PREVALENCE AND FACTORS ASSOCIATED WITH IRON DEFICIENCY AND INADEQUATE DIETARY ZINC INTAKE AMONG CHILDREN AGED 6 – 59 MONTHS AT MOI TEACHING AND REFERRAL HOSPITAL, ELDORET- KENYA

BY

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DECLARATIONS

Declaration by Student:

This thesis is my original work and has not been presented to any other university/institution for any academic award.

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DEDICATION

To professionals and persons in all fields of care preventing and treating micronutrient and macronutrient malnutrition

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ABSTRACT

Background: Micronutrients are chemical elements required in small quantities that are vital for normal growth and development. Micronutrient deficiency affects at least 2 billion people globally. In Kenya, Zinc (83%) and Iron deficiencies (35%) are most prevalent. Pregnant, lactating women and children less than 5 years of age are most affected.

Objectives: This study aims to estimate prevalence and determine factors associated with zinc deficiency, inadequate dietary zinc intake, iron deficiency, and iron deficiency anaemia among children 6 – 59 months treated at Moi Teaching and Referral Hospital (MTRH). It also describes the infant and young child feeding practices associated with these deficiencies.

Methods: This was a cross sectional study with a sample size of 354 participants recruited using systematic random sampling. Sociodemographic, clinical, laboratory and anthropometric data was collected. The laboratory tests included: serum iron, ferritin, total iron binding capacity and complete blood counts. We used 24-hour dietary recall to assess for adequacy of dietary zinc intake. Dietary diversity scores were assessed using the WHO food groups. Levels of stunting were used as a population indicator for zinc deficiency. Odds ratios were calculated at 95% confidence interval and p values < 0.05 were considered statistically significant. Univariate, bivariate and multivariate analyses were carried out on the categorical variables.

Results: The median age of the study participants was 31 months (IQR 15, 46) with a male majority (61%). The prevalence of inadequate zinc intake was 60% with a median age of 20 months (IQR 11, 48). Fifty percent (50%) of the study participants did not meet the minimum dietary diversity (MDD) score of at least four WHO food groups. Inadequate MDD was associated with inadequate dietary zinc intake (OR 3.1; CI 2.0 – 4.8; p<0.001). Twenty-six percent (26%) of the participants were stunted. Factors associated with increased odds of stunting included: Inadequate zinc intake (OR 1.5; CI 0.9 -2.6; p=0.09), Pre-term children (OR 3.1; CI 1.1 – 8.5; p=0.02), no prior deworming (OR 3.7; CI 2.2 – 6.1; p<0.001). There prevalence of iron deficiency was 77% (based on transferrin saturation levels) and 63% (based on serum ferritin levels). The factors associated with decreased the odds of iron deficiency (ID) included: Adequate MDD (OR 0.9; CI 0.6 – 1.9; p = 0.83) and deworming (OR 0.2; CI 0.1 – 0.5; p<0.001), exclusive breastfeeding for 6 months (OR 0.6; CI 0.1 – 2.7; p= 0.47). Factors associated with increased odds of iron deficiency included: pre-term birth (OR 3.7; CI 0.5 – 28.7; p=0.18), hypochromia (OR 3.8; CI 2.1 – 6.8; p<0.001), microcytosis (OR 1.4; CI 0.8 – 2.3; p = 0.23). The median age for iron deficiency anaemia (IDA) was 23 months (IQR 11, 43). Microcytosis (OR 2.5; CI 1.6 – 4.0; p<0.001) and hypochromia (OR 2.8; CI 1.5 – 5.5; p = 0.001) were associated with IDA.

Conclusions: Inadequate dietary zinc intake is still common. Iron deficiency and iron deficiency anaemia are still highly prevalent diseases of public health importance. Concurrent iron and zinc deficiency are common. Adequate minimum dietary diversity is necessary for prevention of iron and zinc deficiency

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LIST OF ABBREVIATIONS

AMPATH	Academic Model Providing Access to Healthcare
ANC	Antenatal Clinic
AOR	Adjusted Odds Ratio
BCG	Bacille of Calmente Guerin
CI	Confidence Interval
CRP	C-Reactive Protein
CWC	Child Welfare Clinic
DDS	Dietary Diversity Score
DHIS	District Health Information System
HAZ	Height-for-Age Z-score
HC	Head Circumference
HIV	Human Immunodeficiency Virus
ID	Iron Deficiency
IDA	Iron Deficiency Anaemia
IPV	Injectable Polio Vaccine
IQR	Inter-Quartile Range
IYCF	Infant and Young Child Feeding
MAM	Moderate Acute Malnutrition
MCH	Mean Corpuscular Haemoglobin
MCHC	Mean Corpuscular Haemoglobin Concentration
MCV	Mean Corpuscular Volume
MDD	Minimum Dietary Diversity
MND	Micronutrient Deficiency
MOH	Ministry of Health
MTRH	Moi Teaching and Referral Hospital
MUAC	Mid Upper-Arm Circumference

OPD	Out-Patient Department
OPV	Oral Polio Vaccine
OR	Odds Ratio
PCV	Pneumococcal Vaccine
Pg	Picogram
RBC	Red Blood Cell
RDW	Red cell distribution width
REC	Research Ethics Committee
ROTA	Rotavirus Vaccine
SAM	Severe Acute Malnutrition
SCC	Sick Child Clinic
SD	Standard Deviation
TIBC	Total Iron Binding Capacity
TSAT	Transferrin saturation
UIBC	Unsaturated Iron Binding Capacity
UNICEF	United Nations Children's Fund
WAZ	Weight-for-Age Z-score
WBC	White Blood Cell
WHO	World Health Organization
WHZ	Weight-for-Height Z-score
ZD	Zinc Deficiency
Z-score	Standard Score

OPERATIONAL DEFINITION OF TERMS

1. **Inadequate Dietary Zinc Intake**

   Defined as a mean daily zinc intake below the Estimated Average Requirement (EAR) for children aged 6–59 months, assessed using 24-hour dietary recall and analyzed using the Kenya Food Composition table 2018.

2. **Iron Deficiency**

   Defined as depleted body iron stores, indicated by low serum ferritin concentration or transferrin saturation level**,** after accounting for the presence of inflammation.

3. **Iron Deficiency Anaemia (IDA)**

   Defined as the co-existence of anaemia and iron deficiency in the same child.

4. **Stunting**

   Defined as chronic undernutrition**,** indicated by a length-for-age (children <24 months) or height-for-age (children ≥24 months) Z-score below −2 standard deviations (SD) from the median of the WHO Child Growth Standards**.** Severe stunting will be defined as LAZ/HAZ < −3 SD.

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background

Micronutrients are chemical elements required in small quantities that are vital for normal growth and development. Micronutrient deficiency (MND), also known as hidden hunger, is a form of under-nutrition and it affects normal body physiological functions, growth and development. Micronutrient deficiency disproportionately affects persons from limited resource settings such as countries in Sub-Saharan Africa, South Asia and some parts of South America. Some of the factors predisposing the persons in these countries to micronutrient deficiency include inadequate dietary intake, inadequate dietary diversity based on the World Health Organization food groups and low availability of animal-source foods. Other factors that contribute to these deficiencies include: food taboos and cultural practices limiting consumption of certain foods and inadequate nutritional counselling and education (Nancy F Krebs et al., 2014).

Globally, zinc and iron deficiency are still the most prevalent forms of micronutrient deficiency. Zinc and iron are important micronutrients required by the body for normal growth and development (Black, 1998; Branca & M., 2002; Thejpal, 2015).It is essential for cellular growth, immune system function, enzymatic functions at the cellular level and wound healing . Iron is vital for many functions such as energy production and cognitive function. However, iron is most important for adequate oxygen carrying capacity in blood. Zinc is important for the immune system and for proper functioning of the mucous membranes. After iron, zinc is the second most abundant trace element that is vital for life (Kambe et al., 2015).

Globally, micronutrient deficiency affects at least 2 billion persons worldwide. Among the children under 5 years; zinc, iodine, folate, vitamin A and iron are the most prevalent forms of micronutrient deficiencies. Iron deficiency is the most common micronutrient deficiency worldwide (Bailey et al., 2015; Ananda S Prasad, 2012) and it affects at least one third of world's population (Yadav & Chandra, 2011). Zinc deficiency is estimated to be responsible for at least 4% of all morbidity and mortality among children (Kambe et al., 2015). This proportion is much higher in developing countries. In developing countries, zinc deficiency accounts for 14.4% of deaths from diarrheal diseases, 10.4% of deaths due to malaria and 6.7% of pneumonia deaths among children aged 6 – 59 months (Shah, 2011).

Iron Deficiency (ID) and iron deficiency anaemia (IDA) is also associated with the developmental, motor, and behavioral changes in children (Aspuru et al., 2011). The cognitive and psychomotor impairment related to these deficiencies occurring during the period of brain developmental growth spurt in children less than 2 years of age may be irreversible in some cases (Yadav & Chandra, 2011).

Sub-Saharan Africa still bears a large brunt of the global burden of zinc and iron deficiency. Iron deficiency affects up to 60% of children under 5 years and up to 40% of pregnant women. This subsequently predisposes the pregnant women to anaemia, maternal mortality, premature deliveries and low birth babies. The premature babies subsequently are at risk of iron deficiency, and anaemia of prematurity hence continuing a vicious circle of iron deficiency. Some of the factors associated with these deficiency states in the Africa include; food security challenges, poverty and comorbidities such as Malaria and parasitic infections. Parasitic infections such as hookworms lead to intestinal loss of blood as well as impair absorption of micronutrients by causing local inflammation of the gut mucosa. Human immunodeficiency virus and subsequent acquired immunodeficiency syndrome have also been implicated in the development of iron deficiency.

The three most prevalent micronutrient deficiencies in Kenya are iron, zinc and iodine. Pregnant, lactating women and children less than 5 years of age are most affected by micronutrient deficiency. These micronutrient deficiencies affect as high as 83% of children under 5 years (Ministry of Health - Kenya, 2015; Ministry of Public Health and Sanitation - Kenya, 2012). As such, they are significant contributors to overall morbidity and mortality. Currently, the government through the Ministry of Health in Kenya aims at to reduce the prevalence of iron deficiency among newborns by targeting the pregnant and lactating women by giving them iron supplements from conception till six weeks post-partum. Beyond six weeks of age, there are no directed iron or zinc deficiency preventive measures for the children infants except through diet and fortified feeds.

The main factor associated with development of zinc deficiency is inadequate dietary intake. Zinc is not stored in the human body as such regular dietary intake from both animal and plant sources are necessary to maintain adequate serum levels (Gibson, 2012; Kenya, 2011; N F Krebs, 2013; Nancy F Krebs et al., 2014; Roohani et al., 2013). Therefore, the role of adequate dietary intake of foods rich in zinc and iron is pivotal in ensuring decrease in the prevalence of these micronutrients. Adequate dietary intake is however affected by low socioeconomic status that can affect a family’s food security.

Dietary diversity that involves daily consumption of various varieties of food groups leads to better nutrition outcomes. World Health Organization (WHO) recommends the following 7 food groups for use in assessment of dietary diversity: grains, roots and tubers; legumes and nuts; dairy products (milk, yogurt, cheese); flesh foods (meat, fish, poultry and liver/organ meats); eggs; vitamin-A rich foods; other fruits and vegetables.

For good nutrition outcomes, the minimum dietary diversity should include the consumption of at least 4 food groups(World Health Organization, 2008). Dietary diversity scores calculated using this WHO food grouping have been used for assessing risk of micronutrient deficiencies. Dietary diversity scoring utilizes 24-hour diet recall to minimize recall bias and uses this data as an approximation for dietary diversity (Arsenault et al., 2010; Gina et al., 2010; Habte & M., 2016; Hailemariam et al., 2018; Shah, 2011; Steyn et al., 2014).

Several interventions and advances have been put in place to address nutrition challenges mainly targeting adequate caloric intake and macronutrient deficiencies. However, zinc and iron deficiencies are still significant health concerns contributing to morbidity and mortality, impaired cognitive development, weakened immunity predisposing those affected to infections, and reduced economic productivity. These factors are further compounded by poor minimum dietary diversity, staple family foods that are low in micronutrients, low availability of animal-source foods and co-morbidities causing micronutrient malabsorption.

By applying the use of dietary diversity scoring combined with clinical and laboratory findings, we can be able to identify the children most at risk of these micronutrient deficiencies and institute secondary preventive measures to ensure better morbidity and mortality outcomes in this age group.

1.2 Problem Statement

Kenya, just like other developing countries, suffers a double burden of malnutrition for macro-and micronutrients. Kenya has put in several policies and mechanisms to decrease the burden of nutritional deficiencies. Some of the interventions include: nutritional education, micronutrient supplementation, food fortification and provision of nutritional services at health facilities. The leading micronutrient deficiencies includes; iodine, vitamin A, iron and zinc.

Despite these efforts, zinc and iron deficiencies are still at high levels such that the World Health Organization (WHO) considers them diseases that have high public health significance. It is estimated that nearly 50% of morbidity and mortality in children are attributable to malnutrition and micronutrient deficiencies. Among children aged 6 – 59 months, prevalence of iron deficiency (ID) ranges from 21.8% to 36.4% while zinc deficiency (ZD) is estimated at 83.3% (Ministry of Health - Kenya, 2015) . There is paucity of data regarding these micronutrient deficiencies among patients seen at Moi Teaching and referral hospital.

The health and socio-economic consequences of zinc and iron deficiency are profound. Some of the effects of zinc and iron deficiency can have irreversible and lifelong effects since they affect the formative stages of life. These effects lead to a vicious cycle of poor health, retarded growth and development, reduced cognitive development as well as diminished economic productivity.

Dietary factors are a major contributor for both iron and zinc deficiency. Inadequate intake of these micronutrients subsequently leads to their deficiency. Globally it is estimated that at least 17.3% of the population is at risk of inadequate dietary zinc intake based on availability of zinc in National food supplies. This estimate is much higher, up to 29.6%, in Sub-Saharan Africa and in developing countries. Inadequate zinc intake and can be used as a proxy for assessment of risk of zinc deficiency in a population (Abrams, 2021; Gupta et al., 2020b).

Lack of dietary diversity in Kenya poses a big challenge in the management of micronutrient deficiencies. Many Kenyan household rely on plant based staple diets such as maize, sorghum and rice that have a lower bioavailability of these micronutrients. Animal and flesh-based foods that are rich in both iron and zinc tend to be either unavailable or expensive for most low-income households.

Dietary intake and food security require multi-sectoral approaches and may therefore take a long time to fully address the factors leading to inadequate intake. This is further exacerbated by other factors such as climate change, drought and socio-economic challenges preventing access to nutrient-dense foods. As such, it could be necessary to include dietary diversity scoring as part of routine assessment of children presenting to hospitals in order to help in early identification of children at risk of zinc and iron deficiency. These children could subsequently be followed up more closely during child welfare clinic visits in order to promptly address any features of zinc and iron deficiency.

1.3 Justification

Zinc and iron deficiency among children aged 6 – 59 months still leads to unacceptably high morbidity and mortality in this age group. Some of the factors leading to these deficiencies can be easily addressed whereas others require multi-dimensional and multi-disciplinary approaches to properly intervene.

This study will adequately establish the magnitude of zinc and iron deficiency in this age group. In addition, it will also determine the associative factors with the aim of guiding policy on alleviation of the minimizing the burden caused by these deficiencies. Finally, it will explore the existence of concurrent zinc and iron deficiency that is common in this age group.

There is paucity of data regarding these deficiencies among this age group among patients treated at Moi Teaching and Referral Hospital. The available information by the Ministry of Health has national-level data on malnutrition. This data, and especially regarding micronutrient deficiency, is broad-based and does not capture local variations and contexts that may be brought about by factors such as: dietary habits, population mix (urban, rural and rural-urban) and food security. As such, it will generate data on the measures that can be put in place to mitigate the burden arising from these micronutrient deficiencies within this local context. Moi Teaching and Referral Hospital is the ideal site because it serves a large catchment population spanning more than 22 Counties. Therefore, a varied population mix will allow for a true picture of iron and zinc deficiency in the western region of Kenya.

In the end, this study shall lead to studies targeting supplementation of these micronutrients, adequacy of intake and dietary diversity among children in this age group.

1.4 Objectives

1.4.1 Primary Objective

- To describe the prevalence and factors associated with iron deficiency and inadequate dietary zinc intake among children aged 6 -59 months treated at Moi Teaching and Referral Hospital

1.4.2 Secondary Objectives

- To determine prevalence of iron deficiency, iron deficiency anaemia and inadequate dietary zinc intake among children aged 6 – 59 months treated at Moi Teaching and Referral Hospital
- To determine the risk of zinc deficiency among children aged 6 – 59 months treated at Moi Teaching and Referral Hospital
- To determine factors associated with iron deficiency, zinc deficiency and inadequate dietary zinc intake among children 6 – 59 months treated at Moi Teaching and Referral Hospital
- To describe dietary and feeding practices associated with iron deficiency and inadequate dietary zinc intake among children 6 – 59 months treated at Moi Teaching and Referral Hospital

1.5 Research questions

The research questions that this study seeks to answer are as follows:

- What is the prevalence of iron deficiency, iron deficiency anaemia and inadequate dietary zinc intake among children aged 6 – 59 months treated at Moi Teaching and Referral Hospital?
- What is the risk of zinc deficiency among children aged 6 – 59 months treated at Moi Teaching and Referral Hospital?
- What factors are associated with iron deficiency, zinc deficiency and inadequate dietary zinc intake among children 6 – 59 months treated at Moi Teaching and Referral Hospital?
- What are the dietary and feeding practices associated with iron deficiency and inadequate dietary zinc intake among children 6 – 59 months treated at Moi Teaching and Referral Hospital?

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Epidemiology of Zinc and Iron deficiency

Micronutrients are essentially minerals and vitamins required in small quantities for the normal physiological functioning of human bodies. Micronutrient deficiency (MND) or hidden hunger is a form of under-nutrition. The most prevalent forms of micronutrient deficiencies are zinc, iodine, folate, vitamin A and iron with children under 5 years and pregnant women being disproportionately affected. Iron deficiency is the most common micronutrient deficiency worldwide. However, more often than not, micronutrient deficiency occurs concurrently. Global estimates are that micronutrient deficiency affect at least 2 billion persons worldwide (Bailey et al., 2015; Ananda S Prasad, 2012)

Iron and zinc deficiency are the most widespread and ubiquitous globally. These trace elements play crucial physiological roles in growth and development especially, in growing children and women of reproductive age group. Their deficiencies have far reaching consequences in morbidity and mortality. They have also been associated with weakened immunity and impaired cognitive development.

Micronutrient deficiency globally affects persons from limited resource settings more due to higher inadequate dietary intake (Nancy F Krebs et al., 2014). Iron deficiency is the most common micronutrient deficiency affecting at least one third of world's population (Yadav & Chandra, 2011). Geographically, Africa and South East Asia are the most affected by iron deficiency. According to the World Health Organization (WHO), the incidence of iron deficiency is 2 -5 times higher than that of iron deficiency anaemia in developing countries due to reasons such as malnutrition, infections and low dietary intake (Aspuru et al., 2011).

2.2 Zinc and Iron deficiency in Kenya

Kenya, like many other developing countries, is grappling with a double burden of malnutrition, which includes both undernutrition and the rising prevalence of overnutrition-related conditions, as well as the persistent issue of hidden hunger—a form of malnutrition caused by a lack of essential vitamins and minerals, also known as micronutrient deficiencies. These deficiencies are often less visible than acute hunger but can have long-term consequences on physical and cognitive development, particularly among children and women of reproductive age. According to the Ministry of Health in Kenya (2015), nearly 50% of all morbidity and mortality in children under the age of five can be attributed to malnutrition, with deficiencies in crucial micronutrients such as iodine, vitamin A, iron, and zinc being of particular public health significance.

Among these micronutrients, iron and zinc deficiencies are especially widespread and impactful. Iron deficiency is a leading cause of anaemia, which compromises oxygen transport in the blood and can result in fatigue, weakened immunity, and poor cognitive performance. In Kenyan children aged 6 to 59 months, iron deficiency rates range from 21.8% to 36.4%, indicating a serious health concern that affects a significant portion of the paediatric population. Zinc, another vital micronutrient, supports immune function, cell division, and wound healing. Its deficiency is associated with increased susceptibility to infections such as diarrhea and pneumonia, both of which contribute to high child mortality rates in the region. Alarmingly, zinc deficiency in Kenya is estimated at 83.3% among young children, pointing to an urgent need for public health interventions(Ministry of Health - Kenya, 2015) .

One of the primary contributors to these deficiencies is dietary insufficiency. In Kenya, as in many other parts of sub-Saharan Africa, staple foods consist largely of grains, cereals, and legumes, such as maize, beans, and rice. While these foods provide essential calories and some nutrients, they are high in phytates, natural compounds that inhibit the absorption of both iron and zinc in the digestive tract. Moreover, these plant-based staples do not provide the same level of bioavailable micronutrients as animal-based foods.

Meat, fish, and poultry foods that are naturally rich in highly absorbable forms of iron and zinc are consumed infrequently, especially in low-income households. This is largely due to socioeconomic constraints. Many families cannot afford to purchase meat products regularly, resulting in a diet that may meet caloric needs but falls short in terms of nutritional quality. As a result, children and women, who have higher nutritional requirements, are disproportionately affected (Cai et al., 2015; Ministry of Health - Kenya, 2015).

Addressing this issue requires a multifaceted approach, including improving dietary diversity, promoting nutrition education, and enhancing access to fortified foods and supplementation programs. Interventions such as school feeding programs, micronutrient powders for infants and toddlers, and food fortification policies could significantly reduce the burden of micronutrient deficiencies. Without targeted action, the long-term effects of iron and zinc deficiency will continue to hinder national development by compromising the health, cognitive ability, and productivity of future generations.

2.3 Etiology of Zinc and Iron deficiency

Micronutrient deficiencies, especially those involving zinc and iron, remain significant public health concerns globally, particularly in low- and middle-income countries like Kenya. Understanding the etiology, or root causes, of these deficiencies is essential for designing targeted interventions and preventive strategies.

While multiple biological, dietary, and socio-environmental factors contribute to the development of micronutrient deficiencies, inadequate dietary intake remains the most consistent and modifiable cause. However, underlying health conditions, life stages that demand higher nutrient intake, and cultural or economic limitations also play significant roles.

2.3.1 Zinc deficiency

Zinc deficiency arises primarily due to insufficient dietary intake, making it one of the most common micronutrient deficiencies worldwide, particularly in populations that rely heavily on cereal-based diets. Unlike some other nutrients, zinc is not stored in significant amounts in the human body, which means that regular and adequate consumption from dietary sources is essential to maintain optimal serum zinc levels (Gibson, 2012; Nancy F Krebs et al., 2014; Ministry of Health - Kenya, 2015; Roohani et al., 2013). This continual requirement makes the body highly sensitive to fluctuations in intake, especially in populations with limited access to diverse foods.

In most Kenyan households, the diet is predominantly plant-based, relying heavily on maize, legumes, and other staples. While these foods provide energy and basic nutrition, they also contain phytates, compounds that bind to zinc in the gastrointestinal tract and inhibit its absorption. The high phytate content in these foods significantly reduces zinc bioavailability, making it harder for the body to absorb enough zinc even when it is present in the diet. Moreover, the zinc found in plant sources is inherently less bioavailable than that in animal-based foods, further complicating the issue.

Zinc requirements also increase during certain physiological states, such as growth spurts in infants and adolescents, periods of illness, and during pregnancy and lactation. One common cause of zinc loss is diarrhea, which is both a symptom and a contributor to zinc deficiency. During diarrheal episodes, the body loses significant amounts of zinc through the gastrointestinal tract, further reducing serum levels (Gibson, 2012). This creates a vicious cycle: zinc deficiency weakens the immune system, increasing the frequency and severity of infections, which in turn depletes zinc stores even more rapidly.

Additionally, conditions that impair gastrointestinal function can reduce the body's ability to absorb zinc effectively. These may include chronic inflammatory diseases, intestinal infections, or malabsorption syndromes. Excessive loss of zinc through other routes, such as through urine or sweat, may also contribute to deficiency, especially in individuals who are physically active in hot climates or have chronic kidney conditions.

In neonates and infants, maternal zinc status plays a critical role. Zinc is transferred to the foetus during the third trimester of pregnancy, making preterm infants especially vulnerable to deficiency due to shortened gestation and reduced maternal-foetal nutrient transfer (Nancy F Krebs et al., 2014). Similarly, low birth weight and maternal malnutrition further increase the risk. For exclusively breastfed infants, if the lactating mother is zinc-deficient, the zinc content in breast milk may be insufficient to meet the child's requirements, particularly after the first few months of life.

The absorption of zinc in the small intestine is also a complex process, influenced by numerous factors. Zinc is excreted along with unabsorbed dietary matter, making net absorption difficult to estimate accurately. Moreover, zinc is absorbed more effectively in liquid form than in solid foods, particularly those high in fibre or phytate content. The average zinc absorption rate is estimated at about 33%, although this can vary significantly based on the population group, dietary patterns, and overall nutritional status (Roohani et al., 2013). This variability underscores the need for tailored dietary guidelines that account for regional food practices and availability.

2.3.2 Iron deficiency

Iron deficiency, another critical micronutrient issue, has a multifactorial etiology. It results from three main pathways: inadequate dietary intake, increased physiological demands, and excessive loss of iron from the body. Of these, the most prevalent cause globally especially in resource-limited settings is insufficient intake of iron-rich foods (Aspuru et al., 2011; N F Krebs, 2013; Ministry of Health - Kenya, 2015).

Many populations, particularly in rural Kenya, consume diets low in iron-rich foods like red meat, organ meats, and fortified cereals. The iron content of common staple foods is relatively low, and much of it is in the non-heme form, which is poorly absorbed compared to heme iron found in animal sources. As with zinc, high phytate levels in the diet reduce iron absorption, and the lack of enhancers like vitamin C further exacerbates this issue (Ministry of Public Health and Sanitation - Kenya, 2012).

Increased physiological demands during certain life stages also contribute significantly to iron deficiency. Children, adolescents undergoing rapid growth, pregnant women, and menstruating women all have higher iron requirements. In children, the combination of rapid growth and low dietary intake makes them particularly vulnerable. During periods of growth, the body's need for iron increases to support the expansion of blood volume, muscle mass, and overall development. Without sufficient intake, iron stores are quickly depleted, leading to anemia and other health complications (Aspuru et al., 2011).

Health conditions and environmental factors can also lead to excessive iron losses. These include chronic gastrointestinal disorders, such as ulcers, inflammatory bowel disease, or infections that cause blood loss. In many parts of Kenya, parasitic infections, such as hookworm and schistosomiasis, are common and result in chronic intestinal blood loss, thereby increasing the risk of iron deficiency anemia. Additionally, helminthic infections are a major contributor to blood and nutrient loss in children, especially in rural areas with limited sanitation and access to clean water (Ministry of Health - Kenya, 2015; Ministry of Public Health and Sanitation - Kenya, 2012).

Decreased gastric acidity, whether due to chronic illness or the use of medications like proton pump inhibitors, can reduce the solubility of iron and impair its absorption. Furthermore, intravascular hemolysis, a condition in which red blood cells are destroyed prematurely, can lead to rapid depletion of iron stores. While less common, genetic disorders and some chronic diseases can also impair the body’s ability to recycle or absorb iron efficiently.

Overall, iron deficiency in children in developing countries like Kenya is most often a result of dietary insufficiency coupled with infections and high physiological demands. This presents a serious public health challenge, as iron deficiency can impair cognitive and physical development, reduce academic performance, and compromise immune function, thereby increasing the risk of illness and mortality (Ministry of Public Health and Sanitation - Kenya, 2012; Muleviciene et al., 2018).

2.4 Dietary sources of Iron and Zinc

Micronutrients such as iron and zinc are essential for human health, especially in vulnerable populations like infants, children, pregnant women, and lactating mothers. These nutrients play vital roles in immune function, growth and development, cognitive function, and overall metabolic activity. Since the body cannot synthesize these micronutrients, they must be obtained through dietary sources. Inadequate intake, poor absorption, and lack of dietary diversity are major contributors to iron and zinc deficiencies, particularly in resource-limited settings such as rural parts of Kenya.

Understanding the dietary sources of iron and zinc, as well as the factors that affect their absorption and bioavailability, is crucial for addressing micronutrient deficiencies in Kenya and similar settings. While breast milk provides a foundational source of these nutrients during the early months of life, it becomes insufficient as infants grow and their nutritional needs increase. The reliance on plant-based diets in resource-poor settings further compounds the risk of deficiency due to poor bioavailability associated with phytates and lack of dietary diversity.

Efforts to prevent and reduce iron and zinc deficiencies must include improving maternal nutrition, promoting appropriate complementary feeding, and increasing access to nutrient-dense foods, particularly those of animal origin. In settings where food diversity is limited, supplementation and food fortification strategies can serve as effective short-term interventions. However, long-term solutions must focus on sustainable food systems, economic empowerment, and nutrition education to ensure that all individuals, especially the most vulnerable, can access and utilize the nutrients necessary for healthy growth and development.

2.4.1 Iron

Iron is a vital component of hemoglobin and myoglobin, playing a crucial role in oxygen transport and cellular respiration. During fetal development, iron is primarily accreted by the fetus during the last trimester of pregnancy. Consequently, preterm infants are at a higher risk of iron deficiency due to their reduced gestational iron stores. These infants may also develop anemia of prematurity, a condition often linked to low iron status and the immaturity of erythropoiesis (Cai et al., 2015).

For infants under six months, breast milk is the sole source of iron if they are exclusively breastfed. While breast milk contains relatively low iron concentrations compared to other sources, the bioavailability of iron in breast milk is remarkably high, meaning the small amount present is efficiently absorbed by the infant. The concentration of iron in breast milk is highest in colostrum, the early milk produced in the first few days after birth. As lactation progresses, the iron concentration declines steadily. Reported iron levels in breast milk typically range between 0.27–0.90 mg/L, with the highest levels found shortly after delivery (Cai et al., 2015).

Interestingly, maternal iron intake and iron status have little influence on the concentration of iron in breast milk. This suggests that, unlike some other micronutrients, iron levels in human milk are tightly regulated, regardless of the mother's nutritional status. However, this does not reduce the importance of ensuring good maternal nutrition, as iron-deficient mothers may experience fatigue, impaired immunity, and complications during delivery.

Beyond infancy, dietary sources become crucial in meeting iron needs, particularly after six months of age when breast milk alone can no longer supply adequate iron. Iron in food exists in two forms: heme iron and non-heme iron. Heme iron is found in animal products, including red meat, poultry, liver, and seafood. It is readily absorbed, with an average bioavailability of 20% to 30%. In contrast, non-heme iron, which is present in plant-based foods like legumes, grains, vegetables, and fortified cereals, has significantly lower bioavailability typically less than 10% (Aspuru et al., 2011).

The absorption of non-heme iron is highly influenced by gastric acidity and dietary components. For example, vitamin C (ascorbic acid) enhances non-heme iron absorption by converting it to a more soluble form. Consuming non-heme iron together with heme iron-rich foods also improves its uptake. On the other hand, several dietary inhibitors can reduce absorption. These include calcium, polyphenols, and phytates compounds found in whole grains, legumes, and certain vegetables. Phytates are particularly concerning in plant-based diets prevalent in low-income communities, as they bind iron and prevent its absorption in the gut.

Given that many populations in rural Kenya rely heavily on cereals, roots, and legumes as dietary staples, the prevalence of non-heme iron sources in the diet, combined with the high phytate content, makes iron deficiency a persistent and widespread issue. Addressing this challenge requires promoting dietary diversity and the inclusion of iron-rich and iron-enhancing foods in complementary feeding, especially for young children transitioning from exclusive breastfeeding.

2.4.2 Zinc

Zinc is another essential trace element required for immune competence, enzyme function, growth, and cellular repair. Similar to iron, zinc is transferred from the mother to the foetus primarily during the third trimester of pregnancy. Therefore, preterm infants are at increased risk for zinc deficiency because of their limited prenatal accumulation of this micronutrient. In addition, maternal nutritional status particularly deficiencies in zinc and other micronutrients can negatively impact foetal stores. These issues are often addressed through micronutrient supplementation during pregnancy, which is a critical intervention to improve both maternal and neonatal health outcomes (N F Krebs, 2013; Nancy F Krebs et al., 2014).

In the early months of life, breast milk is the primary source of zinc for exclusively breastfed infants. Zinc concentration in breast milk is highest during the early postpartum period (also known as the peuperium), with levels typically exceeding 3 mg/L. However, zinc content declines rapidly over time, and by six months postpartum, average levels drop to less than 1 mg/L. Because of this decline, complementary feeding that includes zinc-rich foods is essential after six months to meet the growing infant’s nutritional needs (Nancy F Krebs et al., 2014).

The bioavailability of zinc in the diet is largely determined by the type of food consumed and the presence of dietary enhancers or inhibitors. Research has shown that zinc absorption is relatively efficient up to 92% from low-zinc diets and around 81% from high-zinc diets, although this efficiency can vary depending on the overall diet composition (Roohani et al., 2013). Animal-based foods such as meat, poultry, seafood (particularly oysters), and organ meats are excellent sources of highly bioavailable zinc. These foods contain zinc in forms that are easily absorbed by the intestinal lining.

In contrast, plant-based sources, including legumes, cereals, and some vegetables, contain zinc in less absorbable forms due to the presence of phytates, which bind zinc and inhibit its uptake. This challenge is particularly pronounced in low-resource settings where access to animal-based foods is limited by economic constraints or cultural dietary practices. In many Kenyan households, especially in rural areas, animal-source foods are consumed infrequently due to high cost or low availability. This creates a nutritional gap that increases the likelihood of zinc deficiency, especially among infants and young children during the critical weaning and early childhood periods (Ministry of Health - Kenya, 2015).

To mitigate this risk, dietary diversity becomes a key strategy. Ensuring that children receive a mix of foods from different food groups including animal products, legumes, whole grains, fruits, and vegetables can improve overall zinc intake and reduce the impact of phytate interference. In public health programs, zinc supplementation and fortification of staple foods have been implemented in some countries to address population-level zinc deficiencies, though their reach and effectiveness vary.

In addition to dietary strategies, public health education is necessary to inform caregivers about the importance of introducing nutrient-rich foods during the complementary feeding period. Many mothers are unaware of which foods provide essential micronutrients or how to combine foods to enhance nutrient absorption. Behaviour change communication through community health workers and maternal-child health clinics can play a significant role in improving nutrition practices at the household level.

2.5 Effects of zinc deficiency

Zinc is a vital trace element essential for a wide range of physiological, cellular, and biochemical functions within the human body. It plays a critical role in enzymatic activity, cell signaling, protein and DNA synthesis, gene expression, and cell division. Due to its involvement in such a diverse array of biological processes, zinc is indispensable for normal human growth, development, immune function, wound healing, and tissue repair. As such, deficiency of zinc can lead to profound and wide-ranging clinical effects, particularly in vulnerable populations such as children, pregnant women, and individuals with chronic illness (Nancy F Krebs et al., 2014; Maggini et al., 2010; Ananda S Prasad, 2012).

One of the most well-documented consequences of zinc deficiency is growth retardation, especially in children. Zinc is essential for cellular proliferation and the synthesis of nucleic acids and proteins, all of which are vital for proper physical growth. Inadequate zinc impairs these processes, resulting in stunted growth, which is a common manifestation of chronic zinc deficiency. Children experiencing stunting are not only shorter than their peers, but often suffer from delayed sexual maturation and overall developmental delays. Stunting is also associated with long-term adverse outcomes, including poor educational performance, reduced economic productivity in adulthood, and increased risk of chronic diseases (Abdollahi et al., 2019; S Bening et al., 2017).

In males, zinc deficiency has been linked to hypogonadism, a condition characterized by decreased function of the gonads. This condition results in reduced testosterone levels, delayed puberty, and in severe cases, impotence and infertility. Zinc is essential for the synthesis of testosterone and the normal functioning of the male reproductive system. Chronic zinc deficiency, especially if it begins early in life, can therefore have lasting consequences on reproductive health and sexual development.

Zinc also plays a pivotal role in maintaining a healthy immune system, particularly in supporting cell-mediated immunity. It contributes to the function and proliferation of T-lymphocytes, neutrophils, and natural killer cells. A deficiency in zinc compromises the immune response, making individuals more susceptible to a variety of infections. This is especially critical in children, as a weak immune response can lead to recurrent and prolonged infections, including respiratory tract infections, pneumonia, and gastrointestinal illnesses (Maggini et al., 2010; Ananda S Prasad, 2012). This immunosuppressive effect of zinc deficiency creates a vicious cycle, as repeated infections can further deplete zinc levels through loss in bodily fluids and reduced dietary intake due to illness-induced anorexia.

In addition to compromising immune function, zinc deficiency has been linked to cognitive impairment and neurosensory dysfunction. Zinc is involved in neurogenesis, neuronal migration, and synaptic plasticity all vital for proper brain function. Animal and human studies have indicated that low zinc levels are associated with learning disabilities, memory impairment, emotional instability, and even mood disorders. In children, this can present as poor school performance, reduced attention span, and learning difficulties, potentially undermining educational outcomes and future opportunities (Maggini et al., 2010).

Another important role of zinc is in maintaining the integrity and function of the gastrointestinal (GI) tract. Zinc supports the repair of intestinal mucosa and helps maintain the tight junctions between epithelial cells. Deficiency in zinc increases intestinal permeability, often referred to as a "leaky gut," making it easier for pathogens to cross into the bloodstream and cause systemic infections. This impaired GI integrity also contributes to diarrheal illnesses, a major concern in children in developing countries. Numerous clinical studies have shown that children with zinc deficiency have a higher incidence and longer duration of diarrhea. Moreover, supplementation with zinc during episodes of diarrhea significantly reduces the severity and recurrence of symptoms (Nancy F Krebs et al., 2014). As a result, the World Health Organization (WHO) recommends zinc supplementation as part of the treatment for acute diarrhea in children.

Dermatological manifestations of zinc deficiency are often among the earliest visible signs, especially in moderate to severe deficiency states. These can include alopecia (hair loss) and characteristic dermatitis, which typically affects the perioral (around the mouth), perineal (around the genitals), and acral (extremities such as hands and feet) regions. The dermatitis often presents as erythematous, scaly, and crusted lesions, which can sometimes be mistaken for other dermatological conditions such as eczema or fungal infections (Corbo & L., 2013). These skin manifestations not only indicate nutritional deficiency but can also serve as entry points for secondary infections, further complicating the clinical picture.

Another common and often overlooked symptom of zinc deficiency is delayed wound healing. Zinc is crucial for all phases of the wound-healing process, including inflammation, cell proliferation, and tissue remodeling. It plays a role in collagen synthesis and cell membrane stabilization. In zinc-deficient individuals, even minor cuts and abrasions may take significantly longer to heal, increasing the risk of infection and chronic wound development (Cole et al., 2010). This is particularly problematic in individuals with comorbid conditions such as diabetes or malnutrition, where wound healing is already compromised.

In severe or prolonged cases, zinc deficiency can manifest as taste abnormalities (hypogeusia or dysgeusia), night blindness, and impaired appetite. These symptoms not only affect the quality of life but can further reduce food intake, thereby exacerbating the deficiency. Appetite suppression is particularly dangerous in children, as it leads to reduced nutrient intake at a time when the body requires increased energy and micronutrients for growth and development.

Zinc deficiency also interacts with other micronutrient deficiencies, notably iron and vitamin A. For example, zinc is required for the synthesis of retinol-binding protein, which is essential for the transport of vitamin A in the blood. Consequently, zinc deficiency may worsen vitamin A deficiency, leading to visual disturbances and a higher risk of infections.

From a public health perspective, the consequences of zinc deficiency extend beyond individual health. In populations where zinc deficiency is prevalent, such as many regions in sub-Saharan Africa, including Kenya, the burden of disease is disproportionately high. This includes higher child mortality rates, increased incidence of infectious diseases, and impaired cognitive development at a population level. These outcomes ultimately contribute to the intergenerational cycle of poverty and underdevelopment.

Given the above listed effects, zinc deficiency, therefore, represents a significant public health challenge, particularly in developing countries. Addressing it requires a comprehensive approach, including improving maternal and child nutrition, promoting dietary diversification, supplementing at-risk populations, and strengthening healthcare systems to detect and manage deficiency symptoms early. Only through a multi-sectoral strategy can the burden of zinc deficiency and its cascading health effects be meaningfully reduced.

2.6 Effects of iron deficiency in children

The most widely recognized and clinically significant manifestation of iron deficiency is the development of iron deficiency anaemia (IDA). This condition occurs when iron levels in the body are insufficient to support the production of healthy red blood cells, which are essential for transporting oxygen throughout the body. In the absence of adequate iron, the bone marrow produces smaller and less haemoglobin-rich red blood cells, a condition termed microcytic hypochromic anaemia. The resulting anaemia leads to a decrease in oxygen delivery to tissues, which triggers a wide array of physiological symptoms and systemic consequences (World Health Organization & Chan, 2011).

The classic symptoms of iron deficiency anaemia include fatigue, general weakness, pallor, dizziness, and headaches. These symptoms stem from the body's reduced capacity to carry and deliver oxygen efficiently, which affects cellular metabolism and energy production. Exertional dyspnoea shortness of breath during physical activity is another common complaint, as the body attempts to compensate for decreased oxygen availability by increasing respiratory rate and cardiac output. In more severe or prolonged cases, IDA can lead to tachycardia, palpitations, and even congestive heart failure, particularly in individuals with pre-existing cardiovascular compromise. The body works harder to circulate the limited oxygen it receives, which places excess strain on the heart over time (Kumar et al., 2022).

Beyond its haematological effects, iron deficiency has far-reaching implications, especially for infants, young children, and pregnant women. In children, iron plays a vital role not just in haemoglobin production but also in neurological development, myelination, neurotransmitter synthesis, and brain energy metabolism. Iron deficiency during critical periods of brain development—especially the first two years of life—can have lasting and potentially irreversible effects on cognitive and psychomotor development. Studies have consistently shown that children who experience iron deficiency early in life may display delayed language acquisition, poor attention span, decreased social engagement, and lower IQ scores compared to their well-nourished peers (Yadav & Chandra, 2011).

In fact, the timing and severity of iron deficiency are key determinants of long-term neurodevelopmental outcomes. When iron deficiency occurs during the “brain growth spurt” a period from late foetal life to around 24 months of age it can disrupt the formation and maturation of essential brain structures. Iron is crucial for the development of the hippocampus and prefrontal cortex, areas responsible for memory, learning, and behaviour regulation. Iron-deficient children often struggle with school readiness, academic achievement, and social interaction, setting the stage for a cycle of disadvantage that extends into adolescence and adulthood (Zheng et al., 2021).

In addition to its impact on cognition, motor development is also adversely affected by iron deficiency. Children may exhibit delayed gross and fine motor milestones, reduced muscle tone, and diminished physical endurance. These developmental delays can impact a child’s ability to explore their environment and interact with others, further compounding cognitive and emotional delays. Even after iron supplementation, some of the deficits observed in early childhood may persist, especially if the deficiency was severe or prolonged. This highlights the urgent need for early detection and prevention of iron deficiency in infancy and early childhood (Thalanjeri et al., 2016).

Behaviourally, children with iron deficiency anaemia may show signs of irritability, anxiety, inattentiveness, and reduced responsiveness to stimulation. These symptoms not only affect the child's learning capacity but also influence caregiver-child interaction, which is a critical component of healthy emotional and psychological development. Children may also exhibit pica, a condition characterized by the craving and consumption of non-food substances like soil, chalk, or ice potentially leading to further health complications such as parasitic infections or heavy metal poisoning (Pivina et al., 2019).

In adolescents and adults, particularly women of reproductive age, iron deficiency contributes significantly to decreased productivity, poor work performance, and absenteeism. Women may experience exacerbated symptoms during menstruation due to additional blood and iron loss. In pregnant women, iron deficiency increases the risk of preterm delivery, low birth weight infants, and maternal mortality. In some cases, IDA can also lead to increased risk of postpartum depression, further influencing maternal-infant bonding and early child development.

Iron deficiency also affects immune function, making individuals more susceptible to infections. It impairs the activity of neutrophils and lymphocytes, reduces cytokine production, and compromises the skin and mucosal barriers that serve as the body's first line of defence (Aspuru et al., 2011). This is particularly dangerous in settings where infectious diseases like malaria, tuberculosis, and parasitic infections are prevalent, such as many regions in sub-Saharan Africa, including Kenya.

From a public health perspective, the consequences of widespread iron deficiency are profound. It is estimated that iron deficiency anaemia accounts for a significant portion of the global burden of disease, contributing to increased mortality and morbidity, particularly among women and young children. The loss of cognitive potential, educational attainment, and workforce productivity due to iron deficiency represents a substantial economic cost for countries already struggling with poverty and underdevelopment (Özdemir, 2015; Woldie et al., 2015).

Preventive strategies are therefore essential and must be multi-faceted. These include promoting maternal iron supplementation during pregnancy, ensuring timely introduction of iron-rich complementary foods, and implementing food fortification programs for staple foods such as flour and cereals. In regions with high prevalence of parasitic infections, periodic deworming, improved water and sanitation access, and public health education are critical to reduce the burden of disease that contributes to or exacerbates iron loss.

Moreover, health systems must be equipped to identify and manage iron deficiency early. Screening programs in maternal and child health clinics, training of health workers in nutrition assessment, and community-based interventions can help detect at-risk populations before deficiency becomes severe or irreversible. Iron supplementation or therapeutic interventions should be combined with dietary counselling to ensure lasting behaviour change and adherence.

Given the above listed effects, iron deficiency and its most prominent clinical outcome iron deficiency anaemia are more than just haematological concerns. They represent a complex interplay of nutritional, developmental, behavioural, and public health challenges. Addressing iron deficiency requires not only medical treatment but also sustained, community-based efforts to improve nutrition, education, and access to healthcare. Given its profound and long-lasting impact, particularly during early childhood, iron deficiency should be a priority in national and international health agendas aimed at improving population well-being and promoting human development.

2.7 Diagnosis of zinc deficiency

In population-based studies, zinc deficiency is commonly assessed using three recommended indicators: serum zinc concentration, prevalence of inadequate dietary zinc intake, and the prevalence of stunting, defined as a height-for-age Z-score less than -2 standard deviations based on WHO growth standards for children under five years (Gupta et al., 2020a; Harika et al., 2017; Vuralli et al., 2017).

These indicators provide a comprehensive approach to understanding zinc deficiency at the population level, as each reflects different aspects of nutritional status and health. Specific thresholds for identifying populations at risk include a stunting prevalence greater than 20%, serum zinc levels below the recommended cut-off in more than 20% of individuals, and inadequate zinc intake affecting more than 25% of the population (Hess, 2017b). These benchmarks are essential for public health planning, guiding targeted interventions such as dietary diversification, supplementation, or food fortification programs to improve zinc status in vulnerable groups.

Currently, there are no simple or universally accepted markers to detect marginal, mild, or moderate zinc deficiency, which complicates diagnosis and population-level assessments. Among available tools, serum zinc concentration remains the most reliable and widely used biomarker for evaluating zinc status in large-scale nutritional studies (Roohani et al., 2013). Zinc deficiency (ZD) is typically defined as serum zinc levels below 70 µg/dL (or 10.7 µmol/L), a threshold associated with increased risk of adverse health outcomes (Cole et al., 2010). However, normal reference ranges vary across studies, with some suggesting values between 64–124 µg/dL as a typical range in healthy individuals (Lin et al., 2012). While serum zinc provides valuable insights into current zinc status, it is influenced by factors like inflammation, time of day, and recent dietary intake. As a result, interpretation of results should consider additional indicators such as dietary assessments, clinical signs, and anthropometric measures for a more comprehensive understanding.

For population-based studies, serum zinc concentration remains the most commonly used biomarker for assessing zinc status despite its known limitations. The recommended lower cut-off values for serum zinc levels vary depending on the time of day the sample is collected and whether the individual has eaten. Among children under 10 years of age, the suggested thresholds are less than 65 µg/dL for morning non-fasting samples and less than 57 µg/dL for afternoon samples (Roohani et al., 2013). These thresholds are based on the natural circadian rhythm of serum zinc, which causes significant fluctuations up to 20% within a 24-hour period. Typically, zinc levels are highest in the morning and decline throughout the day. Additionally, serum zinc concentrations are known to rise following food intake, which can further influence the accuracy and consistency of results.

Given these fluctuations, relying solely on serum zinc levels to determine deficiency can be problematic. Nonetheless, due to the lack of more practical and cost-effective alternatives, serum zinc measurement remains the primary biomarker used in most nutritional surveillance programs. However, its interpretation should be made cautiously and in conjunction with other clinical and contextual factors. These include the presence of clinical signs such as growth failure, dermatological symptoms, or poor wound healing, as well as a thorough review of the individual's dietary habits and anthropometric indicators like stunting (Cole et al., 2010; Roohani et al., 2013; Willoughby & N., 2014).

It is important to understand the distribution and function of zinc in the body to appreciate the limitations of serum measurements. Zinc is not stored in a single, readily mobilizable reserve in the body. Instead, approximately 60% of total body zinc is stored in skeletal muscle, about 30% in bone, around 5% in the liver and skin, and the remaining 2–3% is distributed in other tissues and organs. Notably, serum zinc constitutes only about 0.1% of the body's total zinc content. Around 70–80% of this circulating zinc is bound to albumin, serving as a transport mechanism. In times of need, zinc can be released and delivered to tissues that require it, but this dynamic also means serum levels can shift quickly in response to acute physiological changes.

Low serum zinc levels are also not a definitive manifestation of zinc deficiency. It is a state that can result from normal physiological changes but can also occur in conditions such as: acute infections and inflammation, stress, myocardial infarction and in conditions that lead to hypo-albuminemia such as liver cirrhosis and protein energy malnutrition. Similarly, haemolysis increases serum zinc levels (A S Prasad, 2013; Roohani et al., 2013).

Stunting, height-for-age Z score of less than -2SD, is an indicator for risk of zinc deficiency. This is because stunting in children under 5 years has been associated with inadequate zinc intake and zinc deficiency (Salsa Bening et al., 2017). Stunting is not pathognomonic for zinc deficiency due to the many other causal factors associated with it as an indicator of chronic malnutrition. However, dietary zinc supplementation has been shown to improve growth outcomes such as stunting (Liu et al., 2018).

2.8 Diagnosis of iron deficiency

Iron deficiency (ID) is refers to a drop in total iron levels both in serum and in body iron stores. The gold standard for the diagnosis of iron deficiency is to conduct a bone marrow aspirate and Prussian blue staining for ferric iron to assess the bone marrow iron stores. However, due to its invasive nature, high cost, and the need for specialized expertise, this test is not routinely performed in clinical practice. Consequently, clinicians rely on a combination of laboratory tests and clinical assessments to diagnose ID in a non-invasive and cost-effective manner (Baker & G., 2010).

In cases where iron deficiency progresses to iron deficiency anaemia (IDA), the diagnosis of anaemia is typically made based on the World Health Organization (WHO) definitions and classifications. This relies on haemoglobin thresholds specific to age, sex, and physiological status such as pregnancy (World Health Organization & Chan, 2011).

However, confirming that iron deficiency is the specific cause of the anaemia is not always straightforward and often requires additional laboratory investigations. A complete blood count (CBC), also known as a full haemogram, can provide valuable clues. In classic presentations of iron deficiency anaemia, the complete blood count typically reveals microcytosis (small red blood cells), hypochromia (pale red blood cells due to reduced haemoglobin content), and elevated red cell distribution width (RDW), which reflects increased variability in red blood cell size. These findings are characteristic of iron deficiency anaemia and can support the diagnosis. However, it's important to note that not all cases of iron deficiency anaemia present with these classical features. In fact, studies show that up to 40% of confirmed iron deficiency anaemia cases may appear normocytic where the red blood cells are of normal size particularly in the early stages of the condition or when co-existing conditions are present (Aspuru et al., 2011). This underscores the importance of combining haematological findings with biochemical iron studies for accurate diagnosis.

The next crucial step in confirming iron deficiency, especially in the context of anaemia, involves the evaluation of iron-specific biochemical markers. These include total iron-binding capacity (TIBC), serum ferritin, transferrin saturation (TSAT), and serum iron levels. Among these, serum ferritin is widely regarded as the most specific and sensitive single indicator of total body iron stores. A serum ferritin level of less than 30 ng/L in the absence of inflammation is strongly indicative of iron deficiency. Ferritin, being a storage protein, reflects the iron reserves in the liver, spleen, and bone marrow, and thus, low levels suggest depleted iron stores.

Transferrin saturation is calculated by dividing serum iron by total iron binding capacity and converting it into a percentage. This value provides insight into the extent to which transferrin is saturated with iron. A transferrin saturation value of less than 16%–20%, especially when accompanied by a low ferritin level, supports a diagnosis of iron deficiency. In clinical practice, the interpretation of transferrin saturation is particularly useful when ferritin results are inconclusive due to the presence of coexisting inflammatory conditions.

Other laboratory parameters supportive of iron deficiency include low serum iron levels (<30 mcg/dL) and elevated total iron binding capacity values (>480 mcg/dL). Total iron binding capacity measures the blood's capacity to bind iron with transferrin and typically increases when iron levels are low. Another helpful diagnostic marker is the Metzner Index, calculated as red blood cell count divided by the mean corpuscular volume; values less than 13 are suggestive of iron deficiency, particularly in cases of microcytic anaemia (Özdemir, 2015).

In inflammatory states, serum ferritin levels can be falsely elevated, masking underlying iron deficiency. Therefore, higher ferritin cut-off values (<100 ng/L) are recommended to define iron deficiency in such conditions. In patients with chronic diseases like heart failure or chronic kidney disease, even higher thresholds are used, such as ferritin levels below 300 ng/L in combination with transferrin saturation levels below 30% (Aspuru et al., 2011; Camaschella, 2015).

Once laboratory indicators confirm iron deficiency, clinicians should proceed to identify the underlying aetiology. This often requires individualized assessment based on the patient's clinical history, nutritional status, comorbidities, and other contributing factors (Aspuru et al., 2011).

Serum ferritin remains the most sensitive and specific laboratory marker for detecting iron deficiency in both clinical and research settings. Ferritin reflects the level of iron stores in the body, and in the absence of inflammation or infection, low levels are directly indicative of depleted iron reserves. A serum ferritin level below 30 ng/mL is generally diagnostic of iron deficiency in healthy individuals. However, because ferritin is also an acute phase reactant, its concentrations can be falsely elevated in the presence of inflammatory states, infections, malignancies, or chronic liver disease. Therefore, when interpreting serum ferritin levels, it is crucial to simultaneously assess markers of inflammation, such as C-reactive protein (CRP), to avoid misdiagnosis (Baker & G., 2010).

On the other hand, total iron-binding capacity (TIBC) is considered one of the least reliable tests for diagnosing iron deficiency. TIBC exhibits diurnal variation, with levels fluctuating throughout the day, and it is particularly prone to artefactual errors due to potential contamination during sample handling or equipment inconsistencies. Moreover, TIBC levels can normalize shortly after iron supplementation, thereby masking underlying deficiency and producing misleading results (Aspuru et al., 2011; Camaschella, 2015). For these reasons, TIBC is often interpreted alongside other iron markers rather than relied upon independently for diagnosis.

On a complete blood count (CBC), several haematological parameters are indicative of iron deficiency and iron deficiency anaemia. One of the earliest and most sensitive indicators is an elevated red cell distribution width (RDW), typically greater than 14%, which reflects increased variation in red blood cell size (anisocytosis). Haemoglobin (Hb) levels are reduced and should be interpreted using World Health Organization (WHO) criteria, which provide age- and sex-specific thresholds for diagnosing anaemia.

Other red cell indices that suggest iron deficiency include a low mean corpuscular volume (MCV), signifying microcytosis; a low mean corpuscular haemoglobin (MCH), usually less than 27 picograms, indicating hypochromia or decreased haemoglobin content per cell; and a low mean corpuscular haemoglobin concentration (MCHC), generally under 30%, which further confirms diminished haemoglobin saturation within red blood cells.

Peripheral blood film examination provides additional qualitative evidence. Classic morphological features seen in iron deficiency include hypochromia (pale red cells), microcytosis (small red cells), anisochromia (variable colour density), anisocytosis (variation in cell size), and the presence of pencil cells—elongated, cigar-shaped red blood cells. These findings together form a diagnostic picture that, when interpreted alongside iron studies, support a diagnosis of iron deficiency anaemia (Aydogan et al., 2019; Özdemir, 2015).

2.9 Dietary diversity scoring

Dietary diversity that involves daily consumption of various varieties of food groups leads to better nutrition outcomes. It is a critical determinant of nutritional adequacy, particularly in infants and young children. Consuming a variety of food groups daily ensures the intake of essential macro- and micronutrients, thereby reducing the risk of deficiencies that can impair growth and development. Coupled with optimal infant and young child feeding (IYCF) practices, dietary diversity contributes significantly to improved nutritional outcomes. The World Health Organization (WHO) emphasizes that proper IYCF practices, including early initiation of breastfeeding, exclusive breastfeeding for the first six months, continued breastfeeding at one year, appropriate weaning practices, and the consumption of a diverse diet, are fundamental to reducing both macro- and micronutrient deficiencies (World Health Organization, 2008).

The core indicators for proper infant and young child feeding (IYCF) practices among children aged 6 to 23 months are crucial in ensuring the healthy growth and development of children. These indicators include early initiation of breastfeeding, which should occur within the first hour after birth, and exclusive breastfeeding for the first six months to provide optimal nutrition and immunity. Continued breastfeeding at one year of age is encouraged to support ongoing nutritional needs. Proper weaning practices should begin at six months, introducing safe and nutritious complementary foods while continuing breastfeeding. Ensuring a minimum dietary diversity, where children consume foods from at least four of the seven recommended food groups, is vital. Additionally, children should have adequate meal frequency, meaning they receive enough meals each day. A minimum acceptable diet, combining both dietary diversity and meal frequency, should be complemented by the consumption of iron-rich or iron-fortified foods to combat micronutrient deficiencies (World Health Organization, 2008).

The World Health Organization (WHO) provides a standardized set of seven food groups for assessing dietary diversity. These food groups are: grains, roots, and tubers; legumes and nuts; dairy products (including milk, yogurt, and cheese); flesh foods (which encompass meat, fish, poultry, and liver or organ meats); eggs; vitamin-A-rich foods (such as orange vegetables and fruits, leafy greens); and other fruits and vegetables. These food groups cover a wide range of essential nutrients necessary for growth and development, and their inclusion in the diet ensures an adequate intake of micronutrients such as vitamins and minerals (World Health Organization, 2008).

The WHO suggests that a minimum dietary diversity score (DDS) should be based on the consumption of at least four of these seven food groups. This ensures that children, especially those aged 6 to 23 months, receive a balanced variety of nutrients from different sources. The DDS is a valuable tool for assessing the risk of micronutrient deficiencies within a population, as it provides a simple and reliable indicator of dietary quality (World Health Organization, 2008).

To calculate the DDS, the 24-hour dietary recall method is typically used. This method minimizes recall bias by asking caregivers to report all foods and beverages consumed by the child in the past 24 hours. This data is then used to approximate the dietary diversity and assess the child's nutritional status. Studies have shown that DDS is effective in identifying children at risk of undernutrition and micronutrient deficiencies, allowing for targeted interventions and improvements in child feeding practices (Arsenault et al., 2010; Gina et al., 2010; Hailemariam et al., 2018; Shah, 2011; Steyn et al., 2014).

2.10 Conceptual Framework

This study aims to explore how a variety of socio-demographic, economic, nutritional, and clinical factors contribute to the development of zinc and iron deficiencies, as well as the insufficient intake of zinc. The conceptual framework for this study recognizes the complex interplay between these factors and how they collectively impact an individual's nutritional status, particularly in relation to micronutrient deficiencies like zinc and iron.

At the core of this framework are the socio-demographic factors, which include variables such as age, gender, and educational background of caregivers or household heads, as these can significantly influence dietary patterns and access to essential nutrients. For example, children in households where caregivers have lower levels of education may be more likely to have poor dietary practices, leading to higher risks of micronutrient deficiencies. Gender can also play a role, as in many cultures, nutritional resources may be allocated unequally between males and females, potentially putting women and girls at higher risk for deficiencies.

Economic factors, such as household income, access to health services, and food security, are also critical in shaping a household's ability to provide nutrient-rich foods. Families with lower income levels often face constraints in purchasing diverse and nutrient-dense foods, which are vital for preventing deficiencies in zinc and iron. Additionally, food insecurity, which is commonly linked to poverty, can limit the availability of foods rich in these essential micronutrients, further exacerbating the problem.

Nutritional factors, particularly dietary intake patterns, are central to this study. The intake of zinc and iron, whether from food sources or supplements, is directly influenced by the availability and accessibility of foods rich in these nutrients. Poor dietary diversity, characterized by low consumption of iron-rich or zinc-rich foods, is a significant contributing factor to the development of deficiencies. For instance, populations that rely heavily on staple foods like rice or maize, which are low in these nutrients, may be more prone to developing deficiencies. Furthermore, factors such as the use of fortified foods and adherence to proper infant and young child feeding (IYCF) practices, including exclusive breastfeeding, are crucial for meeting the nutritional needs of children.

Clinical factors also play a vital role in the development of zinc and iron deficiencies. Chronic diseases or conditions such as malabsorption, gastrointestinal disorders, or infections can impair the body’s ability to absorb or utilize nutrients, thereby increasing the risk of deficiencies. Furthermore, poor growth indicators such as stunting in children, as well as clinical signs of deficiency (e.g., pale skin, lethargy, or gastrointestinal symptoms), can act as visible markers for zinc and iron deficiencies. These clinical indicators are essential for early identification and treatment of micronutrient deficiencies.

In summary, the conceptual framework for this study demonstrates that zinc and iron deficiencies result from a combination of socio-demographic, economic, nutritional, and clinical factors. These factors do not operate in isolation; rather, they interact in complex ways that determine nutritional outcomes. This framework highlights the need for a multifaceted approach to addressing micronutrient deficiencies, one that considers not only individual dietary habits but also broader socio-economic and clinical determinants. By recognizing these interconnections, the study aims to provide a comprehensive understanding of the underlying causes of zinc and iron deficiencies, thereby informing targeted interventions that address the root causes of malnutrition at both the individual and community levels. This is as shown in figure 1 below:

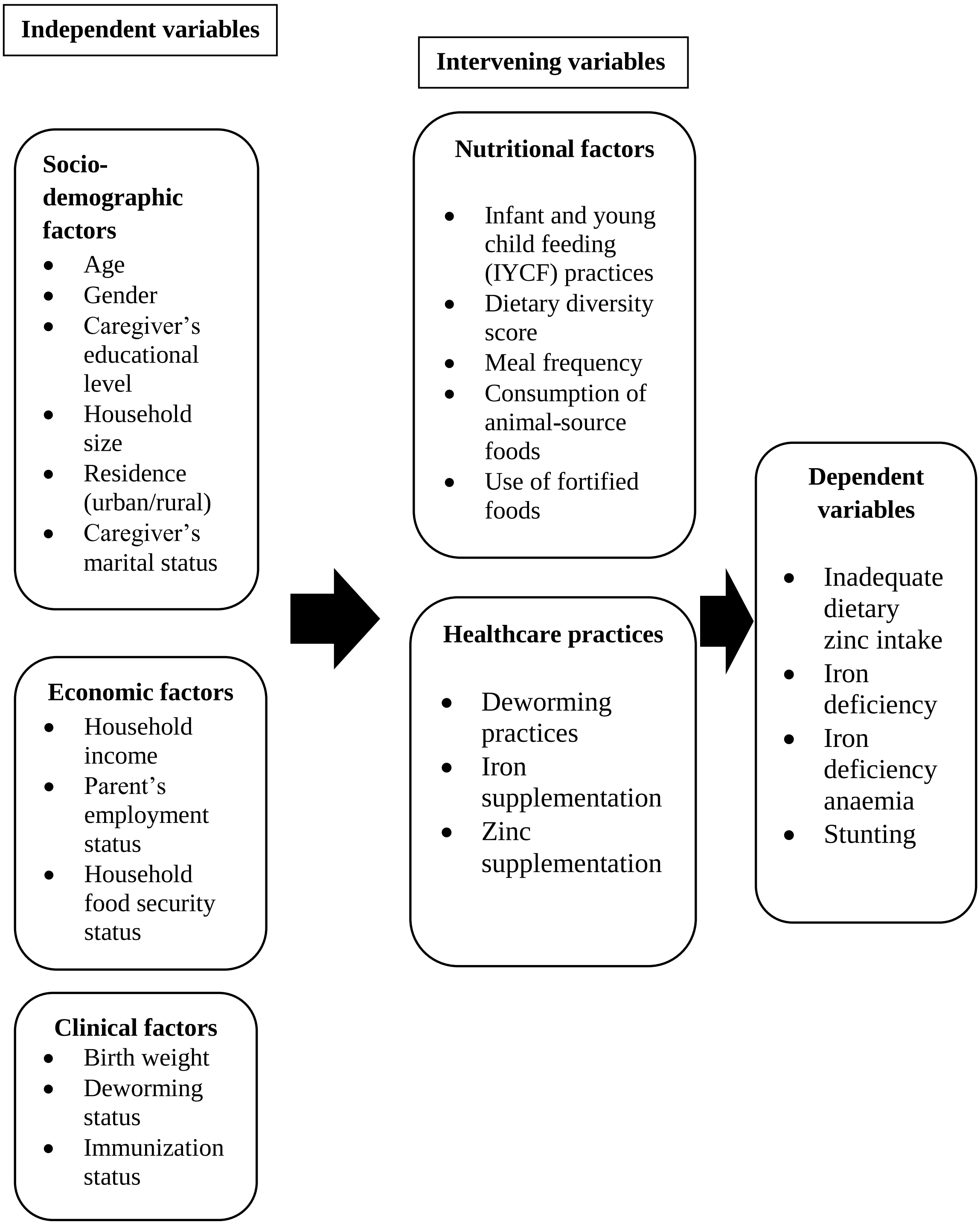

Figure 1: Conceptual framework.

Adapted from World Health Organization. (2013). Essential nutrition actions: Improving maternal, newborn, infant and young child health and nutrition.

CHAPTER THREE

3.0 METHODS

3.1 Study design

The study was an analytical cross-sectional study. The study was carried out between March 2022 and July 2022.

3.2 Study setting

The study was conducted at the Moi Teaching and Referral Hospital (MTRH) paediatric outpatient department. MTRH is the second-largest tertiary healthcare facility in Kenya, providing medical services to a population spanning at least 22 counties in the western region of the country.

As a Level 6 facility, it functions as a referral hospital for lower-level healthcare institutions within its catchment area, playing a crucial role in the region's healthcare delivery. The hospital is situated in Eldoret town, Uasin Gishu County, approximately 320 kilometers northwest of Nairobi, the capital city of Kenya. MTRH serves a large and diverse patient population, including children with various medical conditions, making it an ideal setting for studying paediatric health issues, including zinc and iron deficiencies.

The facility has a well-established paediatric unit that provides both in-patient and out-patient services. The paediatric services are offered at Shoe4Africa children's' hospital, a stand-alone paediatric wing of MTRH. This wing of the hospital has the outpatient clinics for the sick and well-baby clinics. In this unit, children that are coming for wellness checks and immunization are attended to as well as those who are sick and require routine or emergency care.

This facility is ideal for this study, since it will give a good case-mix of children with a wide variety of health states or conditions to be able to provide a clear picture of the state of zinc and iron micronutrient deficiency.

3.3 Study population

Children aged 6 -59 months seen at MTRH outpatient department.

3.4 Sample size calculation

The sample size was determined using Cochran's formula using prevalence estimates for Zinc and Iron deficiency by the Ministry of Health that estimates that at least 34.6% and 83.3% of children aged 6 -59 months suffer from iron and zinc deficiency respectively (Ministry of Health - Kenya, 2015).

We used the iron deficiency prevalence estimate since it yielded the higher sample size so that the study is powered to draw conclusions on deficiency of both micronutrients.

$$n = \frac{Z^2 p(1 - p)}{e^2}$$

Z = 1.96; p = 0.346 (Prevalence of iron deficiency as per Kenya National Micronutrient Survey, 2011); e = 0.05

n = 354 persons

3.5 Sampling technique

We used systematic random sampling technique. MTRH paediatric outpatient clinic sees approximately 6,000 children every month. The data was collected over a five-month period. The sampling interval was every ninetieth (90^{th}) patient was recruited into the study. This interval was determined by dividing the estimated number of children to be seen over the period divided by the sample size. In the previous year

(2021), over a five-month duration, a total of 31,694 children were seen at the out-patient clinic.

$$(k)^{th} = \frac{31,694}{354}$$

$$= \mathbf{89.5 \text{ or} \approx \text{the } 90^{th} \text{ participant}}$$

3.5.1 Inclusion Criteria

- Children aged 6 – 59 months seen at MTRH out-patient department

3.5.2 Exclusion Criteria

- Children aged 6 – 59 months with bleeding tendencies and bleeding disorders
- Children aged 6 – 59 months whose parents/guardians are not able to communicate in English or Kiswahili and no translator is available hence leading to communication barrier

3.6 Study procedure

The study participants were enrolled and recruited at the point of entry into the hospital. The participants were recruited from the registration desk at Shoe for Africa which is a central point where all children seen at the outpatient department are first seen and their details captured.

The parents/guardians of the children aged 6 – 59 months were approached. The purpose, risks and benefits as well as the key aspects of the study were explained to them. Upon understanding, they were taken through the informed consent process.

The first participant was selected randomly then subsequently the 90^{th} participant was systematically enrolled. After triage and written informed consent, the study questionnaire was administered to the parent/guardian of the study participant. They then proceeded to the nutritionist who administered the nutritional component of the questionnaire and carried out anthropometric and nutritional assessment. After which, they proceeded with the normal hospital consultation and review processes by hospital staff. Laboratory tests and blood samples were taken thereafter. The results of the laboratory tests were then filed in the patient's file for use by the clinical teams in the management of the participants.

3.7 Data collection and laboratory tests

Prior to the onset of the data collection, we recruited and trained research assistants. During the project, we recruited clinical officers with specialized training in paediatrics and nutritional officers. We took them through training on the study as well as the ethical considerations relating to the project. We then familiarized them on the consent process as well as the data collection tool.

Data was collected through pre-tested structured interviewer-administered questionnaires, anthropometric data as well as laboratory tests. The data collected on the independent variables included; socio-demographic data (age, gender, residence, family size, education level and employment status of the parents/guardians); clinical history of the patient included prenatal, natal and postnatal history, treatment history (both in and out-patient), immunization history, deworming, HIV status as well as current signs and symptoms during the current hospital visit.

The anthropometric measurements were taken by the nutritionist. The measurements of interest included: the weight, height/length and middle upper arm circumference (MUAC) as per standard operating procedures. Data on the meals, beverages and snacks consumed by the child in the preceding 24 hours was collected. The details obtained on the meals included: the number / frequency of meals consumed, the estimated portion sizes (using diagrams and standard size household utensils) and the ingredients used to prepare the meals.

Blood samples were collected by hospital phlebotomists who handled the samples and subsequently delivered the results. Blood samples from the participants was collected, handled and processed at MTRH laboratory using their standard operating procedures (SOPs). We collected 3 mls of blood from each participant. 1ml was be used for haematology analysis (complete blood count) then the serum extracted from the remaining 2mls sample was used for biochemical analysis (serum iron, unsaturated iron binding capacity, serum ferritin).

For complete blood count, we used the following haematology analyzers; ADVIA 2120i and ADVIA 560 whereas for biochemical analysis they shall use COBAS Integra 400plus, COBAS E411 or COBAS 311.

Serum iron and unsaturated iron binding capacity were summed up to get total iron binding capacity. Serum iron and total iron binding capacity were used to derive the transferrin saturation levels by diving serum iron and TIBC then converting it into a percentage. Transferrin saturation levels are a marker for how much of the serum iron is transported in serum while bound to transport proteins.

Ferritin is the primary intracellular iron storage protein that stores iron and releases it in a controlled manner. Ferritin is mainly an intracellular protein however small amounts are secreted into serum where it serves as an iron transporter. As such, serum ferritin is considered as an indirect marker of total storage of iron in the human body and by extension a marker for iron deficiency/adequacy status.

The above tests are run on serum that is drawn from whole blood. Serum iron and UIBC are colorimetric tests. They are biochemistry tests in which serum is mixed with reagents and quantification of the various tests are run based on the intensity of the colour and interpreted by the machine/analyzer. Serum iron is quantified on the basis of the intensity of the violet complex formed when iron reacts with ferrozine at 562nm wavelength of light. UIBC is determined by saturating transferrin with iron then colourimetrically measuring the remaining unbound iron (Sampson, 2008). TIBC is derived by combining the serum iron with the UIBC results.

Complete blood counts will help show the level of haemoglobin in blood as well as the red blood indices such as mean corpuscular volume, red blood cell distribution width and mean corpuscular heamoglobin. Features of iron deficiency can manifest by having changes in the above blood indices. The complete blood count machine counts number and sizes of cells in a whole blood sample. It does so by passing both the blood cells and an electric current simultaneously through a small aperture and senses the volume of electrolyte displaced by the cell or particle thereby generating a voltage pulse that is interpreted by the machine and reported in terms of cell sizes and types.

All the laboratory tests and related costs such as printing, carried out during the study were borne by the researcher. No costs related to the laboratory tests were transferred to the parents / guardians of the participants. The results from the tests were relayed to the clinical team for use in the care and treatment of the participants.

3.8 Interpretation of laboratory results (Normal ranges and cutoffs)

3.8.1 Complete Blood Count (CBC)

Haemoglobin (Hb) counts were adjusted for altitude of Uasin Gishu or the place of residence of the participant as per WHO guidelines. The cut off values were as adapted from WHO guidelines on normal haemoglobin ranges and cut off values for anaemia (World Health Organization & Chan, 2011). Table 1 below show the cut off values used for the classification of anaemia.

Table 1: Normal haemoglobin ranges and classification of anaemia

Classification	Cut off values
Normal Hb	≥11.0g/dl
Mild anaemia	10.0 – 10.9 g/dl
Moderate anaemia	7.0 – 9.9 g/dl
Severe anaemia	<7.0g/dl

The normal reference ranges for the red blood cell indices used for analysis were as provided by the laboratory reference figures for this region. The indicators we assessed to determine presence of microcytosis included: increased red cell distribution width (RDW) of greater than 14%, mean corpuscular volume (MCV) with a normal range of 76 – 96 femtolitres (below 76 was considered as microcytosis). We also assessed mean corpuscular haemoglobin (MCH) with normal ranges taken as 27 – 32 picograms (Özdemir, 2015)

3.8.2 Serum Ferritin

Serum ferritin was interpreted in tandem with C-reactive protein CRP, as the marker of inflammation. We used the following serum ferritin level cut off values to signify iron deficiency: <30ng/L in the absence of inflammation, <100ng/L in the presence of inflammation, or <300ng/L for patients with heart failure and chronic kidney disease (Aspuru et al., 2011; Camaschella, 2015; Özdemir, 2015).

3.8.3 C-Reactive Protein (CRP)

We used the reference ranges provided by the laboratory for this region. Persons with CRP values of >5mg/L were considered to be having ongoing inflammatory processes (World Health Organization, 2014).

3.8.4 Transferrin saturation levels

This was derived by dividing the serum iron by the TIBC and converting it into a percentage. We used cut off values of < 16% or <30% in patients with heart failure or chronic kidney disease as an indicator for iron deficiency (Özdemir, 2015).

3.9 Data analysis

The data was entered into Microsoft Excel 2013 for cleaning and validation. For categorical variables, EpiInfo® Version 7.2.0.1 was used in the calculation of measures of association such as odds ratios and adjusted odds ratios. Anthropometric data was analyzed using World Health Organization (WHO) Anthro® software version 3.2.2 to assess nutrition indicators. The nutrition indicators of interest were Weight-for-Height z-score (WHZ), Weight-for-Age z-score (WAZ) and Height-for-Age z-score (HAZ). From the 24-hour food intake recall, we calculated the WHO dietary diversity score from the 7 major food groups.

Participants with anthropometric measurements (WHZ, WAZ, HAZ) less than or equal to -2SD were considered to have moderate acute malnutrition (MAM) while those with measurements less than or equal to -3SD were considered to be having severe acute malnutrition (SAM). We did not use anthropometric measurements that have weight as a factor in those children who met the criteria for SAM and MAM but had oedema since it would compromise on the accuracy of the data. (World Health Organization, 2008).

For this study, the risk for zinc deficiency was assessed using two indicators; adequacy of zinc intake and prevalence of stunting among the study population. Stunting was considered as participants with height-for-age Z score of less than -2SD of WHO reference ranges and severe stunting for those participants with height-for-age Z score of less than -3SD. The risk of zinc deficiency among in this population was be considered as; prevalence of stunting > 20%, and prevalence of inadequate dietary zinc intake of >25% (Gupta et al., 2020b; Hess, 2017a). For the adequacy of dietary zinc intake, we estimated the weight of the consumed foods based on the data obtained on the portion of consumed meals. From the weights, we estimated the zinc content based on the Kenya food composition table 2018. The quantity of zinc intake was then be assessed for adequacy based on recommended dietary zinc requirements by age.

For continuous variables, measures of central tendency such as means, medians as well as their measures of dispersion were calculated. For categorical variables, odds ratios were calculated at 95% confidence interval and p values < 0.05 were be considered statistically significant. Univariate, bivariate and multivariate analysis was carried out. Logistic regression analysis was carried out to establish factors independently associated with zinc and iron deficiency. For the logistic regression model, we considered variables with p-values <0.20. We used unconditional logistic regression analysis using forward selection.

3.10 Data Security and Management

Collected data was stored in two formats: soft and hard copy. Soft copies were kept in private and password protected computers. The hard copies were stored in lockable cupboards to prevent unauthorized access.

3.11 Study funding

The study was financed with funds from Moi Teaching and Referral Hospital intramural research fund to the tune of Kshs. 995,000. Any additional costs were borne by the investigators.

3.12 Data dissemination plan

The research findings shall be communicated to MTRH hospital administration. They shall also be presented to the clinicians and health care providers at the MTRH paediatric out-patient department through continuous medical education (CMEs). The findings shall also be presented at scientific conferences such as the Kenya Paediatric Association (KPA) conference. The research findings shall also be published in reputable nutrition and paediatric journals

3.13 Ethical Considerations

Ethical approval was obtained from Moi University/Moi Teaching and Referral Hospital Institutional Review and Ethics Committee (IREC) prior to the commencement of the study (Formal approval number: 0003375). A research license was also obtained from the National Commission for Science, Technology and Innovation (NACOSTI): License number - NACOSTI/P/19/664. Permission was also obtained from Moi Teaching and Referral Hospital administration prior to the onset of the study. Informed consent was sought and obtained from the parent/guardian of the participants prior to enrollment into the study. All information obtained from the participants was treated with the utmost confidentiality and all data de-identified. No financial or cash incentives were offered to participate in the study.

3.14 Limitations of the study

The study was a single-center hospital-based study hence affects its generalizability to the population. However, MTRH serves populations from diverse sociodemographic and economic backgrounds and therefore offer a heterogeneous sample that reflects key characteristics of the surrounding community. Furthermore, the study participants were not limited to the sick children seen at the hospital. Other well-babies seeking routine services such as immunization and growth monitoring were also recruited, thereby reducing the likelihood of over-representation of extreme nutritional outcomes.

The weight of the meals was estimated based on the meal portion data from the 24-hour dietary recall and not weighed at the time of feeding. This approach was adopted because direct weighing of foods during feeding was not feasible in the study setting and could have interfered with normal feeding practices, potentially altering caregivers' behaviour and children's usual intake.

Furthermore, the 24-hour dietary recall is a widely used and validated dietary assessment method in population-based and clinical nutrition studies. To minimize measurement error associated with portion size estimation, caregivers were assisted using standardized household measures and utensils, food models, and visual aids to improve recall accuracy.

CHAPTER FOUR

4.0 RESULTS

4.1 Sociodemographic characteristics of the study population

A total of 354 participants aged 6 – 59 months were recruited into the study. The median age of the participants was 31 months (IQR 15, 46). Majority were male and accounted for 61% of the total participants. Majority of the patients (81%) resided in Uasin Gishu County where MTRH is located. Most of the participants' respondents (91%) were their mothers. The summary of the socio-demographic characteristics of the study participants is as shown in table 2 below:

Table 2: Summary of socio-demographic characteristics of the study participants

Variable	**Category**	**Number (n)**	**Percentage (%)**
Age group (Months)	<12	64	18
	12-24	83	23
	24-36	59	43
	36-48	67	31
	>48	81	23
Gender	Female	137	39
	Male	217	61
Residence (County)	Uasin-Gishu	286	81
	Nandi	16	5
	Elgeyo-Marakwet	15	4
	Kakamega	7	2
	TransNzoia	7	2
	Others	23	6
Number of children in the household	<4	286	81
	≥4	68	19
Mother's highest education level attained	No education	7	2
	Primary	60	17
	Secondary	164	46
	Tertiary	123	35
Father's highest education level attained	No education	4	2
	Primary	37	10
	Secondary	143	40
	Tertiary	152	43
	Unknown	18	5
Mother's employment status	Formal employment	70	20
	Self-employed	165	47
	Unemployed	115	32
	Unkown / Not applicable	4	1
Father's employment status	Formal employment	142	40
	Self-employed	148	42
	Unemployed	44	12
	Unknown/Not applicable	20	6

4.2 Iron deficiency

Based on transferrin saturation levels, 77% of the study participants had iron deficiency. Based on low serum ferritin levels; after adjusting for inflammation, 63% of the study participants had iron deficiency. Figure 2 below shows the distribution of iron deficiency status with age and is based on the serum transferrin saturation levels.

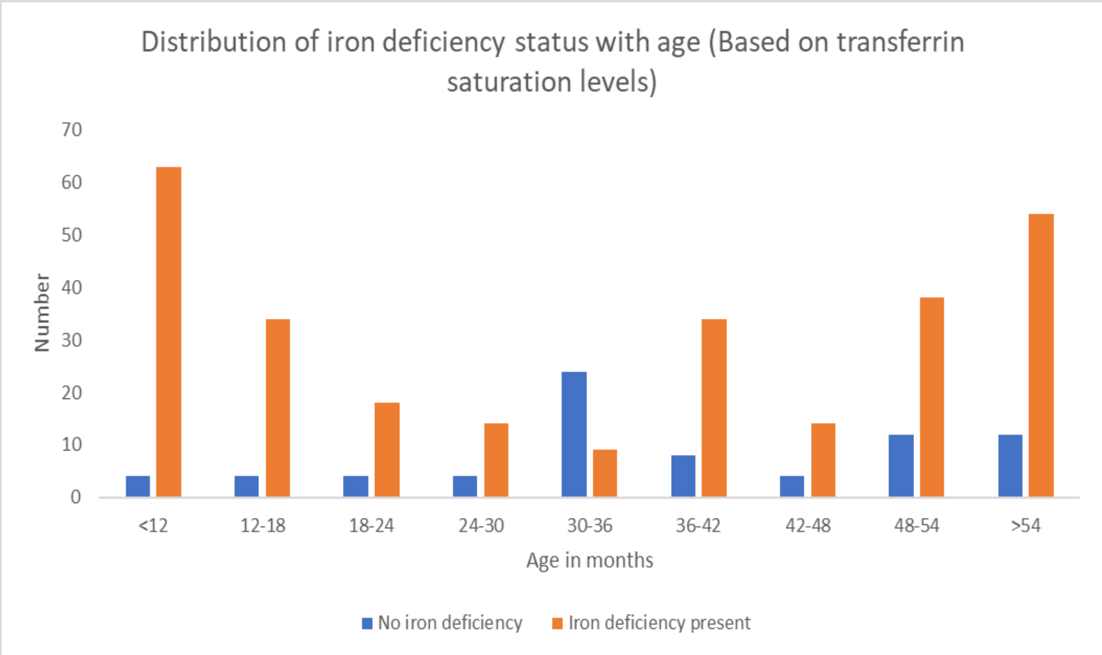

Figure 2: Distribution of iron deficiency status with age (Based on transferrin saturation levels)

Female study participants had higher odds of having iron deficiency compared to their male counterparts (OR 2.8; CI 1.6 – 5.0; $p<0.001$). Children who had been born preterm had higher odds of developing iron deficiency compared to those who were born at term (OR 3.7; CI 0.5 – 28.7; $p=0.18$; $X^2 = 1.8$)

Children who had been dewormed at least once since birth had lower odds of developing iron deficiency compared to those who had never been dewormed (OR 0.2; CI 0.1 – 0.5; $p<0.001$). Only 19% of those who were diagnosed to be iron deficient had clinical signs and symptoms associated with iron deficiency.

Children who had been exclusively breastfed for 6 months post-delivery had lower odds of developing iron deficiency compared to those who had not been exclusively breastfed for 6 months after delivery (OR 0.6; CI 0.1 – 2.7; p= 0.47).

Children who had adequate minimum dietary diversity had lower odds of developing iron deficiency (OR 0.9; CI 0.6 – 1.9; p = 0.83). Children who had received multivitamin supplements at least once since birth had lower odds of having iron deficiency compared to those who had never received the supplements (OR 0.4; CI 0.2 – 0.6; p<0.001). The factors associated with iron deficiency are as summarized in table 3 below:

Table 3: Factors associated with iron deficiency among children aged 6 – 59 months treated at MTRH

	Iron Deficiency?					
	Yes	**No**	**OR**	**CI**	**P-value**	**X2**
Male	118	17	2.8	1.6-5.0	<0.001	
Female	156	63				
Stunted	87	8	4.2	1.9-9.1	<0.001	
Not stunted	187	72				
Pre-term	12	1	3.7	0.5-28.7	0.18	1.8
Term	261	80				
Ever dewormed	189	72	0.2	0.1-0.5	<0.001	
Never dewormed	85	8				
Exclusively breastfed for 6 months	265	76	0.6	0.1-2.7	0.48	
Not exclusively breastfed for 6 months	12	2				
Adequate MDD	122	38	0.9	0.6-1.6	0.83	
Inadequate MDD	129	38				
Ever received MV supplements	109	51	0.4	0.2-0.6	<0.001	
Never received MV supplements	142	25				
Microcytosis on full haemogram	185	51	1.4	0.8-2.3	0.22	
Normocytosis on full haemogram	76	29				
Hypochromia on full haemogram	227	51	3.8	2.1-6.8	<0.001	
Normochromia on full haemogram	34	29				

On logistic regression analysis, the following factors were shown to be associated with iron deficiency: Male children had lower odds of being iron deficient as compared to females (AOR 0.3; CI 0.1 – 1.1; p=0.08). Children who were stunted had higher odds of being iron deficient as compared to those who were not stunted (AOR 1.9; CI 0.3 – 10.6; p=0.46). Children who had been dewormed at least once since birth had lower odds of developing iron deficiency as compared to those who had never been dewormed (AOR 0.3; CI 0.1 – 1.8; p=0.20). Children who had ever received multivitamin supplements since birth had lower odds of being iron deficient as compared to those who had never been given multivitamin supplements (AOR 0.4; CI 0.1 – 1.4; p=0.14). Children who had hypochromia on complete blood count analysis had higher odds of being iron deficient as compared to those without hypochromia (AOR 2.5; CI 0.6 – 9.7; p=0.20). The summary of the logistic regression analysis of the factors associated with iron deficiency are as shown in table 4 below:

Table 4: Logistic regression analysis of factors associated with iron deficiency among children 6 -59 months at MTRH

Variable	AOR	95% CI	p value
Male gender	0.3	0.1 - 1.1	0.08
Children who were stunted	1.9	0.3 - 10.6	0.46
Children who had been dewormed at least once since birth	0.3	0.1 - 1.8	0.20
Children who had ever received multivitamin supplements	0.4	0.1 - 1.4	0.14
Hypochromia on complete blood count analysis	2.5	0.6 - 9.7	0.20

4.3 Anaemia

Fifty-four percent (54%) of the study participants had anaemia. Among those with anaemia, 35% had mild anaemia, 52% had moderate anaemia and 13% had severe anaemia.

On full haemogram, study participants with increased red cell distribution width (>14%) had higher odds of having anaemia compared to those with normal red cell distribution width (OR 3.8; CI 2.1 – 6.8; p<0.001)

On full haemogram, up to 67% of the study participants had microcytosis. Those with microcytosis had higher odds of having iron deficiency compared to those who had normocytosis (OR 1.4; CI 0.8 – 2.3; p = 0.23).

Seventy-eight percent (78%) of the study participants had hypochromia on full haemogram and also had higher odds of having iron deficiency compared to those who had normochromia (OR 3.8; CI 2.1 – 6.8; p<0.001).

4.4 Iron deficiency anaemia

Thirty-eight percent (38%) of the study participants had iron deficiency anaemia. The mean haemoglobin (Hb) level among those with iron deficiency anaemia was 8.9 (SD 1.7). The median age among the study participants who had iron deficiency anaemia was 23 months (IQR 11, 43). Sixty-one percent (61%) of the study participants who had iron deficiency anaemia were male.

The children who had microcytosis had higher odds of having iron deficiency anaemia (OR 2.5; CI 1.6 – 4.0; p<0.001). The children who had hypochromia also had higher odds of having iron deficiency anaemia compared to those who had normochromia (OR 2.8; CI 1.5 – 5.5; p<0.001). The factors associated with iron deficiency anaemia are as summarized in table 5 below:

Table 5: Factors associated with iron deficiency anaemia among children aged 6 – 59 months treated at MTRH

	Iron deficiency anaemia?				
	Yes	**No**	**OR**	**CI**	**P-value**
Hypochromia on full haemogram	118	160	2.8	1.5-5.5	0.001
Normochromia on full haemogram	13	50			
Microcytosis on full haemogram	86	107	2.5	1.6-4.0	<0.001
Normocytosis on full haemogram	38	119			

4.5 Inadequate zinc intake

The prevalence of inadequate zinc intake among the study population was 60%. The median age among those with inadequate zinc intake was 20 months (IQR 11, 48). Majority of those with inadequate zinc intake were males (63%) and higher odds of inadequate zinc intake compared to females (OR 1.1; CI 0.7 -1.7; p=0.65). Twenty-nine percent (29%) of the children with inadequate zinc intake were stunted. Figure 3 below shows the distribution of inadequate zinc intake by age and gender.

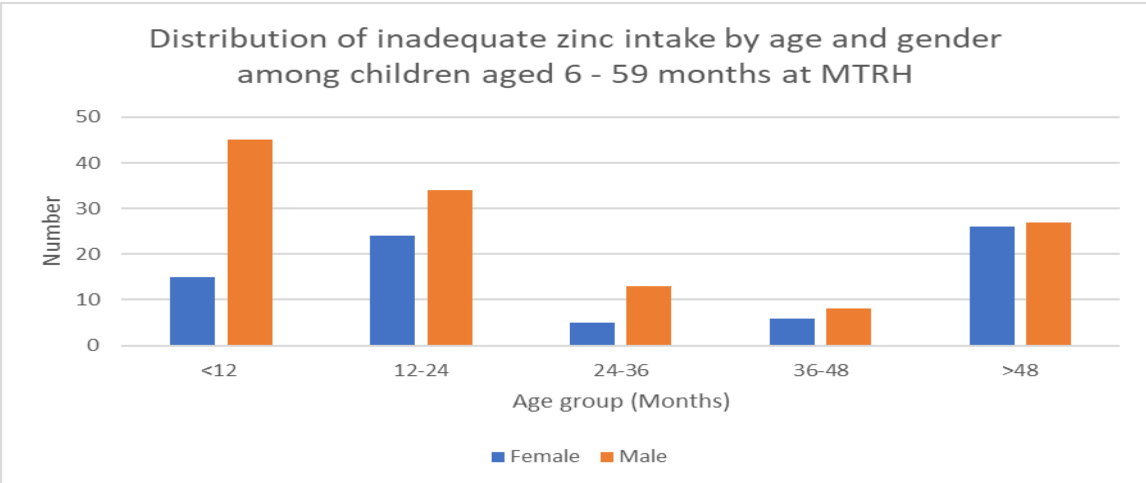

Figure 3: Distribution of inadequate zinc intake by age and gender among children aged 6 - 59 months at MTRH

Seventy-four percent (74%) of the children with inadequate zinc intake had been treated as outpatient clients at least once since birth. Thirty-four percent (34%) of the children with inadequate zinc intake had been treated at hospital at least once as outpatients since birth. The also had higher odds of having been admitted compared to those with adequate zinc intake (OR 1.2; CI 0.7 -2.0; p=0.46).

Fifty percent (50%) of the study participants did not meet the minimum dietary diversity (MDD) score of at least four WHO food groups. Among the seven WHO food groups, the proportion of intake in this study population was as follows; Grains and cereals 92%, Roots and tubers 27%, Legumes and nuts 36%, Dairy products 87%, Flesh foods 18%, Eggs 7%, Fruits and vegetables 69%. Children who did not meet the minimum dietary diversity score had higher odds of having inadequate dietary zinc intake (OR 3.1; CI 2.0 – 4.8; p<0.001). In the 24-hour recall period, only 18% of the study participants had consumed flesh foods. Children who had not consumed flesh foods had higher odds of having inadequate zinc intake (OR 7.1; CI 3.7 – 13.8; p<0.001). Factors associated with inadequate dietary zinc intake is as summarized in table 6 below:

Table 6: Factors associated with inadequate dietary zinc intake among children aged 6 - 59 months at MTRH

	Inadequate dietary zinc intake?				
Variable	**Yes**	**No**	**OR**	**CI**	**P-value**
Male	127	83	1.1	0.7 - 1.7	0.65
Female	76	55			
History of being admitted to hospital	51	30	1.2	0.7 - 2.0	0.46
No history of being admitted to hospital	150	107			
Inadequate minimum dietary diversity	120	46	3.1	2.0 - 4.8	<0.001
Adequate minimum dietary diversity	78	92			
No flesh foods consumed in the last 24 hours	185	92	7.1	3.7 - 13.8	<0.001
Flesh foods consumed in the last 24 hours	13	46			

4.6 Stunting

A total of 26% of the participants were stunted and 10% (of the entire study population) were severely stunted. The median age of those who were stunted was 21 months (IQR 12, 42). Among the stunted children, 67% were male. Children of the male gender had higher odds of being stunted (OR 1.4; CI 0.8 – 2.3; p=0.19). Figure 4 below shows the distribution of stunting by age and gender among the study population.

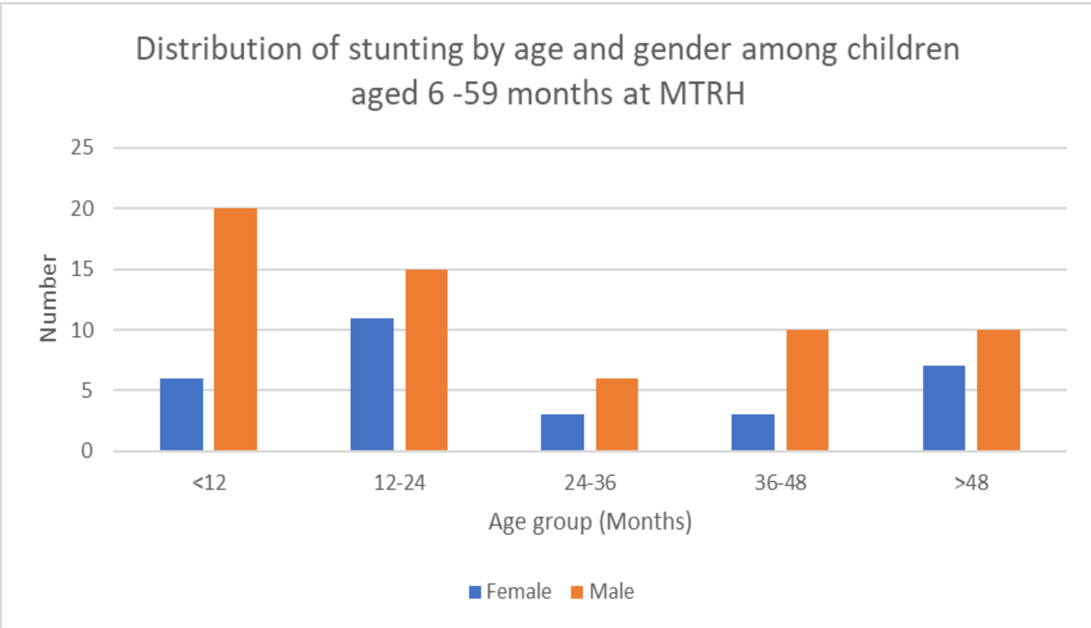

Figure 4: Distribution of stunting by age and gender among children aged 6 -59 months at MTRH

The proportion of mothers who attended antenatal clinic (ANC) during pregnancy bearing the children in the study population was 99%. Among the children who were stunted, 96% of the mothers attended ANC. Children whose mothers did not attend ANC during pregnancy had higher likelihood of being stunted (X^2 = 5.2; p= 0.02).

Five percent (5%) of the study participants were born preterm. Among the children who were stunted, nine percent (9%) were born preterm. Pre-term children had higher odds of being stunted compared to the term babies (OR 3.1; CI 1.1 – 8.5; p=0.02).

Among the stunted, seventy-three percent (73%) had been treated as outpatients at least once since birth. Stunted children had higher odds of having been treated as outpatients for diarrhoeal illnesses compared to those who were not stunted (OR 1.3; CI 0.7 – 2.2; p=0.43) and higher odds of respiratory tract illnesses as well (OR 1.1; CI 0.7 – 1.7; p=0.83).

Children who were stunted had higher odds of having been admitted to hospital at least once since birth due to respiratory tract illnesses compared to those who were not stunted (OR 2.3; CI 1.1 – 4.7; p=0.02) as well as diarrhoeal illnesses (OR 1.1; CI 0.4 – 3.3; p=0.82).

In the study population, 4% had delayed developmental milestones. Among the children who were stunted, 54% had delayed developmental milestones. Children who were stunted had higher odds of having delayed milestones compared to those who were not stunted (OR 3.5; CI 1.2 – 10.9; p=0.02).

With regard to deworming, 72% of the study participants had been dewormed at least once since birth. Fifty one percent (51%) of the children who were stunted had been dewormed at least once since birth as at the time of the study. The children who had not been dewormed at least once since birth had higher odds of being stunted as compared to those who had been dewormed (OR 3.7; CI 2.2 – 6.1; p<0.001).

Only 1% of the study population had never been breastfed since birth. Among those who were ever breastfed since birth, 99% were exclusively breast fed for six months. Children who had ever been breastfed had lower odds of being stunted (OR 0.2; CI 0.0 – 1.3; p=0.07; X^2 = 3.1) similar to those who had been exclusively breastfed for at least 6 months after birth (OR 0.2; CI 0.0 – 1.3; p=0.07; X^2 = 3.1). Continued breastfeeding up to 2 years of age did not have a positive or negative association with stunting (OR 1.0; CI 0.5 – 2.5; p=0.89).

Sixty-seven percent (67%) of the stunted children had inadequate zinc intake. The children who had inadequate zinc intake had higher odds of being stunted (OR 1.5; CI 0.9 -2.6; p=0.09). Fifty-five percent (55%) of the stunted children had inadequate minimum dietary diversity (MDD) scores of less than 4 WHO food groups and also had higher odds of being stunted as compared to children with adequate MDD scores (OR 1.3; CI 0.8 -2.1; p=0.27).

Fifty-four percent (54%) of the study population had received multivitamin supplements at least once since birth while the proportion among those who were stunted was 49%. Children who had received multivitamin supplements at least once since birth had lower odds of being stunted compared to those who had never received it (OR 0.7; CI 0.5 – 1.2; p=0.24). Only 6% of the study population had ever received micronutrient powder supplements since birth. Those who had ever been given micronutrient powder since birth had lower odds of being stunted (OR 0.5; CI 0.2 -1.9; p=0.32).

Thirty-three percent (33%) of the study participants mothers were employed as at the time of the study. Children whose mothers were unemployed had higher odds of being stunted as compare to those whose mothers were employed as at the time of the study (OR 1.3; CI 0.8 – 2.2; p=0.25).

As regards white blood cell (WBC) count on the full haemogram results, 25% of the study participants had leukocytosis while 4% had leukopaenia. Children who were stunted had higher odds of having WBC derangements as compared to those who were not stunted (OR 6.1; CI 3.7 – 10.7; p<0.001). Thirty-six percent (36%) of the study participants had anaemia. Children who were stunted had higher odds of having anaemia compared to those who were not stunted (OR 5.9; CI 3.7 – 9.6; p<0.001).

The factors associated with stunting among the children aged 6 – 59 months at MTRH are as shown in table 7 below:

Table 7: Factors associated with stunting among children aged 6 – 59 months treated at MTRH

	Stunted?					
	Yes	**No**	**OR**	**CI**	**p-value**	**X^2**
Male	61	156	1.4	0.8 - 2.3	0.19	
Female	30	107				
Mother did not attend antenatal clinic	3	1	9	0.9 - 87.7	0.02	5.2
Mother attended antenatal clinic	87	261				
Pre-term at birth	8	8	3.1	1.1 - 8.5	0.02	
Term at birth	82	253				
Ever treated as out-patient for diarrhoeal illness	21	51	1.3	0.7 - 2.2	0.43	
Never treated as out-patient for diarrhoeal illness	69	211				
Ever treated as out-patient for respiratory tract illness	46	131	1.1	0.7 - 1.7	0.83	
Never treated as out-patient for respiratory tract illness	44	132				
Ever treated as in-patient for diarrhoeal illness	21	51	1.1	0.4 - 3.3	0.82	
Never treated as in-patient for diarrhoeal illness	69	211				
Ever treated as in-patient for respiratory tract illness	5	13	2.3	1.1 - 4.7	0.02	
Never treated as in-patient for respiratory tract illness	85	250				
Delayed milestones	7	84	3.5	1.2-10.9	0.02	
Normal	6	257				
Not dewormed	45	55	3.7	2.2-6.1	<0.001	
Dewormed	46	206				
Breastfed	87	259	0.2	0.0 - 1.3	0.07	3.1
Not breastfed	3	2				
Exclusively breastfed for 6 months	85	258	0.2	0.0 - 1.3	0.07	3.2
Not exclusively breastfed for 6 months	3	2				

Inadequate zinc intake	59	144	1.5	0.9 - 2.6	0.1
Adequate zinc intake	29	109			
Indaequate minimum dietary diversity (MDD)	48	119	1.3	0.8 - 2.1	0.27
Adequate minimum dietary diversity (MDD)	40	130			
Ever given multivitamin supplements	42	140	0.7	0.5 - 1.2	0.24
Never given multivitamin supplements	44	109			
Ever given micronutrient powder supplements	3	16	0.5	0.2 - 1.9	0.32
Never given micronutrient powder supplements	84	240			
Mother unemployed	34	81	1.3	0.8 - 2.2	0.25
Mother employed	56	179			
Elevated white blood cell counts	63	32	6.1	3.7 - 10.7	<0.001
Normal white blood cell counts	63	196			
Anaemia present	29	16	5.9	3.7-9.6	<0.001
No anaemia present	19	62			

On logistic regression, the following factors were shown to have an association with stunting: Children who were born prematurely (pre-term birth) had higher odds of being stunted as compared to those who were born at term (AOR 1.9; CI 0.6 – 6.3; p=0.27). Children who had been dewormed at least once since birth had less odds of being stunted as compared to those who had never been dewormed at least once since birth (AOR 0.25; CI 0.14 – 0.44; p<0.001). Children who were stunted had higher odds of having delayed milestones as compared to those who were not stunted (AOR 2.7; CI 0.6 – 11.6; p=0.17). Children who had ever been breastfed had lower odds of being stunted as compared to those who had never been breastfed (AOR 0.33; CI 0.04 – 2.5; p=0.29). Children who had adequate dietary zinc intake in the preceding 24 hours as per the dietary recall had lower odds of being stunted as compared to those who had inadequate dietary zinc intake (AOR 0.9; CI 0.5 – 1.7; p=0.87). Children who had adequate minimum dietary diversity scores in the preceding 24 hours as per the dietary recall had lower odds of being stunted as compared to those who had inadequate dietary diversity scores (AOR 0.9; CI 0.5 – 1.6; p=0.72). Logistic regression analysis of factors associated with stunting in the age group of 6 -59 months is as shown in table 8 below:

Table 8: Logistic regression analysis of factors associated with stunting among children aged 6 - 59 months at MTRH

Variable	AOR	95% CI	p value
Preterm birth	1.9	0.6 - 6.3	0.27
Children who had delayed developmental milestones	2.7	0.6 - 11.6	0.17
Children who had been dewormed at least once since birth	0.25	0.1 - 0.44	<0.001
Children who had ever been breastfed	0.33	0.04 - 2.5	0.29
Adequate dietary zinc intake	0.9	0.5 - 1.7	0.87
Adequate minimum dietary diversity	0.9	0.5 - 1.6	0.72

CHAPTER FIVE

5.0 DISCUSSION

5.1 Iron deficiency and iron deficiency anaemia

5.1.1 Prevalence

There was a high prevalence of iron deficiency and iron deficiency anaemia among the study participants. Iron deficiency anaemia is still recognized globally as the most common nutritional deficiency predominantly affecting children and women of reproductive age group. The prevalence of iron deficiency and iron deficiency anaemia in Sub-Saharan Africa as well as lower-middle income countries still high as compared to the developed world. Other studies have shown iron deficiency among children of this age group to be as high as 62.7% based on ferritin levels. The findings of most of the studies concur with our findings as regards the high prevalence of both iron deficiency and iron deficiency anaemia showing prevalence of between 41 – 70% based on serum ferritin levels (Lemoine & Tounian, 2020; Muriuki et al., 2020; Orsango et al., 2021; Oyungu et al., 2021). As such, according to the WHO, iron deficiency and iron deficiency anaemia still remain to be diseases of public health concern.

These deficiencies still result mainly from inadequate dietary iron intake. The commonest cause of iron deficiency just like other minerals is inadequate intake. Dietary iron exists in two forms. Either as heme or non-heme iron. Heme iron has a greater bioavailability and absorption from the gut as compared to non-heme iron. Heme iron is derived from consumption of animal derived foods whereas non-heme iron arises from plant-based foods (Aspuru et al., 2011; Kumar et al., 2022; Skolmowska & Głąbska, 2019). As per WHO food groups, our study population had low intake of flesh foods that are rich in heme iron. However, they had high intake of fruits and vegetables. Green leafy Improving micronutrient status by increasing dietary iron intake is one of the key measures proposed by WHO to decrease the prevalence of iron deficiency and iron deficiency anaemia (World Health Organization (WHO), 2023a)

5.1.2 Factors associated with iron deficiency and iron deficiency anaemia

In the study population, consumption of plant-based meals as compared to flesh foods was high. This in turn, leads to lower intake of dietary iron that has comparatively lower bioavailability hence predisposing them to higher levels of iron deficiency and iron deficiency anaemia as its sequalae. Several other studies conducted in Kenya have had similar high prevalence and determinants of iron deficiency and iron deficiency anaemia in children aged between 6 to 59 months. In their studies, inadequate dietary intake was cited as a major contributor to their development. Inadequate dietary diversity was seen in at least 48.1% of their participants. The participants also had low intake of flesh foods at 21.8% and green leafy vegetables at 34.6% which are important dietary sources of both heme and non-heme iron. (Kimiywe et al., 2020; Kisiangani et al., 2015; Oyungu et al., 2021; Wangusi et al., 2016; Waswa et al., 2021).

Minimum dietary diversity is an intervention that can improve the intake of micronutrients such as iron. Our study showed that those with adequate minimum dietary diversity had lower odds of iron deficiency. This is because it increases the chances of intake of foods that are rich in either heme or non-heme iron. However, for prevention of anaemia and iron deficiency, emphasis needs to be laid on flesh foods. Other studies globally have also shown this association (Belachew & Tewabe, 2020; Kathuria et al., 2023; Kimiywe et al., 2020; Mendes et al., 2021; Woldie et al., 2015).

Children who were stunted were found to be having higher odds of having concurrent iron deficiency. This could point to concurrent deficiencies of both iron and zinc in the study population. This is however not unique to this study population. Both are associated with chronic undernutrition. However, anaemia would manifest earlier than stunting. As such, presence of anaemia should warrant further nutritional and anthropometric assessment of these children who during routine hospital visits are found to be anaemic. Both of these conditions can be addressed through balanced dietary intake. This has been demonstrated in other studies where this double burden on iron deficiency and stunting are present in children (Malako et al., 2019; S. H. Mohammed et al., 2019).

Deworming decreased the odds of developing iron deficiency in this study. Administration of deworming medications helps in eliminating intestinal worms and helminths that are responsible for blood loss from the intestinal tract. UNICEF considers deworming as one of the interventions to help in preventing anaemia. WHO recommends annual or bi-annual deworming of children under 5 years to decrease the prevalence of iron deficiency and iron deficiency anaemia (World Health Organization (WHO), 2023c). This finding is similar to other studies on the same globally (Bauleni et al., 2022; Moshi et al., 2023). Other studies have compared the effect of only deworming vis a vis concurrently deworming and giving iron and folate supplements. These studies have shown a higher reduction in the prevalence of anaemia by the combination of multiple intervention strategies (Sarma et al., 2022).

Iron is stored within the body as such excess iron not immediately utilized for physiological processes is stored. In our study, children who received multivitamin supplementation, some of which contain iron, had lower odds of developing iron deficiency. WHO recommends daily iron supplementation for children aged 6 – 23 months daily who are in areas where the prevalence of anaemia is at levels of public health concern (>40%). Given that our study population fall within these levels (54%); in addition to adequate dietary iron intake, iron supplementation should be considered (World Health Organization (WHO), 2023b).

Children who were born premature were more likely to have iron deficiency as compare to those born at term. This is because most of the iron transfer from the mother to the fetus while in utero occurs in the third trimester hence those born pre-term will not benefit from this transfer. As such all children with very low birth weight who also tend to be premature need enteral iron supplementation (Fatima et al., 2022; Muleviciene et al., 2018).

Exclusive breastfeeding for six months was found to be protective against development of iron deficiency in this study population. Other studies have also shown this negative association between exclusive breastfeeding for six months and development of iron deficiency beyond the six months (Fentaw et al., 2023; Uyoga et al., 2017). Breast milk though low in iron content has a high bioavailability which increases its absorption from the gut. As such, it is sufficient for the needs of an infant up until the age of six months (Unger et al., 2019). Other studies done in Kenya and beyond have also shown similar findings regarding the content of iron in breastmilk and it sufficiency during the first six months of infancy (Fentaw et al., 2023; Marques et al., 2014; Uyoga et al., 2017). However, exclusive breastfeeding beyond the recommended six months increases the risk of developing iron deficiency and iron deficiency anaemia especially if the breastfeeding mother is iron deficient. This is because the iron needs significantly increase beyond six months of age. As such, complementary feeds that are rich in iron should be introduced at the end of the exclusive breastfeeding period (Buck et al., 2019; Maguire et al., 2013; Meinzen-Derr et al., 2006; Wang et al., 2016).

Iron is an integral part of haemoglobin that is found within the red blood cells. It is therefore vital for oxygen carriage and delivery within the body. In iron deficiency states, normal production of haemoglobin is affected. This leads to the formation of smaller red blood cells (microcytosis) which typically will have low levels of haemoglobin that give the red blood cells their red pigmentation (Chaudhry & Kasarla, 2019). As such, the produced red blood cells will be small (microcytic) and appear pale (hypochromia). This factor also leads to a wider distribution in the width of the produced red blood cells (Aulakh et al., 2009; Carlos et al., 2018). This parameter, red cell distribution width (RDW), is usually given as a result in most routine complete blood count tests done in most hospitals. Including those done in resource limited settings.

Even though other causes of microcytic hypochromic anaemia exist, iron deficiency is the commonest cause globally (Carlos et al., 2018; Kafle & Lakhey, 2016). In our study, those with microcytosis and hypochromia had higher odds of being iron deficient. This has been shown to be the case in many studies. With this in mind, microcytosis and hypochromia be put in predictive models to detect iron deficiency as key variables. Could iron replacement therapy be commenced in such patients with microcytosis and hypochromia without the express need to carry out iron studies which tend to be costly and not readily available in most hospitals in resource limited settings?. This is an area that can be explored in prospective studies (Aulakh et al., 2009; Aydogan et al., 2019; S. Mohammed et al., 2020)

5.2 Zinc deficiency and inadequate dietary zinc intake

5.2.1 Prevalence

Inadequate dietary zinc intake, stunting and serum zinc levels are the three globally recognized methods of estimating prevalence of zinc deficiency (Gibson, 2012; Wessells & Brown, 2012). For this study we utilized inadequate dietary intake and stunting as indicators for estimating zinc deficiency. In this study population, there is still a large proportion of children under the age 6 to 59 months who have insufficient dietary zinc intake and a high prevalence of stunting. In any population, when the proportion of those who are stunted is >20% and the proportion of those with inadequate dietary zinc intake is >25%, then they can be considered to be at a high risk of zinc deficiency (Dembedza et al., 2023; Gupta et al., 2020a; Hess, 2017b).

Zinc deficiency and inadequate dietary zinc intake are still global challenges of public health concern. They disproportionately affect persons living in underdeveloped and low middle-income countries (Bains et al., 2015). In these countries the levels of inadequate dietary zinc intake go as high as 77% (Gupta et al., 2020b) which is almost comparable to the findings of our study. Children under five are adversely affected since zinc as a micronutrient is essential for normal growth and development (Abdollahi et al., 2019).

This study demonstrated that inadequate zinc intake was associated with stunting in children aged 6 to 59 months of age. The most recent Kenya Demographic and Health Survey (2022) estimated the number of children under 5 years who were stunted to be 18%. In Uasin Gishu, which constituted the larger proportion of our study population, the survey estimated that the proportion of children under 5 years who are stunted is 14%. Our study, as opposed to the population-based demographic and health survey, was a hospital-based. Children who are stunted are more likely to fall ill and present to hospital. This can partly explain the higher percentage of stunting as shown in our study. We also demonstrated that children who were stunted had higher probability of having history of having been treated at hospital either as in- or outpatient. This finding is congruent with other studies that have shown that zinc is essential for the normal functioning of the immune system (Abdollahi et al., 2019; Ahsan et al., 2021; Bains et al., 2015; Gupta et al., 2020b)

5.2.2 Factors associated with inadequate zinc intake

The inadequate dietary zinc intake can be partly explained by factors such as lack of minimum dietary diversity among this study population. Minimum dietary diversity is a good indicator of adequacy of macro and micronutrient intake (Dangura & Gebremedhin, 2017). This is especially important in children under 5 years who require adequate nutrition for growth and development. Having a wider variety of foods/dietary intake increases the chances of having an adequate intake of micronutrients (Ayana et al., 2018; Molani-Gol et al., 2023).

In our study population, the majority of the study participants had mainly consumed grains and cereals followed by fruits and vegetables as classified according to the WHO food groups. Consumption of flesh foods, which are a rich source of zinc, was low. Plant based foods, as opposed to flesh foods and animal-derived foods, have other chemicals such as phytates, polyphenols and oxalates that negatively affect the absorption of minerals from the gastrointestinal tract (Gatobu et al., 2016; Nancy F Krebs et al., 2014; Molani-Gol et al., 2023).

As such, grains and cereals, that seem to the staple food consumed in this population may have lower bioavailability of the minerals such as zinc and iron amongst others. Other studies in Africa and other low-income countries have also shown that communities in which the staple foods are heavy on plants and cereals tend to be at risk of inadequate zinc and iron intake and as such develop these deficiencies and their sequalae (Gegios et al., 2010; Marcos et al., 2019).

These dietary staples in turn, lead to higher prevalence of micronutrient deficiencies that, in these children may present with or without clinical manifestation. Part of the manifestations include; increased morbidity due to lower immunity, anthropometric discrepancies such as wasting and stunting and features of iron deficiency/anaemia (Abdollahi et al., 2019; Dangura & Gebremedhin, 2017; Gatobu et al., 2016; Molani-Gol et al., 2023). This was evident in our study as shown by the high prevalence of stunting and anaemia.

In our study, the children who had received multivitamin supplements and or micronutrient powder at least once since birth had lower odds of being stunted compared to those who had not taken them. Multivitamin supplements usually also contain trace elements such as Zinc in their composition. However, since zinc is not stored in the body, a single administration is unlikely to account for the positive association with lower odds of stunting.

This has been demonstrated in long term clinical trials that have not shown statistically significant association between multivitamin and micronutrient supplementation with decreased odds of stunting (Alfonso Mayén et al., 2022; Castro et al., 2017; Dossa et al., 2002; Hassanzadeh-Rostam et al., 2014). Other studies have tried to address the issue of inadequate zinc intake by fortifying the sources of water.

This is achieved at household level by using water purifying devices that deliver a precise amount of zinc into the water that passes through it. They have shown that this can increase the amount of bioavailable zinc and partly address the inadequacy of dietary zinc intake (Kujinga et al., 2018).

CHAPTER SIX

6.0 CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

- Iron deficiency and iron deficiency anaemia among children aged 6 – 59 months are still highly prevalent diseases of public health importance
- Concurrent iron and zinc deficiency is common in children aged 6 – 59 months
- There is still a large proportion of children aged 6 – 59 months that have inadequate dietary zinc intake and as such are at risk of zinc deficiency
- Adequate minimum dietary diversity of at least 4 food groups as per WHO categories is necessary for prevention of iron and zinc deficiency among children aged 6 – 59 months
- Deworming in children aged 6 – 59 months helps in prevention of iron deficiency
- Exclusive breast feeding for 6 months helps in prevention of iron deficiency among children aged 6 – 59 months
- Children who were born pre-term are at a greater risk of being stunted as well as being iron deficient

6.2 Recommendations

- Ministry of Health, and by extension, health care workers should be on the lookout for signs and symptoms of iron and zinc deficiency since they are still prevalent to levels of public health importance
- Hospital nutritionists should enhance dietary counselling to mothers with children aged 6 – 59 months during clinic visits with special emphasis on WHO food groups and dietary diversity
- Hospitals should enforce deworming practices of children aged 6 – 59 months at least every 6 – 12 months as recommended by WHO during their visits to child welfare clinics
- Mothers should be counselled to exclusively breastfeed newborns and infants for the first six months of life as recommended by WHO and the ministry of health in Kenya
- Paediatricians should closely monitor children born preterm during child welfare clinic visits so that any features of stunting, zinc or iron deficiency are addressed promptly

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APPENDICES

Appendix 1: Project Budget

ITEM			Item Cost	Number	Total
Laboratory tests	Iron tests	Full haemogram	300	354	106,200.00
		Serum iron	300	354	106,200.00
		Serum ferritin	300	354	106,200.00
		Unsaturated iron binding capacity	300	354	106,200.00
	Inflammation tests	C-Reactive Protein	300	354	106,200.00
	Sample storage		200	354	70,800.00
Sub-total					**601,800.00**
IREC Fees					5,000.00
Stationery and supplies					65,000.00
Research Assistants					30,000.00
Manuscript development and publication					173,200.00
Conference presentation					120,000.00
Total					**995,000.00**

Appendix 2: Data Collection Tool

Questionnaire No__________________________

Date of Interview__________________________

1.0 Demographic Information

Case ID: _____________Guardian/Parent Tel No. __________________

Date of Birth: ________________ Birth Order _________ of ___________

Gender: Male ☐ Female ☐ *(Tick as applies)*

Residence

____

Respondents relationship to participant Mother ☐ Father ☐ Guardian ☐

Other, specify _____________________

2.0 Anthropometric Data

(Please fill in the data as measured on the participant)

Measurement	**Value**
Weight (Kilograms)	
Height/Length (centimetres)	
MUAC (centimetres)	
HC (centimetres)	
Oedema present	Yes ☐ No ☐ If yes is it, Pedal ☐ Periorbital ☐ Anasarca ☐

3.0 Clinical Data order

3.1 Did the mother attend ANC while pregnant with *(name)?*Yes ☐ ☐No ☐
Don't know

If yes, how many times did you attend the clinic?

1 ☐ 2 ☐ 3 ☐ 4 ☐ >4 ☐

3.2 Did you take iron and folic acid supplements during the pregnancy? *(mother)*

Yes ☐ No ☐ Don't Know ☐

At what gestation?______________ and for how long__________?

3.3 At what gestation was the baby born? Preterm ☐ Term ☐

3.4 What was the child's birth weight? ________________________________

3.5 Has your child been treated as outpatient for any illness since birth? Yes ☐ No ☐

If yes, which one, and how many times?

Diarrhoeal Illness ☐ How many times? __________

Malaria ☐ How many times? __________

Respiratory Tract illness ☐ How many times? __________

Others, specify_________________, How many times? ____________

3.10 Has your child been admitted for any illness since birth? Yes ☐ No ☐

If yes, which one, and how many times?

Diarrhoeal Illness ☐ How many times? __________

Malaria ☐ How many times? __________

Respiratory Tract illness ☐ How many times? __________

Others, specify_________________, How many times? ______________

3.11 Has your child been immunized? Yes ☐ No ☐

If yes, is ANC booklet available? Yes ☐ No ☐

BCG scar seen? Yes ☐ No ☐

Immunization status Up to date ☐ Completed ☐ Incomplete ☐

(*Interviewer to compare with immunization chart provided*)

3.12 How are the child's developmental milestones? *(Interviewer to compare the milestones with age and expected milestones-for-age in the development chart provided)*

Normal ☐ Delayed ☐

3.13 What is the HIV status of the child?

Positive ☐ Negative ☐ Sero-exposed ☐ Don't Know ☐

3.14 Has your child ever been dewormed? Yes ☐ No ☐ Don't Know ☐

If Yes, When was the last de-worming? < 3 mon☐ 3-6☐nths >6 months ☐

4.0 History and Key examination Findings

4.1 Presenting complaints

Cough ☐ Diarrhea ☐ Vomiting ☐
Fever ☐

Other specify: ____

4.2 For how long *(in days)* has your child had these symptoms? (*Circle as applies)*

<1 day 1 2 3 4 5 6 7 >7 days

4.3 Symptoms and signs of iron and zinc deficiency *(Tick all that apply)*

Symptoms and Signs of Iron Deficiency	**Present**	**Symptoms and Signs of Zinc Deficiency**	**Present**
Extreme fatigue.		Diarrhea	
Pale skin.		Excessive hair loss	
Dizziness		Fine tremor	
Shortness of breath		Poor wound healing	
Inflammation/soreness of your tongue		Dermatitis	
Brittle nails		White spotting of nails	
Pallor *(On examination)*		Others *(Specify)*……………….	
Koilonychia *(On examination)*			
Others *(Specify)*……………….			

5.0 Nutritional Data

5.1 Was your child ever breastfed? Yes ☐ No ☐ Don't Know ☐

If yes, how long after birth was your child breastfed?

Within1 hour ☐ Within 1 day ☐ More than 1 day ☐ Don't Know ☐

5.2 How long was your child fed on breast milk alone?

<6 months ☐ At least six months ☐ >6 months ☐ Don't Know ☐

5.3 Is your child still being breastfed? Yes ☐ No ☐ Don't Know ☐

If no, at what age did you stop breastfeeding your child? ______________

5.4 Did you breast feed your child yesterday *(Day or Night)* Yes ☐ No ☐ Don't Know ☐

5.5 Did your child eat any solid, semi-solid or soft foods yesterday *(Day or Night)*?

Yes ☐ No ☐ Don't Know ☐

If yes, how many times did you feed them on these foods? __________Don't remember ☐

5.6 Could you describe/list the foods your child ate yesterday *(Day or Night)* both at home and outside the home

BREAKFAST					
Name	**Food description**	**Preparation ingredients**	**Household amount**	**Time**	**Amount (g/ml)**
SNACK					
Name	**Food description**	**Preparation ingredients**	**Household amount**	**Time**	**Amount (g/ml)**
LUNCH					
Name	**Food description**	**Preparation ingredients**	**Household amount**	**Time**	**Amount (g/ml)**
SNACK					
Name	**Food description**	**Preparation ingredients**	**Household amount**	**Time**	**Amount (g/ml)**
SUPPER					
Name	**Food description**	**Preparation ingredients**	**Household amount**	**Time**	**Amount (g/ml)**
OTHER MEALS					
Name	**Food description**	**Preparation ingredients**	**Household amount**	**Time**	**Amount (g/ml)**

5.7 Has your child ever been given multivitamin supplements?

Yes ☐ No ☐ Don't Know ☐

If yes, when did they last take the supplements? __________Don't Know ☐

5.8 Did your child take any multivitamin supplements yesterday *(Day or Night)*?

Yes ☐ No ☐ Don't Know ☐

If yes, how many times did they take the supplements? ________ ☐

5.9 Has your child ever been given micronutrient powder supplements?

Yes ☐ No ☐ Don't Know ☐

If yes, when did they last take the supplements? _________Don't Know ☐

5.10 Did your child take any micronutrient powder supplements yesterday *(Day or Night)*?

Yes ☐ No ☐ Don't Know ☐

If yes, how many times did they take the supplements? _______ ☐

6.0 Social Data

6.1 How many children do you have in the house in total? (*Including non-biological chi*☐*n*) 1 ☐ ☐ ☐ ☐
5Other, specify__________

6.2 How many occupants live in the house?

<3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ >7 ☐

6.3 What is the highest education level you have attained?

No education☐ Primary ☐ Secondary ☐ Tertiary ☐

6.4 What is the highest education level attained by your spouse?

No education ☐ Primary ☐ Secondary ☐ Tertiary ☐

7.0 Economic Data

7.1 What is your employment status *(Child's parent/Guardian)*?

Unemployed ☐ Self-employed ☐ Employed ☐

7.2 What is your spouse's occupation *(Child's parent/Guardian)*?

Unemployed ☐ Self-employed ☐ Employed ☐

7.3 What is the average monthly family income? (*In Kshs.*)

<1,000 ☐ 1,001 – 5,000 ☐ >5,000 ☐

8.0 Laboratory Results

Attach all laboratory results as per checklist *(Attach, verify then tick as applies)*

1. Full haemogram ☐
2. C-Reactive proteins ☐
3. Serum ferritin ☐
4. Serum iron levels ☐
5. Unsaturated iron binding capacity ☐

Appendix 3: Consent form - English

MOI UNIVERSITY COLLEGE OF HEALTH SCIENCES / MOI TEACHING AND REFERRAL HOSPITAL
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC) INFORMED CONSENT FORM (ICF)

Study Title: Prevalence and factors associated with Iron deficiency and inadequate zinc intake among children aged 6 – 59 months seen at Moi Teaching and Referral Hospital

Name of Principal Investigator: DR. EDWIN GUDU

Supervisor: DR. DIANA MENYA
DR. SUSAN KEINO

Name of Sponsor:
Informed Consent Form for: Parents/guardians with children aged 6 – 59 months attending Moi Teaching and Referral Hospital

This Informed Consent Form has two parts:
- Information Sheet
- Certificate of Consent

Part I: Information Sheet

Introduction: You are being requested to take part in this research study. Please read this consent form carefully. We shall answer any questions you may have regarding this study. Should you decide to participate in this study, a copy of the consent form shall be provided to you.

Participation in this study is voluntary. Refusal to participate will not affect your medical treatment at this facility. You are free to withdraw from this study at any time.

Purpose of the study:

The purpose of the study is to determine factors associated with zinc and iron deficiency among children aged 6 -59 months treated at MTRH

Type of Research Project/Intervention:

This research shall involve use of interviewer-administered questionnaires to collect information from you to try and establish factors associated with micronutrient deficiency. We shall conduct blood tests to check serum levels of zinc and iron. Whenever possible, we shall collect stool samples to check for intestinal worms.

Why have I been identified to Participate in this study?

Your child has been randomly selected to help us describe the factors associated with zinc and iron deficiency among children aged 6 -59 months treated at MTRH.

How long will the study last?

You will be involved this study only during this interaction and there shall be no follow up interviews. We shall however call you to inform you of the laboratory results from your child as soon as they are ready. For this reason we request you give us your phone number.

What will happen to me during the study?

During this study, your child's growth measurements shall be taken as is routinely done during each visit to the hospital. The only difference shall be the questionnaire that will be administered to you as well as blood and stool samples collected. Some details regarding your socio-economic status, child feeding practices will be asked during the interview. The information shall be kept private and confidential. Laboratory tests shall be carried out at MTRH. Serum samples shall be stored together with other samples and may be transferred to a separate laboratory for those tests that will not be available at MTRH.

What side effects or risks I can expect from being in the study?

There is a minimum risk of bleeding and pain during collection of blood samples. However this shall be minimized since the sample shall be collected by trained professionals.

Are there benefits to taking part in the study?

The benefits you shall get from this study include:

a) Laboratory tests related to the study will be conducted at no extra cost to you. The results will be communicated to you and your clinician who will use them for the benefit of your child in case of zinc and iron deficiency being detected.
b) Nutritional assessment of your child shall be conducted and the information relayed to you and your clinician.

Reimbursements:

There shall be no financial/cash benefits given to you for participating in this study

Whom to contact if I have questions regarding this study?

Study-related questions: DR. EDWIN GUDU 0721 911 790

Questions about your rights as a study participant: Institutional Review Ethics Committee (IREC) 053 33471 Ext.3008.

Will the information I provide be kept private?

All reasonable efforts will be made to keep your protected information (private and confidential).

Part II: Consent of Subject:

I have read or have had read to me the description of the research study. The investigator or his/her representative has explained the study to me and has answered all of the questions I have at this time. I have been told of the potential risks, discomforts and side effects as well as the possible benefits (if any) of the study. I freely volunteer to take part in this study.

____________________ ____________________ ________________

Participant's Parent/Guardian name Signature of subject/thumbprint Date & Time

(Witness to print if the subject is unable to write)

____________________ _______________________________________

Name of Representative/Witness Relationship to Subject

__________________________ ________________________ __________________

Name of person Obtaining Consent Signature Date

DR. EDWIN GUDU ________________________ __________________

Printed name of Investigator Signature of Investigator Date

Appendix 4: Consent form (Kiswahili)

MOI UNIVERSITY COLLEGE OF HEALTH SCIENCES / MOI TEACHING AND REFERRAL HOSPITAL
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC) INFORMED CONSENT FORM (ICF)

Kichwa cha Utafiti: Kueneana mambo yanayo husianana upungufu wa Zinc na Iron kati ya watoto wenye umri wa miezi 6 hadi 59 walionwa katika Hospitali ya Mafunzo na Rufaa ya Moi,

Jina la Mchunguzi Mkuu: DR. EDWIN GUDU

Wasimamizi: DR. DIANA MENYA

DR. SUSAN KEINO

Fomu ya RufaayaWazazi: Wazazi / walezi wenye Watoto wenye umri wa miezi 6 - 59 wanahudhuria Hospitali ya Mafunzo na Rufaa ya Moi

Fomu hii ya Ruhusa ya Ruhusa ina sehemu mbili:

- Karatasi ya Taarifa
- Hati ya Ruhusa

Sehemu ya Kwanza: Karatasi ya Taarifa

Utangulizi:

Taarifa hii hutolewa kuku ambia juu ya utafiti. Tafadhali soma fomu hii kwamakini. Utapewa nafasi ya kuuliza maswali. Ikiwa unaamua kuwa katika utafiti, utapewa nakala ya fomu hii ya idhini kwa rekodi zako.

Kushiriki katika utafiti huu wa utafiti ni kwa hiari yako. Unaweza kuchagua kutoshiriki katika utafiti. Kusema hapana haita athiri haki zako kwa huduma za afya. Wewe pia una huru kujiondoa kwenye utafiti huu wakati wowote.

Kusudi la utafiti:

Kusudi la utafitini kuamua mambo yanayo husiana na upungufu wa zinc na chuma kati ya Watoto wenye umri wa miezi 6 hadi 59 walio tibiwa katika hospitali ya Rufaa ya MTRH

Aina ya Mradi wa Utafiti / Uingilizi:

Utafiti huu utahusisha matumizi ya maswali ya kuhojiwa na mahojiano kukusanya habari kutoka kwako kujaribu na kuanzisha mambo yanayo husiana na upungu fu wa madini mwilini. Tutaendesha vipimo vya damu ilikuangalia kiwango cha serum ya zinki na chuma. Wakati wowote iwezekanavyo.

Kwa nini nimechaguliwa kushiriki katika utafiti huu?

Mtoto wako amechaguliwa kwanasibu kutusaidia kuelezea mambo yanayo husiana na upungufuwa zinc na chuma kati ya Watoto wenye umri wa miezi 6 hadi 59 waliotibiwa katika hospitali ya Rufaa ya MTRH.

Utafiti utaendelea muda gani?

Utashiriki masomo haya tu wakati wa maingiliano haya na hakutakuwa na mahojiano ya kufuata. Hata hivyo tutakuomba kukujulishe matokeo ya maabara kutoka kwa mtoto wako mara tu wapo tayari. Kwa sababu hii tunakuomba upe nambari yako ya simu.

Nini kita tokea kwangu wakati wa utafiti?

Wakati wa utafiti huu, kipimo cha ukuaji wa mtoto wako kitachukuliwa kama kinacho fanyika mara kwa mara wakati wa kila ziara ya hospitali. Tofauti pekee itakuwa swali ambalo litasimamiwa kwako pamoja na sampuli za damu zilizokusanywa. Maelezo mengine kuhusu hali yako ya kijamii na kiuchumi, utaratibu wakulisha Watoto utaulizwa wakati wa mahojiano. Taarifa hiyo itahifadhiwa binafsi na ya siri. Uchunguzi wa maabara utafanyika katika MTRH. Sampuli za Seramu zitahifadhiwa pamoja na sampuli zingine na zinaweza kupelekwa kwenye maabara tofauti kwa ajili ya vipimo hivi ambavyo hazipatikani kwenye MTRH.

Je, ni madhara gani au hatari ambazo ninaweza kutarajia kutoka kwenye utafiti?

Kuna hatari ndogo ya kutokwa damu na maumivu wakati wa kukusanya sampuli za damu. Hata hivyo hii itapunguzwa tangu sampuli itakusanywa na wataalamu wenye mafunzo.

Je, kuna faida ya kushiriki katika utafiti?

Faida unazo pata kutoka kwa utafiti huu ni pamoja na:

a) Uchunguzi wa maabara kuhusiana na utafiti utafanyika bila gharama yoyote. Matokeo yatatumiwa kwa daktarin wako ambaye atatumia kwa manufaa ya mtoto wako ikiwa kuna uhabawa zinki na chuma.

b) Tathmini ya lishe ya mtoto wako itafanywa na taarifa iliyotumwa kwako na daktari wako.

Malipo:

Hakuta kuwa na faida za kifedha / fedha ambazo zimepewa kwa kushiriki katika utafiti huu

Nitaita nani ikiwa nina maswali juu ya utafiti?

Kwa maswali kuhusu utafiti**:** DR. EDWIN GUDU 0721 911 790

Maswali kuhusu haki zako kama mtutu anayehusika kwenye utafiti: Unaweza kuwasiliana na Kamati ya Maadili ya Ukaguzi wa Taasisi (IREC) 053 33471 Ext.3008.

Je! Habari nitayayoyatoa itawekwa ya faragha?

Jitihada zote za busara zitafanywa ilikuhifadhi maelezo yako ya ulinzi (binafsi na ya siri.)

Sehemu ya II: kibali cha suala:

Nimesoma au nimesomewa maelezo ya utafiti. Mpelelezi au mwakilishi wake ameelezea utafiti kwangu na amejibu maswali yote niliyo nayo wakati huu. Nimeambiwa juu ya uwezekano wa hatari, wasiwasi na madhara pamoja na faida iwezekanavyo (kamaipo) ya utafiti. Mimi kujitolea kwa hiari kushiriki katika utafiti huu.

____________________________ ____________________________ _________________

Jina la Mzazi / Mleziwa Mshirika / Sahihi ya Kitambulisho / Kitambulisho cha Muda na Muda

(Shahidi kuchapisha ikiwa mzazi/mlezi wa mtoto hawezi kuandika)

____________________________ ____

Jina la Mwakilishi / Uhusianowa Shahidi kwa mzazi/mlezi wa mtoto
____________________________ _________________________ ___________________

Jina la mtu aliyepata idhini ya Sahihi Saini Tarehe

DR. EDWIN GUDU _________________________ ___________________

Jina la kuchapishwa la Mpelelezi Saini Tarehe ya Upelelezi

Appendix 5: Developmental Milestones

Developmental Milestone	Normal Limits
Social Smile	4-6 weeks
Head holding/Control	1-3 months
Turn towards origin of sound	2-3 months
Extend hand to grasp toy	2-3 months
Sitting	5-9 months
Standing	7-13 months
Walking	12-18 months
Talking	9-24 months

Adapted from Ministry of Health (Kenya), Mother and Child Booklet MOH 216 (Ministry of Health - Kenya, 2010)

Appendix 6: Immunization schedule (Kenya)

VACCINE	AGE OF CHILD/ADMINISTRATION
BCG	At birth or at first contact
OPV (Birth Dose)	At birth or at first contact (Within first two weeks of life)
OPV I	At 6 weeks of life or at first contact
Pentavalent I	
PCV-10 I	
ROTA I	At 6 weeks or at first contact < 1yr
OPV II	At 10 weeks or 4 weeks after OPV I and Pentavalent I
Pentavalent II	
PCV-10 II	
ROTA II	At 10 weeks or 4 weeks ROTA I
OPV III	At 14 weeks or 4 weeks after OPV II and Pentavalent II
Pentavalent III	
PCV-10 III	
IPV	
Measles I	At 9 months or at first contact after 9 months of age
Measles II	At 18 months or at firs contact after 18 months of age

Adapted from training manual for introduction of rotavirus vaccine in Kenya (Ministry of Health - Kenya, 2014)

Appendix 7: Project Work-plan/Project Timelines

Objective	Measure(s) of Success/ Key Milestones	Activities	Month that activity will begin & end (Starting January 2022)			
			1ST Quarter	2ND Quarter	3RD Quarter	4TH Quarter
To seek ethical approval and permission to carry out the study	1. Permission for the study given	1. Submit project protocol to IREC 2. Sensitize the Hospital Management on the project				
To carry out data collection and cleaning	1. Research assistants recruited 2. Clean set of data	1. Recruit and train research assistants 2. Data Collection 3. Data Verification and inconsistencies rectified 4. Data quality audit				
To carry out data analysis	1. Adequately analyzed data	1. Data analysis using excel and other nutrition and statistical analysis software				
To disseminate findings to relevant stakeholders for action	1. Research findings communicated to the stakeholders 2. Publish research findings	1. Sensitize the hospital administration on findings and recommendations 2. Develop a manuscript 3. Present findings at scientific conference				

Appendix 8: IREC Approval

INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471//2/3

MOI UNIVERSITY
COLLEGE OF HEALTH SCIENCES
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

17th July, 2019

Reference: IREC/2019/109
Approval Number: 0003375

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
17 JUL 2019
APPROVED
P. O. Box 4606 - 30100 ELDORET

Dr. Edwin Gudu & Team,
Moi Teaching and Referral Hospital,
P.O. Box 03-30100,
ELDORET-KENYA.

Dear Dr. Gudu & Team,

PREVALENCE AND FACTORS ASSOCIATED WITH ZINC AND IRON DEFICIENCY AMONG CHILDREN AGED 6-59 MONTHS SEEN AT MOI TEACHING AND REFERRAL HOSPITAL, 2019

This is to inform you that ***MU/MTRH-IREC*** has reviewed and approved your above research proposal. Your application approval number is ***FAN:0003375.*** The approval period is ***17th July, 2019 – 16th July, 2020.***

This approval is subject to compliance with the following requirements;

i. Only approved documents including (informed consents, study instruments, MTA) will be used.
ii. All changes including (amendments, deviations, and violations) are submitted for review and approval by ***MU/MTRH-IREC***.
iii. Death and life threatening problems and serious adverse events or unexpected adverse events whether related or unrelated to the study must be reported to ***MU/MTRH-IREC*** within 72 hours of notification.
iv. Any changes, anticipated or otherwise that may increase the risks or affected safety or welfare of study participants and others or affect the integrity of the research must be reported to ***MU/MTRH-IREC*** within 72 hours.
v. Clearance for export of biological specimens must be obtained from relevant institutions.
vi. Submission of a request for renewal of approval at least 60 days prior to expiry of the approval period. Attach a comprehensive progress report to support the renewal.
vii. Submission of an executive summary report within 90 days upon completion of the study to ***MU/MTRH-IREC.***

Prior to commencing your study, you will be expected to obtain a research license from National Commission for Science, Technology and Innovation (NACOSTI) https://oris.nacosti.go.ke and also obtain other clearances needed.

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc	CEO	-	MTRH	Dean	-	SOP	Dean	-	SOM
	Principal	-	CHS	Dean	-	SON	Dean	-	SOD

MTRH/MU-INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471//2/3

MOI UNIVERSITY
COLLEGE OF HEALTH SCIENCES
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

Reference: IREC/2019/109
Approval Number: 0003375

7th February, 2022

Dr. Edwin Gudu & Team,
Moi Teaching and Referral Hospital,
P.O. Box 03-30100,
ELDORET-KENYA.

Dear Dr. Gudu & Team,

RE: APPROVAL OF AMENDMENT

The Moi Teaching and Referral Hospital/Moi University College of Health Sciences- Institutional Research and Ethics Committee has reviewed the amendment made to your proposal titled:-

"Prevalence and Factors Associated with Iron Deficiency and Inadequate Dietary Zinc Intake among Children aged 6-59 Months at Moi Teaching and Referral Hospital, Kenya"

We note that you are seeking to make amendments as follows:-

- To change the title to above from ***"Prevalence and Factors Associated with Zinc and Iron Deficiency among Children Aged 6-59 Months seen at Moi Teaching and Referral Hospital, 2019"***
- To align the broad objective and specific objectives (1, 3 and 4) with the title hence looking at the aspect of inadequate dietary zinc intake instead of zinc deficiency as in the previous protocol.
- To add additional literature showing utility of inadequate dietary zinc intake in the assessment for risk of zinc deficiency among children aged 6-59 months.
- To remove protocols for serum zinc sample collection and laboratory standard operating procedures on this test.
- To remove serum zinc tests results and to redesign the 24-hour dietary recall tool for ease of use.

The amendments have been approved on 7th February, 2022 according to SOP's of IREC. You are therefore permitted to continue with your research.

You are required to submit progress(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.

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
07 FEB 2022
APPROVED
P. O. Box 4606-30100 ELDORET

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc:	CEO	-	MTRH	Dean	-	SPH	Dean	- SOM
	Principal	-	CHS	Dean	-	SOD	Dean	- SON

Appendix 9: MTRH Approval

An ISO 9001:2015 Certified Hospital

MOI TEACHING AND REFERRAL HOSPITAL

Telephone :(+254)053-2033471/2/3/4
Mobile: 722-201277/0722-209795/0734-600461/0734-683361
Fax: 053-2061749
Email: ceo@mtrh.go.ke/directorsofficemtrh@gmail.com

Nandi Road
P.O. Box 3 – 30100
ELDORET, KENYA

Ref: ELD/MTRH/R&P/10/2/V.2/2010

18th July, 2019

Dr. Edwin Gudu & Team,
Moi Teaching and Referral Hospital,
P.O. Box 03-30100,
ELDORET-KENYA.

APPROVAL TO CONDUCT RESEARCH AT MTRH

Upon obtaining approval from the Institutional Research and Ethics Committee (IREC) to conduct your research proposal titled:-

"Prevalence and Factors Associated with Zinc and Iron Deficiency among Children Aged 6-59 Months seen at Moi Teaching and Referral Hospital, 2019".

You are hereby permitted to commence your investigation at Moi Teaching and Referral Hospital.

MOI TEACHING AND REFERRAL HOSPITAL
CEO
APPROVED
18 JUL 2019
SIGN..........
P. O. Box 3 - 30100, ELDORET

18/07/2019

DR. WILSON K. ARUASA, *MBS*
CHIEF EXECUTIVE OFFICER
MOI TEACHING AND REFERRAL HOSPITAL

cc - Senior Director, (CS)
- Director of Nursing Services (DNS)
- HOD, HRISM

All correspondence should be addressed to the Chief Executive Officer
Visit our Website: www.mtrh.go.ke
TO BE THE LEADING MULTI-SPECIALTY HOSPITAL FOR HEALTHCARE, TRAINING AND RESEARCH IN AFRICA

Appendix 10: Nacosti License

REPUBLIC OF KENYA

NATIONAL COMMISSION FOR SCIENCE,TECHNOLOGY & INNOVATION

Ref No: **807783**

Date of Issue: **03/September/2019**

RESEARCH LICENSE

This is to Certify that Dr.. Edwin Gudu of Moi University, has been licensed to conduct research in Uasin-Gishu on the topic: Prevalence and factors associated with zinc and iron deficiency among children aged 6 – 59 months seen at Moi Teaching and Referral Hospital, 2019 for the period ending : 03/September/2020.

License No: **NACOSTI/P/19/664**

807783

Applicant Identification Number

Director General
NATIONAL COMMISSION FOR SCIENCE,TECHNOLOGY & INNOVATION

Verification QR Code

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