge, information and belief.

SWORN at Eldoret this .... Day of 2006

BARZIL MWAKULOMBA   ]

BEFORE ME   ]

                                                                                   ]

COMMISSIONER FOR OATHS   ]

DRAWN BY:-

M/s. Gitonga & Co. Advocates,
P. O. Box 4599,
Eldoret.