RISKS AND CAUSATIVE FACTORS OF RICKETS IN IN-PATIENT CHILDREN AGED BETWEEN 6-59 MONTHS: A CASE STUDY OF KENYATTA NATIONAL HOSPITAL, NAIROBI.

BY

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DECLARATION

Isabella Atieno Onyango hereby declare that this is my original work and has not been presented for a degree or any other awards in any other University

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This dissertation has been submitted for examination with my approval as University Supervisor

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DEDICATION

This work is dedicated to my loving husband Mr. George Odingo.

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ST OF ABBREVIATIONS

A.R I	Acute Respiratory Disease
BMI	Body mass index
D.D	Diarrheal Disease
DXA Scan	Bone densitometry which measures calcium content.
FGD	Focused Group Discussion
GAM	Global Acute malnutrition
I.D.H	Infectious Disease Hospital
KNH	Kenyatta National Hospital
MUAC	Mid upper arm circimference
NR	Nutritional status
UNICEF	United Nation Children fund
U.O.N	University of Nairobi
W .H.O	World Health Organization
VAD	Vitamin D deficiency
25-(OH0)	concentration of circulating Vitamin D

DEFINATION OF TERMS

1. **Rickets** Rickets is the softening and weakening of bones in children usually because of extreme and prolonged vitamin D deficiency and calcium
2. **Risk factors** Pre-disposing factors to the disease condition.
3. **Vitamin** Is an organic compound required as a nutrient in small amounts by an organism for growth and maintains good health.
4. **Nutritional rickets** A deficiency disease resulting from lack of vitamin D or Calcium and from exposure to sunlight characterized by defective borne growth and occurring readily in children.
5. A **mineral** is a naturally occurring homogenous inorganic solid substance having a definite
   chemical composition and a characteristic crystalline structure, color and hardness.
6. **Calcitriol**-Active vitamin D
7. **Rachitic** Inflammation of the spine
8. **Wasting** Weight below -2.000 Z- score
9. **Underweight** weight below -2.000 Z score
10. **Stunting** weight below -2.000Z-score
11. **Hypophosphatemic rickets** Rickets caused by low phosphorous

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ABSTRACT.

ckets has become a significant problem, which needs special attention. The objective of the study to determine factors contributing to rickets in children aged 6-59 months. This was with erence to Malnutrition, socio-economic status and some causative factors such as type of food fed the children, exposure to sunlight, Heredity, birth order and birth condition. The study involved children in-patient in Kenyatta National Hospital, Level 3 wards A, B, C and D.

is study was Retrospective, cross sectional and analytic cohort. The study instruments and tools nsisted of a structured questionnaire .This was administered to all respondents of studied children , luding personal data, type of feeding,socio- economic data, exposure to sunlight and other risk tors to rickets.

sults indicated that Malnutrition was high. Global Acute Malnutrition was 63%.Those who were derweight were 69.9%, Stunting 14% and underweight 64%.There was relationship between ding children with spinach and stunting (odds ratio 4.11), p-value 0.006.Feeding of children nach and wasting was (odds ratio 1.25, p-value 0.0051, Children were also fed on mixed fours, ich contained different ingredients .Consumption of mixed floors (O.R= 1.402, p-value 0.006) eding of children on foods rich in calcium was poor. Feeding on Amaranth was 67%, cyprinids s 11.1%, cowpeas 12.5%.There was a correlation between spinach and stunting, and also derweight.

cio- economic condition was poor in terms of education, 50% were housewives and education was to secondary schools was 67%.Household sizes was 5-8 ,which was high at 67%. All these ntributed to the children's condition.

e exposure to sunlight which was at 81.9%, this was high though the children developed rickets the her risk factors were Heredity, birth condition and birth order. This study did not show any lationship between children developing rickets and heredity, birth order or birth condition.

is study concluded that the main causative factors of rickets was socio-economic and socio - mographic situation of mothers, Nutritional status of children, non exposure to sunlight and ding practices of the children's care givers.

CHAPTER ONE: INTRODUCTION

1.1 Background Information

It was only in the early part of the 20th century with the discovery of vitamin D and of the role of ultraviolet light radiation plays in vitamin D formation. When rational and appropriate therapy became available and rickets was all but eradicated in a number of developed countries (Pettifor, 2008). Since then, there has been a resurgence of the disease in many countries including those in the developed world, such as in Europe and the USA. (Pettifor, 2008). Probably this is due to an increase in the prevalence of breast feeding, the immigration of dark skinned families to countries of high latitude, and the avoidance of direct sunlight because of the risk of development of skin cancers.(Pettifor,2008).The disease is also widely recognized in many developing countries, including some situated in subtropical region. Studies have led to the realization that nutritional rickets may be caused by either vitamin D or calcium deficiency, but in the majority of situations combinations of both probably play a role. Although low dietary calcium intakes appear to be central to the pathogenesis of rickets as reported in Nigeria and other African countries genetic and other environmental factors are likely to contribute (Prentice, 2008). But to date no single factor has been isolated as contributing significantly. (Prentice, 2008).

The results of a recently conducted study suggest that in situation of low dietary calcium intakes, vitamin D requirements may be higher than normal, possibly predisposing those children with vitamin D levels lower than normal range, to rickets (Chen, 2008). If this is so, it would indicate that the currently accepted normal range for vitamin D sufficiency would need to be adjusted depending on dietary calcium intakes. Nutritional rickets is gaining the attention of public health professionals and individual clinicians worldwide as the disease remains an endemic problem in many developing countries and

has re-emerged in a number of developed countries, where it was thought that the disease had been almost eradicated. (Hollick,2006). Interest has been heightened by the considerable discussion currently taking place about what should be the ideal or appropriate circulating levels of 25-hydroxyvitamin D [25(OH) D] to reflect vitamin D sufficiency.(Prentice, 2008).

Vitamin D deficiency is clearly the major cause of nutritional rickets in countries lying at high latitudes both north and south of the equator. (Ward,2001).

It has been shown convincingly that vitamin D production in the skin is negligible at latitudes greater than 35° during November through March in the northern hemisphere (Petitifor,2008). Similar findings have been reported from Cape Town, South Africa (32° S) from May through August. This is due to decreased dermal synthesis of vitamin D as a result of the absorption of UV radiation and the increased melanin pigmentation. (Ward, 2008). However of interest is one study which was conducted in the north-eastern part of the USA. In that study, low dietary calcium intakes in mainly African American infants who developed rickets after weaning, were considered to play a major role as 25(OH) D concentrations were above generally accepted levels of vitamin D deficiency. (Ward, 2008).

A similar mechanism has been proposed in Indian and Pakistani children living in the UK, whose diets are typically low in calcium and high in phytate. (Pettifor, 2004), in the majority of these south Asian children with rickets, 25(OH) D concentrations are low (Pettifor, 2008). The net effect is an increase in vitamin D requirements which, if not met, results in a reduction of 25(OH) D concentrations into the vitamin D deficiency range (Pettifor, 2008). Thus in these children in the UK, rickets is induced by the combined effect of low dietary calcium intakes and vitamin D deficiency. (Pettifor, 2008) Studies in

this group indicate that the disease is effectively cured by increasing the vitamin D status of affected children, although the removal of the high phytate content of the diet has also been shown to be beneficial with vitamin D supplementation. (Pettifor, 2004).

The discovery of essential nutrients and their roles in disease prevention has been instrumental in almost eliminating nutritional deficiency diseases such as rickets and others in the World. (Belashew,2008). Bone serves as the reservoir for 99 percent of the body's total calcium. Calcium is therefore an essential nutrient for bone health. (Belashew,2008). Calcium is also needed for the heart muscles and nerves to function properly and for blood to clot normally. The body loses calcium every day through, urine, feces, sweat, the skin, the hair and nails. The lost calcium is normally replaced by calcium in the diet. (Belashew, 2008). When the diet does not contain sufficient calcium to offset such losses, the body breaks down bone to release the calcium needed to accommodate these physiologic demands (Belashew, 2008). Rickets can be due either to primary deficiencies of vitamin D or calcium or to combined deficiencies of both elements. Even without laboratory and radiologic resources, the diagnosis of rickets is easy. (Belashew, 2008). The symptoms include limb deformities, beaded ribs and widened wrists and ankles. Prevention is possible through increased sun exposure and dietary enhancement of vitamin D and calcium (Pettifor, 2004).

Rickets is increasingly recognized as a failure of health education and health care delivery in many countries (Kinuthia, 2003). Rickets is a crippling vitamin deficiency that Kenyans should not suffer from. This is commonly happening in the country's urban centers, as mothers and children shift to indoor lives, resulting in weakened bones, fractures and deformities. (Kinuthia, 2003). Hospitals have reported a surge in cases of rickets in the last five years. Naivasha District Hospital is now recording 100 cases

monthly, while Kenyatta National Hospital, in Nairobi, is recording two to three cases of rickets daily. (Bwibo, 2003).

The deficiency in vitamin D and calcium softens young children's bones, and often causes deformities of the feet and legs and gets children permanently crippled. This is attributed to the sudden rise of the expansion in high-rise estates in Nairobi. 'Our research shows that children growing up in such places have little sun exposure, which is the only natural source of Vitamin D'. The other concern is clothing of children during cold season. Children are over-clothed unnecessarily preventing skin exposure. Huruma estate was one of the worst hit areas of Nairobi by rickets (Bwibo, 2003), because buildings are congested and some are permanently under shadows. Mothers in higher floors rarely take their children down to bask in the sun. However this may be debated upon because the Sunlight rays required is minimal. (Bwibo, 2003) it was recommended by the food and nutrition board of the institute of medicine of united state of American is 200 international units/per day for adults and 5 ug/day for infant. (Foods and Nutrition board)

Twenty-nine patients with rickets were studied in a one-year period. (Kinuthia,2003). The majority of patients (17) were below 2 years of age. Most of them had rickets resulting from a combination of factors including premature delivery, non-exposure to sunlight, and inappropriate dietary intake. Some had familial hypophosphatemic rickets; others had renal tubular acidosis, while the rest had rickets with a familial tendency (Kinuthia, 2003). Hospital records indicate that rickets is a persistent problem in children in Kenya and should be suspected in children who present themselves with features of failure to thrive, among other conditions. (Nyakundi,1994).

1.2. Problem Statement

Hospitals in Kenya are recording cases of rickets almost daily. For example Naivasha District Hospital receives close to 100 new cases monthly. Similar problem also has been observed in Kenyatta National hospital where 2-3 ricketsia cases are observed every day. (Bwibo, 2003) This problem continues to be a challenge in the whole country despite the availability of sunshine in plenty most months of the year. The disease affects mainly children, as well as adults. This disease is related partly to poverty in vulnerable groups, low income earners and status of education for mothers and maternal and child health care among others. An example is seen in Naivasha flower farms where mothers work long hours in the flower farms and leave their babies in the day care centers. In the daycare centre the children are kept indoors throughout the day. Most of this develops rickets.

1.3 Justification

Rickets has become an increasingly significant problem, which needs special attention. Many children are coming to hospital showing signs which have been diagnosed as Rickets .This study will help to unveil some of the causes of rickets. Some scholars state that the disease is due to children non exposure to sunlight, others believe it is because of children being born underweight, and others believe it is because of malnutrition. This study is based in Kenyatta national hospital because this is a referral hospital with patients coming from all over the country. It will help to give indication of the spread of the problem in the country.

1.4 Main Objective

To establish causative factors of Rickets in in-patient children aged between 6 -59months.

1.5 Specific Objectives

1. To assess the nutritional status of these in-patient children.
2. To determine the socio-demographic and socio-economic status of the children's care givers or mothers, in relation to the children's status.
3. To determine the prevalence of rickets among the in-patient children aged 6-59 months.
4. To determine risk factors to rickets in the children.

1.6 Hypotheses

1. There is no significant relationship between Rickets and nutrition status in children 6-59.
2. There is no relationship between rickets and the risk factors.

1.7 Expected benefits of the project

The findings of this study are expected to be of primarily assistance to the Ministry of Health. It is expected that highlighting the information on gaps in the relationship of Rickets and dietary management of children will aid in improving the quality of nutrition care for children aged 6 and 59 months. Knowing the causative factors of rickets will assist in management of children with rickets.

It will also assist health institution planners and training institution e.g. Government and NGO's interested in working with the communities in training mothers to take care of children to avoid the condition. This study will contribute to the understanding of risk and causative factors of rickets so as to reduce the cost of treating the disease for the Government and save the overstretched bed capacity of hospital beds.

CHAPTER TWO: LITERATURE REVIEW

2.1 Rickets an overview

Rickets, the bone disease classically caused by vitamin D deficiency, was one of the most common diseases of children 100 years ago (Hollick,2006). It has been recognized as a disease of urban living and linked to issues of race and culture for generations (Hollick, 2006). Before 1910, as evidenced by patient records, neither the diagnosis nor the treatment of rickets had been standardized (Hollick, 2006). The disease frequently was presented as a disease of African Americans or Italian immigrants and used to reinforce racial stereotypes, promote the assimilation of immigrants into majority cultures, and call for behavioral change. In the second and third decades of the 20th century, as clinicians and scientists unraveled the twin roles of diet and sunlight exposure in the disease's etiology, both diagnosis and treatment became more standardized (Hollick,2006). But this standardization including exchanging bedside diagnosis for X-ray technology and promoting general preventive measures altered the perceived prevalence and even the definition of the disease. By the mid-1920s, rickets was promoted as universal, at times invisible, to non-experts, but present to some degree in nearly every young child regardless of race or class. It was thus used to promote the young disciplines of preventive medicine, pediatrics, and public health. (Hollick, 2006) .

Rickets therefore provides an excellent window into the early politics of preventive health in the United States and a relevant historical counterpoint for current debates over the role of race and ethnicity as risk factors for disease, the use of diagnostic technology in defining disease, and the promotion of targeted interventions for today's so-called "lifestyle" diseases. (Warner, 2008).

Rickets has been reported in dozens of countries during the past three decades in some places. Nutritional rickets is merely reported sporadically, while in. other areas, up to 9 percent of the childhood population is clinically affected. Rickets in Bangladesh was first brought to broad attention in 1991 by workers from Social Assistance of physically Rehabilitation, visiting the Chakaria region of southeastern Bangladesh after a devastating cyclone (Prentice, 2007). An informal village survey found that approximately 1 percent of children had rachitic deformities. Focus groups and local informants suggested that rickets was 'new' and had not been seen before the early 1970's. Children in Chakaria received care at the Memorial Christian Hospital, and 441 children with rickets were registered during 1991–1997. Anecdotally, of medical treatment with vitamin D were disappointing, and deformities recurred in children subjected to orthopedic surgery (Prentice, 2007).

Nutritional rickets has been described from at least 59 countries in the last 20 years. Its spectrum of causes differs in different regions of the world. A study was conducted to systematically review articles on nutritional rickets from various geographical regions published in the last 20 years (Thatcher, 2006).

Calcium deficiency is the major cause of rickets in Africa and some parts of tropical Asia, but is being recognized increasingly in other parts of the world. A resurgence of vitamin D deficiency has also been observed in North America and Europe. (Thatcher, 2006).Vitamin D-deficiency rickets usually presents in the first 18 months of life, whereas calcium deficiency typically presents after weaning and often after the 2nd year (Thatcher, 2006).

Few studies of rickets in developing countries report values of 25(OH) D to help distinguishing vitamin D from calcium deficiency (Thatcher, 2006). Rickets exists along a spectrum ranging from isolated vitamin D deficiency to isolated calcium deficiency.(Thatcher,2006).Vitamin D supplementation alone

might not prevent or treat rickets in populations with limited calcium intake (Thatcher, 2006).

In 1994, French physicians, with Les Amis des Enfants du Monde, evaluated patients in communities from Chittagong to Moheskhali and identified rickets in 4.5percent of them. In 1997, academicians from Cornell University and other American institutions were appraised of the situation. A collaborative assessment revealed that rickets was more common than suspected in Chakaria, this was not generally associated with vitamin D deficiency but was related to insufficiency of dietary calcium. (Prentice,2008).

An international 'Rickets Consortium' was formed to stimulate collaborative research and practical interventions. This group subsequently reformed as the current Rickets Convergence Group which serves as a repository of information and a source of expertise to facilitate ongoing work relating to rickets in Bangladesh. (Craviari,2004).

The Helen Keller International conducted a nationwide survey in 2000 and repeated it in 2004. (craviari,2004). Rickets was identified as visible deformities in children aged 1–15 year. (Craviari,2004). Nationally rachitic deformities were found in 0.26 percent of 21,571 surveyed children in 2000 and in 0.12 percent of 10,005 surveyed children in 2004. Rickets was found in more than half of the sub- districts with the highest prevalence being found in Sylhet (North-East) and Chittagong (South-East) divisions. The highest prevalence (1.4%) among 1 to 15 year(s) old children with visible rachitic deformities was found in the Cox's Bazaar sub- district. A survey of all inhabitants in Chittagong carried out by the Bangladesh Rural Advancement Committee found rachitic deformities in 0.9 percent. (Craviari 2004). A more detailed survey conducted by the Institute of Child and Mother Health in the Chittagong division found that 8.7 percent of children had at least one clinical finding indicative of rickets, 4 percent had deformities of the lower limb suggestive of rickets, 0.9 percent had radiological evidence of active rickets,

and 2.2 percent had elevated levels of serum alkaline phosphates. (Craviari, 2004).

2.2.1 Causes of rickets

The predominant cause is a vitamin D deficiency, but lack of adequate calcium in the diet may also lead to rickets. Although it can occur in adults, the majority of cases occur in children suffering from severe malnutrition usually resulting from famine starvation during the early stages of childhood (Belashew, 2008). Osteoporosis is the term used to describe a similar condition occurring in adults, generally due to a deficiency of vitamin D. The origin of the word "rickets" is probably from the Old English dialect word wrickken', to twist. The Greek derived word "rachitis" (ραχίτις, meaning "inflammation of the spine") was later adapted as the scientific term for rickets (Belashew, 2008).

Food intake and dietary patterns in Kenyan households have been studied since the 1920s. Reports on breastfeeding, nutrient intake, micronutrient deficiencies and the impacts of malaria and intestinal parasites on nutritional status were reviewed (Orinda, 1994)). Diets are mainly cereal-based, with tubers and a variety of vegetables and fruits when available (Orinda, 1994). White maize, sorghum and millet are high in phytate and fiber, which inhibit the absorption of micronutrients such as zinc and iron and calcium. (Charlotte, 2003).

It has been observed that High phytate will impair intestinal absorption of dietary calcium and may be the main cause of rickets globally. (Abdulbari, 2010). In comparison to non-exclusive breastfeeding, exclusive breastfeeding was more associated with rickets. (Abdulbari, 2010). Human milk contains a vitamin D concentration of ≤25 IU/L making it a low source of vitamin D (Lammi-Keefe, 1995). Infants who are breast-fed but do not receive supplementation of vitamin D or adequate sunlight exposure are at increased

risk of developing rickets. (Lammi-Keef, 1995).It was recommended that all breast-fed infants be given supplementation with vitamin D in case there was less exposure to sunlight. (Lawrence,2003). It is interesting to find out that rickets was more frequent even among children who were breast-fed for more than one year which is a long time breastfeeding. Exclusive breastfeeding beyond six months without vitamin D supplementation was identified as a risk factor for rickets in children. . In Alaska, the contribution of breastfeeding to vitamin D deficiency has likely increased in recent years with an increase in the proportion of women who breast feed longer than six months from 28 percent of infants during 1990 to 50 percent during 2000.
(Middaugh, 2003).

Rickets is a disease of growing bone that is unique to children and adolescents. It is caused by a failure of asteroid to calcify in a growing person-(Getanea 2008). Failure of asteroid to calcify in adults is called osteoporosis. (Getanea, 2008). Vitamin D deficiency rickets occurs when the metabolites of vitamin D are deficient. Less commonly, a dietary deficiency of calcium or phosphorus may also produce rickets (Getanea,2008). Vitamin D-3 (cholecalciferol) is formed in the skin from a derivative of cholesterol under the stimulus of ultraviolet-B light (Getanea ,2008).Ultraviolet light or cod liver oil was the only significant source of vitamin D until early in the 20th century when ergosterol (vitamin D-2) was synthesized from sun (Getanea,2008). Rickets is a rare disease that affects bone development in children It causes the softening and weakening of bones, which can lead to deformities, such as bowed legs and curvature of the spinal. The most common cause of rickets is a lack of vitamin D or calcium in the diet (Getanea, 2008). These minerals are essential for a child to form strong healthy bones. Less commonly, children can be born with genetic forms of rickets or develop rickets if underlying conditions affect the absorption of

vitamins and minerals. For most children, additional vitamin and mineral supplements can usually treat rickets successfully. (Getanea, 2008).Rickets is among the most frequent childhood diseases in many developing countries. (Getanea, 2008). Calcium is said to be abundant in spinach and other green leafy vegetables but spinach contains anti -nutrient called oxalate which binds calcium and prevents its absorption in to human body. (Getanea, 2008), it prevents its absorption by 95 percent and only 5 percent absorption .Oxalate is common on many vegetable products for example soy has high content of oxalate. (Nielsen, 2003).

Growth plate thickness is determined by two opposing processes, chondrocyte proliferation and hypertrophy on one hand and vascular invasion of the growth plate followed by conversion into primary bone spongiest on the other. Vascular invasion requires mineralization of the growth plate cartilage and is delayed or prevented by deficiency of calcium or phosphorus (Stamp, 2005). . In these circumstances, growth plate cartilage accumulates and the growth plate thickens. In addition, the chondrocytes of the growth plate become disorganized, losing their regular straight-columned orientation. In the bone below the growth plate (metaphysic), the mineralization defect leads to the accumulation of asteroid (Stamp, 2005).

These abnormalities decrease the biomechanical resistance of the involved skeletal sites, leading to a secondary increase in the diameters of the growth plate and metaphysic (Stamp, 2005). These changes may be regarded as an attempt to compensate for decreased bone strength by increased bone size. Nonetheless, bone stability is compromised and if the underlying condition Does not improve, bowing occurs. (Stamp,2005).

Hypocalcaemia and hypophosphate rickets manifest initially at the distal forearm, knee, and costochondral junctions. They are the sites of rapid bone

growth, where large quantities of calcium and phosphorus are required for mineralization (Stamp, 2005).

2.2.2 Methods of Diagnosing Rickets

The following are some of the symptoms expected from those suffering from rickets, Pain in bones, skeletal deformities such as soft skull bones, bowed legs, curvature of the spine, thickening of the ankles and wrists and knees. This may include breastbone sticking out which is sometimes known as 'pigeon chest'. Fragile bones become weaker and more prone to fractures. Poor growth and development may cause the bones of the skeleton not to grow and develop properly. Dental problems such as a delay in teeth coming through and weak tooth enamel. For older children symptoms of rickets may also include waddling when walking, bending bones, muscle weakness and pain (Kinuthia1994)

Rickets is most commonly caused by lack of vitamin D or calcium in children. Both of these are essential for a child to develop healthy bones (Pettifor, 2008). A deficiency of vitamin D over a long period causes rickets in children (Pettifor, 2008). Among humans, one of the well-studied communities with a high prevalence of rickets has been the Asian community in the United Kingdom (Pettifor 2008). Since the early 1960s, numerous studies have highlighted the predisposition of this community to rickets and osteomalacia. Several pathogenetic mechanisms have been proposed, including lack of sunlight exposure, increased skin pigmentation, lack of dietary vitamin D intake, genetic predisposition, low-calcium diets, and high phytate contents in the diet (Pettifor 2008). In a rat model, it was possible to demonstrate that elevation of 1, 25$(OH)_2$ D concentrations through feeding of the rats with low-calcium or high-phytate diets resulted in increased catabolism of 25(OH)D to inactive metabolites and increased excretion of these products in the stool, with resultant reduction of 25(OH)D concentrations (Pettifor 2008) .

Similarly, infusion of 1, 25(OH)$_2$ D led to a reduction in the serum 25(OH) D half-life and a 7-fold increase in 24 , 25-dihydroxyvitamin D production by the kidney . In human studies, the half-life of 25(OH) D was reduced by nearly 40 percent among patients with partial gastrectomies, secondary hyperparathyroidism, and elevated 1, 25(OH)$_{2D}$ concentrations and similar findings were noted among patients with intestinal mal-absorption and, subjects consuming high-fiber diets (Pettifor 2008). The administration of 1, 25(OH)$_{2D}$ to normal subjects were shown to reduce the circulating 25(OH) D half-life and to induce vitamin D deficiency among those with relatively low 25(OH) D concentrations. (Hoflman, 2010)

Therefore, the pathogenesis of rickets in the Asian community in the United Kingdom is attributable to the high-cereal, low-calcium diet, which induces mild hyperparathyroidism and elevation of 1, 25(OH)$_{2D}$ concentrations, with a resultant reduction in vitamin D status (Pettifor, 2008). In situations in which the vitamin D status is marginal, because of reduced sun exposure, increased skin pigmentation, and limited dietary vitamin D intake, the reduction in 25(OH) D half-life is sufficient to produce vitamin D deficiency and rickets (Pettifor 2008). It follows that rickets in the Asian community can be treated either by increasing the vitamin D intake or by reducing the phytate content of the diet. Both of these treatments have been found to be effective. (Pettifor,2008). From this discussion, it is clear that the pathogenesis of nutritional rickets, a disease once thought to be attributable solely to vitamin D deficiency, should be viewed as having a spectrum of mechanisms, with classic vitamin D deficiency, as observed among breast-fed infants, at one end and dietary calcium deficiency, as typified by the children studied in Nigeria and South Africa, at the other. Between these 2 extremes, it is likely that vitamin D insufficiency and low dietary calcium or high phytate intakes combine to induce vitamin D deficiency and rickets, which may be the most frequent cause of rickets globally.

2.2.3 Understanding Rickets

Rickets is more common in children from Asian, African Caribbean and Middle Eastern origin because their skin is darker and needs more sunlight to get enough vitamin D (Michael, 2007). However, any child who does not go outside very often, is frequently covered up or has a diet low in vitamin D or calcium, can also be at risk (Michael, 2007).There are other types of rickets caused by Genetic defect.Hypophosphatemic rickets involves a genetic defect that causes abnormalities in the way the kidneys and bones utilizes phosphate (Chen 2008). This leaves too little phosphate in the blood and bones, which leads to weak and soft bones. (Michael, 2007). Hereditary rickets is an abnormal gene .This gene is on one of the sex chromosomes (X) chromosome. If a mother with with two X chromosomes had a faulty gene, she can pass it on to both her sons and daughters. A father (who has one X and one Y chromosome with the faulty gene can only pass it on to a son (Michael, 2007). Other genetic forms of rickets affect the special proteins in the body that vitamin D works with. When a child has low calcium or vitamin D then the child is malnourished. Vitamin D deficiency prevents the efficient absorption of dietary calcium and phosphorus. In a vitamin D–deficient state, only 10–15 percent of dietary calcium and 50–60 percent of dietary phosphorus are absorbed (Michael, 2007)

Poor absorption of calcium causes a decrease in serum-ionized calcium levels. (Trevor, 2005). This is immediately recognized by the calcium sensor in the parathyroid glands, resulting in an increase in the expression, synthesis, and secretion of parathyroid hormone (PTH. (Tom, 2011). PTH conserves calcium by increasing tubular re-absorption of calcium in both the proximal and distal convoluted tubules. PTH, like 1, 25(OH) $_{2D}$, enhances the expression of RANKL on osteoblasts to increase the production of mature osteoblasts to mobilize calcium stores from the skeleton. PTH also decreases phosphorus re-absorption in the kidney, causing loss of phosphorus into the urine. The serum

calcium level is usually normal in a vitamin D deficient infant or child. However, the serum phosphorus level is low, and thus there is inadequate calcium-phosphorus product, which is necessary to mineralize the osteoid laid down by osteoblasts. (Tom, 2011). Thus, typically infants with vitamin D deficiency rickets have a normal serum calcium level, low normal or low fasting serum phosphorus levels, elevated alkaline phosphates levels, and low 25(OH)D levels (<15 g/ml) (Trevor, 2005). The secondary hyperparathyroidism stimulates the kidneys to produce 1, 25(OH)$_{2D}$, and thus, 1, 25(OH)$_{2D}$ levels are normal or often elevated, which is why the measurement of 1, 25(OH)$_{2D}$ is of no value in determining a state of vitamin D deficiency. (Tom, 2011). Only when the calcium stores in the skeleton are totally depleted will the infant or child become hypocalcaemia. (Tom, 2011). Biochemical changes in calcium and phosphorus metabolism due to vitamin D or calcium deficiency or vitamin D resistant syndromes, or Hypophosphatemic syndromes may cause rickets or osteomalacia. However, in severe calcium and vitamin D deficiency, the serum calcium is below normal. (Trevor, 2005). In addition, PTH causes phosphorus loss via the urine, resulting in a decrease in serum HPO_4^{2-}. An inadequate calcium-phosphorus product ($Ca^{+2} \times HPO_4^{2-}$) leads to a defect in bone mineralization that causes rickets in children and osteomalacia in adults. (Tom, 2011). Clinical and radiological bone manifestations predominate in areas of rapid bone growth, including the long bone epiphyses and the costochondral junctions. This is why rickets is mostly observed before 18 months of age, with maximum frequency between the ages of 4 and 12 months. (Tom, 2011). Skeletal deformities are usually a result of long-standing rickets. Hypertrophy of the costochondral junctions leads to beading and the classic rachitic rosary those progresses with involution of the ribs and protrusion of the sternum (pigeon chest) and recession of the costochondral junctions and traverse depressions causing Harrison's groove. (Tom, 2011)

Once the child begins to stand, gravity pushing on the lower limbs results in either inward (genus valgum) or outward (genus varum) tibia and femoral bowing. Muscle pull can also cause bone deformities in both upper and lower limbs even before the infant begins to walk. (Tom, 2011). Muscle traction on the softened ribcage is responsible for the chest deformation, leading to pectus carinatum, thoracic asymmetry, and widening of the thoracic base. Softening of the occipital area (rachitic craniotabes), enlarged sutures and fontanel's, delayed closing of fontanels and occipital or parietal flattening can be observed. Tooth development is impaired, with delayed eruption, enamel hyperplasia, and early dental caries. The pelvic bone structure is flattened in rachitic children. (Tom,2011)

Regular and sensible sun exposure during the months of the year when vitamin D production is promoted is still the most physiologic way to prevent vitamin D deficiency in infants and young children. Sunlight was recommended as a therapeutic method to prevent rickets in infants, and a detailed description was published in the United States Children's Bureau Folder in 1931(Ward, 2007). It was recognized that in the temperate zone, sunlight was feeble in its anthracitic properties in the winter, and thus, it was recommended that children be exposed to UV radiation from a mercury arc or carbon arc lamp in the winter. (Ward, 2007). During exposure to sunlight, the ultraviolet B (UVB) radiation (290 315 nm) is absorbed by 7-dehydrocholesterol in the skin to form provitamin D_3 Provitamin D_3 is inherently unstable and rapidly converts by a temperature-dependent process to vitamin D_3. Once formed, it is ejected out of the skin cell into the extracellular space, was drawn into the dermal capillary bed by the vitamin D binding protein (DBP) (Ward, 2007).

Breastfeeding is an unequalled way of providing ideal food for the healthy growth and development of infants; it is also an integral part of the

reproductive process with important implications for the health of mothers. As a global public health recommendation, infants should be exclusively breastfed for the first six months of life to achieve optimal growth, development and health. Thereafter, to meet their evolving nutritional requirements, infants should receive nutritionally adequate and safe complementary foods while breastfeeding continues for up to two years of age or beyond. ((Holick, 2006)

Exclusive breastfeeding from birth is possible except for a few medical conditions, and unrestricted exclusive breastfeeding results in ample milk production (Holick, 2006)

Over the past decades, evidence for the health advantages of breastfeeding and recommendations for practice have continued to increase. World Health Organization (WHO) can now say with full confidence that breastfeeding reduces child mortality and has health benefits that extend into adulthood. On a population basis, exclusive breastfeeding for the first six months of life is the recommended way of feeding infants, followed by continued breastfeeding with appropriate complementary foods for up to two years or beyond. To enable mothers to establish and sustain exclusive breastfeeding for six months, WHO and United Nation Children Fund (UNICEF) recommend initiation of breastfeeding within the first hour of life. Exclusive breastfeeding, the infant only receives breast milk without any additional food or drink, not even water, Breastfeeding on demand day and night and no use of bottles, teats or pacifiers. However where sunlight is available the children must be exposed to sun rays. (Hollick,2006).

Breast milk promotes sensory and cognitive development, and protects the infant against infectious and chronic diseases. Exclusive breastfeeding reduces infant mortality due to common childhood illnesses such as diarrhea or pneumonia, and helps for a quicker recovery during illness. Breastfeeding contributes to the health and well-being of mothers; it helps to space children,

reduces the risk of ovarian cancer and breast cancer, increases family and national resources, is a secure way of feeding and is safe for the environment. (Hollick, 2006)

2.2.4 Diagnosis of diagnosing rickets

There are four methods used in the diagnosis of rickets. Physical examination to check for any obvious problems with a child's skeleton, including any pain and tenderness coming from the bones. Secondly Medical history diet, family history and any medication the child is undergoing. Thirdly Blood tests may measure levels of calcium, phosphorous and vitamin D. Finally X-ray. Standard X-rays of bones and a special X-ray called a bone densitometry scan (DXA scan), measures the calcium content of the bones.

Figure 1: Rickets presentation in an X-ray

Radiogram of a two-year old rickets sufferer, with a marked gene varum (bowing of the femur) and decreased bone capacity suggesting poor bone mineralization.

2.3.3 Role of Vitamin D and Calcium in rickets

The body needs vitamin D to absorb calcium. Without enough vitamin D, one can't form enough of the hormone calcitriol (known as the "active vitamin D"). This in turn leads to insufficient calcium absorption from the diet. In this situation, the body must take calcium from its stores in the skeleton, which weakens existing bone and prevents the formation of strong new bone.
Experts recommend a daily intake of between 400 and 600 IU (International Units) of vitamin D, which can be obtained from supplements or vitamin D-rich foods such as egg yolks, saltwater fish, liver, and fortified milk. Remember, a balanced diet rich in calcium and vitamin D is only one part of an osteoporosis prevention or treatment program like exercise. Getting enough calcium is a strategy that helps strengthen bones at any age.

Human bodies do not store most vitamins and must consume them regularly to avoid deficiency (Ward, 2007). Calcium intake is critical for everyone from toddlers to mature adults. Calcium builds strong bones during the growing, keeps bones strong during the middle years, and helps regulate blood pressure in women during pregnancy (Ward, 2007). Eating a variety of calcium-rich foods remains the best way to get the calcium you need every day, children, teens and adults get the majority of their calcium from dairy products, which are the best and most convenient source of dietary calcium and eight other essential nutrients. Yogurt, for example, is not only an excellent source of calcium but also a good source of protein. (Ward 2007).

To find out how much calcium you need Calcium is needed for the following. Calcium eases insomnia and helps regulate the passage of nutrients through cell wall. (Ward, 2007). Without calcium, your muscles would not contract correctly, your blood would not clot and your nerves would not carry messages. If you do not get enough calcium from the food you eat, your body automatically takes the calcium needed from your bones (Ward, 2007). If your body continues to tear down more bone than it replaces over a period of years in order to get sufficient calcium, your bones will become weak and break easily .(Ward, 2007). Deficiency may result in muscle spasms and cramps in the short term and osteoporosis. Many other nutrients affect bone health, too because they impact on the absorption or excretion of calcium. Calcium balance-not just intake-is necessary for healthy bone contain enough calcium to offset such losses, the body breaks down bone to release calcium needed to accommodate these. contain enough calcium to offset such losses, the body breaks down bone to release calcium needed to accommodate this.The balance is dependent on the absorption rate of calcium consumed as well as the rate of mostly urinary calcium excretion.(Ward, 2007).

2.4 Methods of preventing rickets

There are three ways of preventing rickets, this includes dietary intake of Vitamin D and calcium rich sources, exposure to sunlight, and supplementation. (Ward, 2007).

2.4.1 Sources of vitamin D

The main sources of vitamin D include Oily fish, liver, eggs margarine, breakfast cereals with added vitamin D while the sources of calcium include dairy products, such as milk, cheese and yoghurt green vegetables, such as broccoli, whole meal bread, dried fruits beans and pulses, such as lentils. (Ward, 2007).

2.4.2 Sunlight

Children get vitamin D from sunlight as the vitamin forms under the skin in reaction to exposure to the sun. About 15 minutes' exposure on the hands and face a few times a week. Too much sun is bad for our skin. In bright weather, it is important to keep out of the sun during the hottest part of the day (11am-3pm), to use sunscreen and keep young children mostly covered up when in the sun. Babies and young children have very sensitive skin that burns easily. If you do not get enough sunlight because you spend a lot of time indoors, or wear clothes that completely cover your skin, you may be at a higher risk of vitamin D deficiency and therefore at risk of rickets or osteomalacia (Ward, 2007).

2.4.3 Supplements

Most people should be able to get all the vitamin D they need from their diet and by getting a little sun. However, certain groups are at a higher risk of vitamin D deficiency and may need to take supplements to prevent rickets and osteomalacia.(Ward, 2007).

2.5 Groups at risk of Rickets

2.5.1 Pregnant and breastfeeding women

The Ministry of Health recommends that all pregnant and breastfeeding women take a supplement of 10 micrograms of vitamin D a day to ensure they get enough for themselves and their baby.

2.5.2 Infants and children under five years

Vitamin D supplementation is recommended for breastfed infants from six months (or from one month if there is any doubt about the mother's vitamin status during pregnancy), formula-fed infants who are over six months old and taking less than 500ml infant formula a day, children under five years old It is

important for children in high-risk groups, for example those getting insufficient vitamin D from their diet, and those with medical conditions i.e. kidney disease, to take supplements.

2.5.3 Geriatrics

Other people at a higher risk of vitamin D deficiency, and who may need to take supplements, include the elderly ,people of Asian, African Caribbean and Middle Eastern origin living in temperate climates ,those who always cover up all of their skin when outside ,people who rarely get outdoor and People who eat no meat or oily fish.

2.5.4 Nutrition status

Nutritional status is predisposing for rickets .when a child has law calcium or vitamin D then the child is malnourished. Vitamin D deficiency is the most common cause of rickets. Vitamin D deficiency prevents the efficient absorption of dietary calcium and phosphorus. In a vitamin D–deficient state, only 10–15% of dietary calcium and 50–60% of dietary phosphorus are absorbed.

CHAPTER: THREE STUDY DESIGN AND METHODOLOGY

3.1 Study setting

Study setting is Kenyatta National Hospital (KNH) in Nairobi. The mandate of the hospital is to act as a teaching and referral hospital to provide specialized health care, to provide facilities for training of health professionals, to research and participate in National health planning and policy. This hospital is a training institution for healthcare personnel in various disciplines and a reference point for training postgraduate medical doctors in various specialties and for providing internship for health professionals including Nutritionists .The hospital has 10 levels each constituting 4 wards. The hospital offers highly specialized services such as radiotherapy, cardiothoracic surgery, neurosurgery, plastic and reconstructive surgery, and critical care services orthopedic surgery renal unit burns unit among others.

3.2 Study Design

This study was retrospective cross sectional and analytic cohort study. The study was for children aged 6-59 months who were suffering from rickets. It is a historical study looking at the causes of rickets which already occurred in the children. This study was a cohort study because I was investigating medical condition of these children with the same condition. They were admitted in level 3 which is a medical ward. The study looked at establishing the risk and causative factors including malnutrition, non exposure to sunlight, heredity, and birth weight and care givers demographic condition as cause of rickets in children of age 6 to 59 months. Study instruments and tools consisted of a structured questionnaire with close ended questions administered to care givers and mothers of children admitted in Level 3, which has wards A, B, C; D.The questionnaire investigated some of the

following topics food frequency, Household dietary Diversity and Anthropometry.

3.3 Study Population and Sampling Frame

The study population comprised of children, girls and boys aged between 6 to 59 months admitted at Kenyatta National Hospital medical wards for children, while the sampling frame consisted of the same children but suffering from Rickets.

3.4 Sample Size Determination

Admissions of children in Kenyatta National Hospital stood at about 500 of age 0 to 59 months. The following formula (Fisher et al., 1999) was used to determine the sample size

$$N = \frac{Z^2 PQ}{d^2}$$

N = the desired population sample size (When the population was less than 10000)

Population in children's wards

Z = Standard normal; deviate set at 1.96 which corresponds to 95% confidence interval.

P = 50 %(proportion of children patients in the medical wards, estimated to be malnourished.)

Q = 1-p (proportion of children in medical wards without malnutrition)

q = 1-0.5=0.5

Hence the desired sample size (n) = $\frac{(1.96)^2(0.5)(0.5)}{0.1^2}$

N= 96 patients

Plus 10% attrition = 9.6 + 96*(10/100) = 96 + 9.6

= 105.6 Patients

+ 106 patients the actual sample size for this study was 144 given that the minimum sample size as calculated is 106.

3.5 Sampling Procedures

The figure below illustrates my sampling procedure. Kenyatta National Hospital was the venue of the study and it was purposively selected because of being a referral hospital. The wards were also purposively chosen being the medical ward.

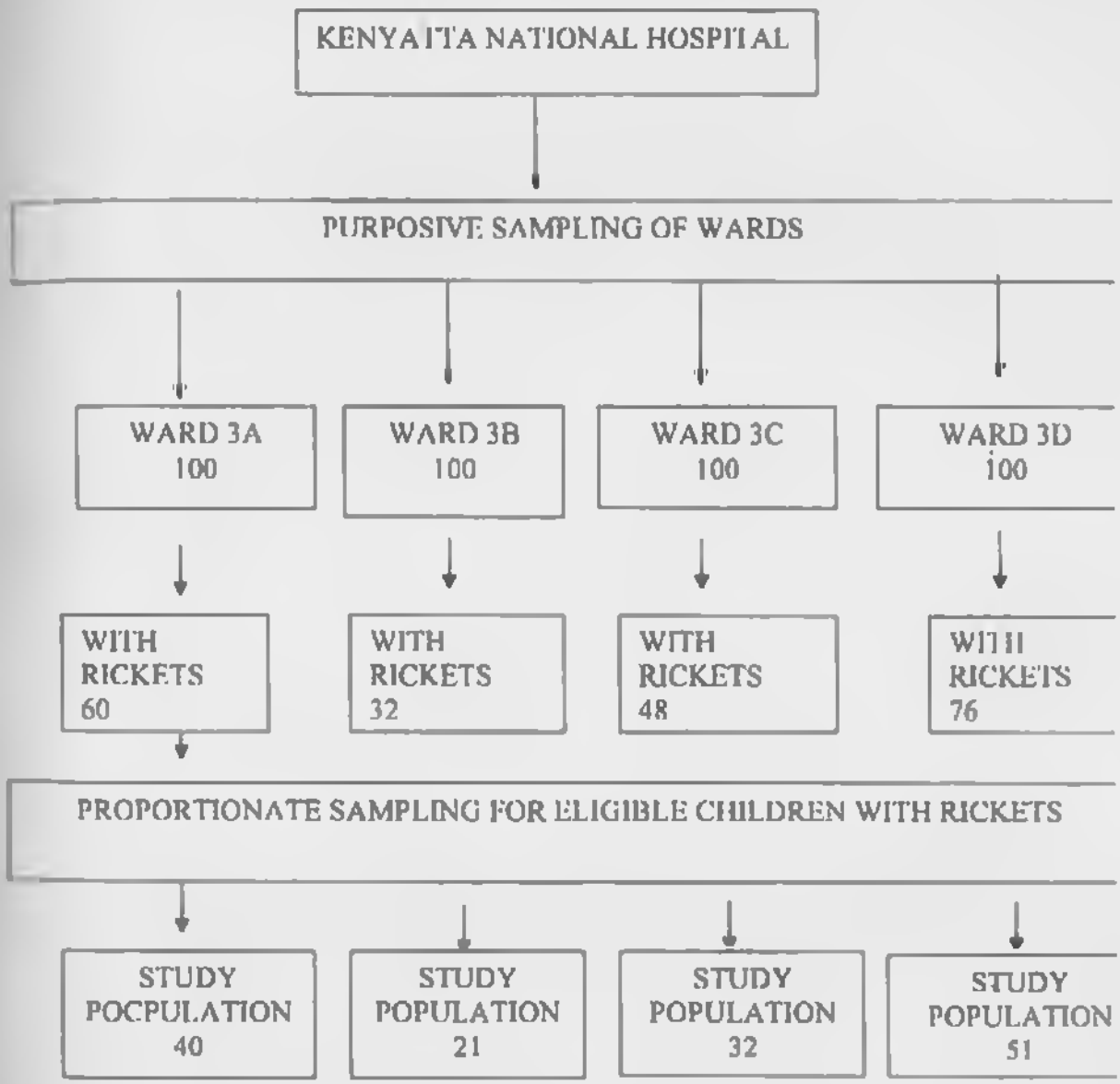

Figure 2: Sampling procedure

3.6 Development of the Questionnaire

Pre-testing of the questionnaire was done in the hospital by the Research Assistants and the Enumerators to verify the interpretation of the instrument. This was done in level 4 of the hospital, because the ward is also a medical ward. Data was collected between December 2010 and March 2011. Data collected was quality as it was done after training of enumerators. SPSS and ENA for smart were used. The instruments for data collection are in the appendix 2

3.7 Data Collection Methods

3.7.1 Anthropometric measurements

3.7.1.1 Height

Height/ length measurements for children aged > 2 years and < 2 years respectively were taken using a length /height boards, placed on a flat surface The following procedure was followed:

1. The research assistant explained the procedure to the child's mother or caregiver.
2. The child's shoes and any other garment or cloth on the head were removed.
3. The child was placed gently on the board on his/her back, with the head against the fixed vertical part and the soles of the feet near the cursor or moving part. The child lay flat in the middle of the board looking directly up.
4. The assistant held the child's head firmly against the base of the board.
5. The other assistant taking the measurement placed one hand on the knees to keep the child's leg straight, leaving the child's feet flat against the cursor with the other hand, and pushing the cursor against the feet firmly but gently.
6. The measurer read the length to the nearest 0.1 cm then recorded. This was repeated and the average calculated and recorded correctly.

7. A measuring board of at least 130cm was used.

3.7.1.2 Weight

A Salter scale was used to take weight. The following steps were followed; The research assistant explained to the mother/ caregiver what he/she intended to do. The scale was hanged from a suitable point ensuring that the dial on the scale is at eye level. The weighing pants were then hanged from the hook of the scale with the needle reading zero. The weighing pants were hanged with the child in them from the hook on the scale with minimal clothing. The assistant then checked that the child was properly suspended then read the scale at eye level to the nearest 0.1 kg. A 25 kg hanging spring scale marked out in increments of 0.1 kg.

3.7.2 Key informant interview

Individual interviews were carried out with selected health professionals including; Doctors, Nurses, and Nutritionists using different set of questions which were open ended to encourage informants to articulate their own opinions and concerns.

3.8 Recruitment and Training of Enumerators

3.8.1Recruitment

Recruitment was done and two enumerators were selected, they both could speak in the local languages. The enumerators had a background in nutrition they had the aptitude skills in data collection and taking of anthropometric measurements.

3.8.2 Training procedures for enumerators

The research assistants were intensively trained for two days. The purpose and general procedure of the study and its expected duration was explained. Emphasis was placed on the sampling methodology, administration of the

questionnaires, taking anthropometric measurements, recording of data collected.

3.9 Data Quality Control

Project monitoring and evaluation to ensure completeness and internal consistency of data was carried out. All measuring equipments were checked periodically during the weighing of the children to prevent instrumental errors that could arise due to faulty equipment. Research assistants were trained in taking anthropometric measurements with emphasis on correct steps when taking measurements to enhance accuracy and validity.

At the end of each day the principal investigator checked the questionnaires for omissions to ensure that each question was answered and the data carefully entered into the computer. Statistical checks for errors were done by examining frequency distributions on all variables for items that were improbable or not logical.

3.10 Data Management and Analysis

The software package Statistical Package for Social Sciences (SPSS) and (ENA for smart) was used to manage and analyze data. Quality Control to improve on validity efficient research questionnaire was used.

3.11 Ethical and Human Rights Considerations

The study did not infringe in the study subjects privacy. The subjects were allowed to decide on whether they wanted to be part of the study. There was a consent form for the study subjects to sign. Confidentiality was upheld. The enumerators were advised to respect the respondents wish to be interviewed or not and respect the respondent's privacy

CHAPTER FOUR: RESULTS AND DISCUSSION

The results presented in this chapter are based on data collected from 144 respondents. Most of respondents were biological mothers represented by 95 percent of mothers and their children admitted in Kenyatta National Hospital in the pediatric ward level 3 A, B, C, and D.

4.1 Socio-Demographic Characteristic of Respondents

Table 1 describes the socio-demographic characteristics including household sizes, ethnicity, education, marital status, age and occupation of the respondents

Table 1: Socio-demographic characteristics of the respondents

Variable	Category	No	%	P-value
Household sizes (N=144)	1-4	40	27.2	0.000
	5-8	90	61.2	
	9-13	13	8.8	
Ethnicity (N=144)	Luo	11	7.5	0.000
	Kikuyu	97	66	
	Kamba	12	8.2	
	Luhya	12	8.3	
	Meru	3	2.1	
	Taita	2	1.4	
	Maasai	1	0.7	
	Kisii	3	2.0	
	Others	3	20	
Education (N=144)	College/university	30	20.8	0.000
	Completed secondary	54	36.7	
	Completed primary	44	30.8	
	Illiterate	7		
	Post secondary	9	4.9	
Marital Status (N=144)	Single	28	19	0.000
	Married	112	76.2	
	Divorced/separated	2	1.4	
	Widowed	2	1.4	

Age category (N=144)	18-25	47	32	0.000
	26-31	64	43.5	
	32-37	24	16.3	
	38-43	9	6.1	
Occupation (N=144)	Housewives	66	44.9	0.000
	Salaried	18	12.5	
	Self employed	23	15.9	
	Casual laborers	8	5.5	
	Unemployed	23	15.9	
	Farmer	1	0.6	
	Students	5	3.4	

4.1.1 Household sizes

The Table 1 indicates that significantly more respondents had family sizes of about 5-8 members while only about 9 percent had family sizes with more than 9 members. Family size has an effect on the capacity of mothers to feed children appropriately. According to the respondents up to 61.2% had families of 5-8 people. Occupation of care givers stood at 50% housewives. This scenario suggests improper feeding practices due to financial difficulties. The current average no of children is 4 (okeno, 2010).Kenyans total fertility rate is 4 children. (Ministry of planning 2010 census).The lowest per capita income stands at ksh. 1550 or 640 dollars. Big families will cause a problem for these families.

4.1.2 Ethnicity of respondents

Kenyatta National Hospital is a referral hospital and it is expected to admit patients from all over the country. The rest of patients were those referred

from other hospitals all over the country. However central province had the highest no of patients suffering from rickets and the no of patients were. 63%.Then reason is the covering of children by mothers to avoid cold with shawls. Housing is a major problem for many families. The current urban setting most families live in flats and children are kept indoors all the time. Children are not exposed to sunlight or sun rays which are important for proper growth of bones. These children are likely to suffer rickets

4.1.3 Education of mothers

From Table 1 it is evident that the education status of most of the respondents was secondary level, education is a prerequisite to good decision in everything including how to feed the children and what to feed. Feeding of the children was bad in terms of choice and method of preparation. Foods left out were the most important in provision of the required nutrient For example; eggs were rarely fed to the children because of some beliefs. There are taboos which allude that children would not develop the sense of speech if fed on eggs.

A study in Egypt by (Zubeida et al 2012) found concurring evidence on risk factors of rickets "Rachitis children come from low socioeconomic status and that most come from slums where poverty and illiteracy was rampant. There was low supplementation of vitamin D at 12%.Thus there was agreement that breastfed children with no supplementation especially those not exposed to sunlight developed rickets.(Zubeida et al ,2012)

4.1.4 Occupation of respondents

Most of the respondents were either housewives or petty traders. Very few were professional or working for the government, others were house employees or casual laborers. The majority had very little income if any and this is a contributing factor to the situation of the children feeding becomes a challenge. Resources may not be available to buy the required foods .Thus if most were not working this would affect feeding. Many studies suggest that

Nutritional Rickets is a social disease, naming poverty as the main culprit. Recently a study conducted in Turkey found that Nutritional Rickets was a disease of the 'underprivileged' being strongly correlated with negative social background and lack of vitamin D (Saddiq, 2005). Rickets was found to be higher among children of unemployed mothers (Saddiq, 2005). Similarly a Lebanese study found that children and adolescents of higher socioeconomic status were more likely to eat calcium and Vitamin D fortified foods thus protecting them from development of nutritional rickets. (Abdulbari, 2010). When mothers have a source of income children are likely to get the required food and nutrients.

4. 1.5 Demographic characteristics of caregivers and mothers

Majority of the Respondents were married at 76.2 percent most mothers were married but the children developed rickets. This is not expected since it is expected that single mothers may not have capacity to take care of children well. However these children had a problem thus parent presence may not be as important as the financial status. Families were together mother and father but they were not able to feed children appropriately The findings of (Pannapakkan et al 2008) revealed that rickets was confounded by increased family size and low family income, deficiencies of calcium, and low socioeconomic status .This concurred with my findings in this study.

Most of the respondents were between 18-31 years of age at 75 percent. This indicates that most of these mothers were young, may be lacking experience in child care. This is the normal child bearing age and we expect many mothers to have children, therefore it may not indicate that mothers of this age had more children with rickets.

4.2 Child Characteristics

The following section describes children characteristics including sex, age birth order, immunization status, vitamin A supplementation status, and breastfeeding status.

4.2.1 Children sex and age

Table 2 below shows the distribution of the study children by sex and different age groups. The number of boys was slightly higher than girls at 51.7 percent and 46.35 percent respectively.

As Table 2 indicates, majority of children who suffered rickets were of the age 4-13 months with a total of 83.2percent. The reason for this may be that this is the time when most children are introduced to complimentary feeding which most of the time poses a great challenge to caregivers in terms of food choices, purchasing power and proper food preparation. Introduction of complimentary feeding is therefore a big challenge to mothers as has been reported before.

Table 2: Characteristics of children in terms of sex and age

Variable	Category	Number	Percent
Sex	Male	76	51.7
(N=144)	Female	68	46.3
Age	4-8	61	42.4
	9-13	60	40.8
	14-18	12	8.2
	19-23	4	2.7
	24-28	3	2.0
	29-32	1	0.7
	33-37	3	2.0

4.2.1 Child's birth order

Most of the children were either 1st or 2nd born, this means that most of the mothers were young with reduced experience in taking care of children. This could have been a factor in children developing rickets. Young mothers with first born have challenges in child care especially in appropriate feeding. The chi-square analysis on child birth showed that there was strong association with the child developing rickets. There was a strong association between child birth order and Age of care givers (P= 0.000)

4.2.2 Immunization status and vitamin A supplementation

As Table 3 indicates, those who were fully immunized were 60.5 percent while those who were not fully immunized were 36.7 percent. There was a significant difference between these two groups (P=0.003). There was no relationship between rickets and immunization status of the children.

The results indicate that significantly more children had been given vitamin A supplementation at 87.8 percent (P=0.000) which is a booster this should improve the health status of the children .However they still developed rickets.

Table 3: Children characteristics

Variable	Category	Number	Percent
[illegible]'s birth order (N=144)	1	39	26.5
	2	44	29.2
	3	40	27.2
	4	9	6.1
	5	3	2.0
	6	3	2.0
	7	5	3.4
[illegible]ion status N=144	Yes	89	60.5
	No	54	36.7
[illegible] provided vitamin A (N=144)	Yes	129	87.8
	No	15	10.4
Child still breastfeeding Status (N=144)	Yes	132	89.8
	No	12	8.2

4.2.3 Child Breastfeeding status

Most of the children (89.8%) were still breastfeeding at the time of the study. While 11.2 percent had either stopped breastfeeding or had never been breastfed because their mothers were HIV positive and had chosen not to breastfeed their children as prevention of mother to child transmission. The following research was done in Carolina United States

Of 30 babies with nutritional rickets seen at the Wake Forest and UNC medical centers, all were black and all were breast fed. Most of them were growth retarded nearly a third, severely so. Except for babies who had recently begun receiving vitamin supplementation or had begun drinking vitamin D-fortified milk, all the babies had vitamin D deficiency. (Schwartz, et al 2000)

Breastfeeding among black women in North Carolina has risen dramatically in the past few years. Breast milk is the ideal form of nutrition for infants, but the amount of vitamin D in breast milk depends on the vitamin D status of the mother. Dark-skinned mothers need more exposure to sunlight than light-skinned mothers to produce the same amount of vitamin D. The same is true of darker skinned babies. Unless they receive vitamin supplements, breast-fed dark-skinned infants are at risk for rickets. (Schwartz, et al 2000)

The amount of sun exposure an infant needs to prevent rickets depends on skin pigmentation, the amount of clothing worn, latitude, time of day, season of the year, amount of smog, and so on. Moreover, many parents do not want to expose their babies to too much sun for fear of skin cancer.

Maternal vitamin D status also plays a role in this vitamin deficiency, but it is impractical to test nursing mothers. Although there is no national reporting system for rickets that can give statistics on these condition nationwide, indications are that rickets is on the rise throughout the United States. Increasingly, it appears that vitamin supplementation for all breast-fed infants are a safe, low-cost, and reasonable option. As a result of these findings, the North Carolina Women's, Infants and Children (WIC) program now distributes a free multivitamin supplement for babies 6 weeks of age or older who are exclusively breastfeeding. It also sends out fact sheets on vitamin D supplementation to both parents and health professionals. (Schwartz et al 2000)

This study confirms findings in regards to breast milk and breast feeding that breast milk is deficient in vitamin D. However in Kenya with plenty of sunlight mothers should expose their children to sun without covering them and secondly there should be a policy on fortification of babies milk formulas sold for mothers who cannot breastfeed.

4.3 Nutritional Status of the Study Children

Table 4 shows that most of the children were underweight and wasted showing high prevalence of acute malnutrition. This could be as result of the other infections that led to their admission in the hospital. The table 4 compares the three different statuses of children who are wasting, underweight and stunting

Table 4: Nutrition status of the study children

Variable	Category	Number	percent	p-value
Underweight	Yes	78	69.9	0.000
(N=130)	No	52	31.3	
Stunting	Yes	25	14.6	0.000
(N=144)	No	119	82.6	
Wasting	Yes	82	64	0.000
(N=130)	No	48	36	

There was significant difference between those children who were wasted, underweight and stunted compared to those who were not as indicated by the p-value in table 4 above. These conditions are predisposing for rickets. The figures on the table indicate high prevalence of malnutrition because they are underweight, stunted or wasted.

4.3.1: Prevalence of global acute malnutrition

According Table 5 the prevalence of Global acute malnutrition was high among the children is compared to the national average of 15 Percent. The reason for this could be because these children were also suffering from other diseases and therefore their nutritional status had also deteriorated due to long illness.

Global acute malnutrition was high among these children at 63.07 percent. Most of these children were sick before they were diagnosed with rickets. Most of the children had

Table 5: Prevalence of Global Acute Malnutrition among study Children

Variable	Nutritional Status				P-value
	Severe	Moderate	Normal	Total	
GAM(n=130)	29.23	33.84	36.92	63.07	0.040

4.3.2 Prevalence of global acute malnutrition by sex

Table 6 indicates that there was no significant difference between boys and girls in terms of prevalence of Global Acute Malnutrition.

Table 6: Prevalence of Global Acute Malnutrition by Sex

Variable	Severe		Moderate		Normal		Total	P-value
	Boys	Girls	Boys	Girls	Boys	Girls		
WHZ/SEX	20	18	24	20	23	24	130	0.644

4.4. Dietary Habits of children

Table 7 describes dietary habits of the study children regarding those foods rich in calcium and Vitamin D .The table describes the types of foods fed to the children .Most of foods rich in calcium were not fed to these children.

Table 7: Dietary habits of the children

Variables	Category	No	%	p-value
Finger millet	Yes	95	66.8	0.000
(N=144)	No	49	34.0	
Spinach	High	60	41.7	0.049
(N=144)	Low	84	58.3	
Amaranthus	Yes	54	37.5	0.003
(N=144)	No	90	62.5	
Sweet potatoes	Yes	111	77.5	0.000
(N=144)	No	33	22.5	
Cowpeas	Yes	18	12.5	0.000
(N=144)	No	126	87.5	
Pumpkin	Yes	54	38.7	0.000
(N=144)	No	90	61.3	
Feeding	Yes	16	11.1	
Dagger	No	128	88.9	
(N=144)				
Feeding Eggs	Yes	67	47	0.00
(N=144)	No	73	53	
Feeding of peas	Yes	27	18.4	0.000
(N=144)	No	117	78.9	
Mixed Flour	1-Finger Millet	10	6.9	0.086
(N=144)	2-Maize finger/millet	17	11.8	
	3-Millet/beans/green grammes	27	18.8	
	4-Maize/soybean/ground nuts sorghum	21	14.6	
	5 -Maize/cassava/dagger/g/nuts/			
	/green grams	9	6.3	
	6-Maize/g/nuts/sorghum/beans/			
	Dagger/cowpeas.	7	4.8	
	Not mixing			
		53	36.8	

4.4.1 Finger millet consumption

Most mothers fed their children on finger millet porridge which was commendable. This is a good source of calcium for the children. Finger millet has 294-390 mg per 100 g of calcium depending on variety. (Babu1987) Average finger millet contains 350 mg calcium in 100 gait is also high in amino acids but does not contain much vitamin D.(Nelloweath,2011) However, cereals are known to have phytic acid and phytate are widespread in plant cereals and may make the calcium which is high in finger millet to be unavailable. Phytic acid is generally regarded as the primary storage of both phosphate and inositol in seeds. Phytic acid constitutes the major portion of total phosphorus in the grain. Finger millet is not a good source of vitamin D.

Table 8 gives the relationship between consumption of finger millet and nutrition status of the children

Association between consumption of finger millet and Nutritional status

As indicated in table 8 below there was a relationship between finger millet and nutritional status of the children.

Table 8: Association between consumption of finger millet and Nutritional status

Variable	Status	Odds ratio	p-value
Finger Millet	HAZ (Stunting)	1.364	0.038
Finger Millet	WAZ (Underweight)	0.754	-0.056
Finger millet	WHZ (Wasting)	1.006	0.006

4.4.2 Consumption of spinach

Most mothers fed their children spinach as a vegetable of choice daily with some feeding the children up to 4 times a day. Almost every respondent gave their children spinach several times a day. Spinach has a very high level of oxalate at 750mg per 100mg.Oxalate acid is naturally accruing chemical in green leafy plants and is poisonous. Oxalate acid binds with calcium salt to

form calcium oxalate and this chemical is not available to the body. A child who feeds on a lot of spinach will lack calcium from the vegetable which could lead to rickets- (Lindsay boyers ,2011). substances that interfere with calcium absorption are excessive fatty acids bind calcium an prevent absorption phytic acids found in wheat and oxalates found in spinach my make calcium unavailable excessive phosphorous (as in soft drinks) binds Ca and prevents absorption, excessive fatty acids bind calcium and prevent absorption(Clifford ,2011) Spinach increases the risk of stunting by 4 times (odds ratio 4.11) that means that feeding of spinach increases the risk of stunting by 4 times. P-value of 0.004 which is statistical significant .Feeding of spinach and being underweight was(1.25 odds ratio).

This means that feeding of spinach increased the risk of underweight by 1.25 times. The p-value was 0.051which is statistically significant.

4.4.3 Consumption of amaranth

A significant number of mothers did not feed children on amaranths (P= 0.003) as a vegetable of choice as indicated in Table 7. This vegetable is high in calcium, and should have been a vegetable of choice. At three weeks it contains 2693.1mg in 100mg calcium and even after cooking humans will get the daily requirement of calcium of 1000 to13000mg/day (Manobo, 2010). However amaranths contain 91g per kg oxalate. This is high and may cause calcium to be unavailable if fixed to oxalate. It contains high levels of vitamin C at 420ppm and 250 ppm. It has negligible vitamin D. (Teutonico, 2008)

4.4.4 Consumption of sweet potatoes

The sweet potato is also a good source of calcium. Sweet potatoes contain a lot of minerals iron 1.4g, phosphorus 108g potassium 950mg. Sweet potatoes are one of tubers high in calcium, however, significantly more children were not fed sweet potatoes. Contrary to what is believed, sweet potatoes are low in

energy density due to water content which is more than two thirds its weights. Carbohydrates are the major nutrient in potatoes. Iron is present in small amounts at 0.7mg/100g; carotene is present in white and orange fleshed sweet potatoes. Orange fleshed contains 1820 to 16000 ug/100g and white 69 ug/ 100g the protein is approximately 1-2 percent. Calcium in sweet potatoes is 76.0 mg per 100g at 8 percent. (Hoven ,2011) Sweet potatoes have more of carbohydrates and water than calcium or any other nutrient.

4.4.5 Cowpeas consumption

As seen in Table 7, consumption of cowpeas is very low among the study children. It is rich in amino acid, lysine and tryptophan but it is low in methionine and cystine. It therefore, was a good source of nutrients that would have kept diseases away but most mothers did not feed this vegetable. Food content of cowpeas is protein, 24.8 percent; fat at 1.9 percent fiber at 6.3 percent and carbohydrates at 63.6 percent (, Dapaah, 1991).This does not have direct effect on rickets but would have effect on the children who were underweight, or stunted in improving their weight.

4.4.6 Consumption of pumpkin leaves

In this study most mothers did not feed pumpkin leaves to their children. Pumpkin contains high quantities of antioxidant and beta-carotene and is low in calories and high in potassium. It contains a fair amount of vitamin C, niacin, vitamin E .Pumpkin therefore would have boosted immunity of the children and hence mounting an effective immune system against infections.Pumkin leaves contain per cup, calcium 15.2mg,phosphorus at 40.6 mg ,potassium at 170 megavitamin A at 757uu and vitamin C at 43 mg.It has no vitamin D(Agyemay 1999).Pumpkin leaves being a cheap vegetable would have been a good source of a little calcium available from it considering that

calcium was not available after fixation by oxalic acid from spinach and amaranthus.Pumkin is also a good source of vitamin A.

4.4.7 Consumption of silver cyprinid *(Rastrineobola argentea)*

This type of fish was rarely fed to children or even used by the respondent's. The cyprinid is small fish which is high in calcium content. It contains 2500 mg calcium 100 g, 48.32 percent crude protein and 14.9 percent fat content .It also contain fat soluble vitamin A and D. (Romwalb, 2006) This fish is cheap and readily available .How ever most mothers did not use this fish to feed to children. The problem as observed by the respondents is they did note like the aroma of this fish. Consumption of cyprinid reduces stunting by 1.3 times (p-value 0.003) wasting by 1.0 times (p-value 0.000),

4.4.8 Consumption of eggs

The reason for not feeding on eggs from most respondents was due to superstitious beliefs. They said that if they fed eggs to the children, the children may not develop the sense of speech or talk. This is unfortunate because an egg contains most nutrients that would have kept Rickets at bay. An egg contains nutrients which would be useful to the growing child Nutrient content of eggs is as follows, protein is present in the York 16.5g/100g and in white 9g/100 g. This is of high biological value. Fat is present in the York at 30.5g/100g. Calcium and phosphorus are present in the York at 130g/100g and 500mg/100 g respectively. Iron is present in the York 6.1mg/100g .zinc is also present 3.9mg/100g. Retinal is also present in the York in significant amount 535ug/100g. vitamin D is also found in the York at 4.94ug/100g and other vitamins i.e. B-vitamin, Riboflavin and vitamin E. Feeding eggs to children reduces chance of wasting by 1.3 times (p-value -0.006), underweight by 7 times (p-value 0.054) and stunting by 8 times (p- value of 0.024).(Davis 2001)

4.4.8 Consumption of peas

Peas are high in calcium though lower than other beans. The content of calcium is 36mg per 100mg and is rich in other nutrients such as calorie 117g per unit, protein 7.8 per unit carbohydrate 20.97 gm and is rich in iron, 2.13gm, magnesium, phosphorus and zinc. (Shercun, 2011).

4.4.9 Consumption of beans

Beans were not feed to the children and in the few cases it was mixed with other foods in what was mixed in the flours. Beans are highly nutritious and would have been important in alleviating malnutrition (Davis 1997). Beans are very high in Protein which accounts for 20 percent of weight for dried pulses however soybean is an exception to this. Protein content is 36 percent and it is of high biological value. The limiting amino acids are methionine and cystine which are high in cereals and the two cooked together give all required nutrients. Folate, iron, zinc is present. Calcium is variably high in beans. For example soya bean (240per100g). However beans contain some anti -nutrient such as trypsin inhibitor phytate, Oligosaccharides and saponins. This may interfere with protein digestion. Soya bean is also one of the beans considered in the study but almost none of the respondents fed the children on soya beans.

4.4.10 Consumption of mixed floors

As observed a high percentage of respondents fed their babies mixed flours at 55.2 percent. The flours were mixed differently. One flour would mean finger millet or maize flour. Two flours would be mixing maize and finger millet. Three flours would mean maize, green grams and beans mixed in different quantities 4 flours would be maize, soybean, groundnuts and sorghum. The mixture depended on the choice of the person buying, the flours are mixed

during purchasing by the merchant and what a respondent requires in some shops and others are already packed.

Most mothers used these flours. The difference was the number of flours mixed. As shown in the results Table 7 the number of mixed flour which was most common with mothers was 3 flours. Of the total only 36.8 percent of the mothers did not feed mixed flour to their children. Feeding of children with mixed four significantly increased the chances of stunting (odds ratio 1.36) and wasting by (1.0 odds ratio).Feeding mixed flour increases chances of stunting by 1.3 times therefore mixed flour is a risk to stunting.*4. 5 Association among factors*

4.5.1 Association between spinach and nutritional status

Table 9 shows association between consumption of spinach and nutrition status. It shows that, feeding of spinach increases chances of being underweight by 1.25 times and increases stunting by 4 times . Consumption of spinach seems to reduce chances of being wasted by 0.756 times

Table 9 Association between spinach and nutritional status.

Variable 1	Variable 2	Odds ratio	P-value
Spinach	WAZ (Underweight)	1.25	0.051
Spinach	WHZ (Wasting)	0.756	0.451
Spinach	HAZ (Stunting)	4.11	0.004

4.5.2 Association between consumption of mixed flours and nutritional status

Feeding of mixed flour increases wasting by 1.08 times and reduces chances of underweight by 0.8 times as indicated in Table 10. Further analysis using bivariate regression showed a negative correlation between consumption of mixed flours and education of care givers(r= -0.079). This means that the more educated the respondent are the less likely that she will feed mixed flour to the child. Rickets can generally be traced back to malnutrition, especially not getting enough vitamin D and calcium in the diet. If an infant is born with a failure to thrive or a nutrient absorption condition, he could be at higher risk for developing rickets. Starvation, inadequate food intake or an extremely poor diet can also be a cause of rickets

Table 10 Association between mixed flours and nutritional status

Variable 1	Variable 2	Odds ratio	P-value
Mixed flour	WHZ (Wasting)	1.087	0.053
Mixed flour	WAZ (Underweight)	0.800	-0.051
Mixed flour	HAZ (Stunting)	1.402	0.006

4.6 Hereditary Factors

According the Table 11 below, significantly more children did not have a family history of rickets compared to those who had at least one person in the family who had suffered from rickets. This may be the case because most of the respondents could not identify a child with rickets. Most mothers responded to not having any family members who had suffered rickets. They

were not sure about the diagnosis of previously sick children. But other studies also confirm that heredity in rickets is less observed

Table 11: Children with family history of rickets

Variable	Yes	No	Chi-square	P- value
Family member with rickets	25.9%	72.1%	32.111	0.000

This study showed that rickets with hyper calciuria is a rare genetic disorder of phosphate homeostasis which is similar to other genetic phosphate wasting disorders such as X-linked Hypophosphatemia and autosomal dominant hypophosphatemic rickets. This type is characterized by decreased renal phosphate re-absorption, and rickets patients often suffer from bone pain, muscle weakness, and growth retardation. (Belashew 2008). This study concurred with my study because hereditary rickets was rare, as observed in the study there were no family members who suffered rickets before.

4.7 Exposure to Sun Shine

Table 12 shows the percentage of children who were exposed at least once a day to sunshine. Most respondents took children out to be exposed to sunlight but they remained covered in clothes. Most mothers cover their children 100 percent with shawls only conscious of cold getting to the child.

Table 12 Exposure to sunshine

Exposed to sun	Frequency	%
YES	118	81.9
No	26	18.1
Total	**144**	**100**

The epidemic scourge of rickets in the 19th century was caused by vitamin D deficiency due to inadequate sun exposure and resulted in growth retardation,

muscle weakness, skeletal deformities, hypocalcaemia, and seizures. The encouragement of sensible sun exposure and the fortification of milk with vitamin D resulted in almost complete eradication of the disease. Vitamin D deficiency has again become an epidemic in children, and rickets has become a global health issue. In addition to vitamin D deficiency, calcium deficiency and acquired and inherited disorders of vitamin D, calcium, and phosphorus metabolism cause rickets. This review summarizes the role of vitamin D in the prevention of rickets and its importance in the overall health and welfare of infants and children. (Petiffor, 2008). This study shows clearly that sunlight exposure is important for keeping rickets at bay.

4.8 Results from Key informant interview

4.8.1 Prevalence of rickets in the wards in the recent past

The Key informant interviews including the Nutritionist in charge, the nurses and the Doctors indicated high incidences of rickets reported among the children admitted in the wards especially in the recent past.\

4.8.2 Reasons for the high prevalence of Rickets among the under fives

Lack of exposure to sunlight

Cultural beliefs and taboos that certain foods were not suitable for children for example, eggs.

Lack of supplementation of lactating and pregnant women, and also infants as it is the case in other countries.

Lack of knowledge increases consumption of mixed floors and spinach amongst the caregivers.

Inadequate consumption of foods rich in Calcium and vitamin D

4.8.3 Management of rickets

Nutrition counseling by nutritionists and education for mothers on appropriate complimentary feeding and good food choices .Food preparation is also a major problem Provision of physiotherapy and supplementation with calcimax for three months.

CHAPTER 5 FIVE CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

The demographic situation of care givers had an effect on the children's condition, because low socio-economic status represented by increased family size and low family income and illiteracy were confounding characteristics to rickets

There was high prevalence of malnutrition among the study population this includes high prevalence of stunting, wasting and underweight was above the national levels. The prevalence of GAM was higher than the national level

The prevalence of rickets was high in the study population as high as 28% when compared with the national level of 15%.This was of a result of illness because most of these children were in acute side rooms where very ill children are admitted.

The following were observed as causative factors in the study:

Breastfeeding was high among the children, but this did not change the prevalence of rickets. Most children were breastfed, except for mothers who voluntarily did not breastfeed because of other health complications. Breastfeeding was confounding because vitamin D was law but this would have improved with exposure to sunlight

Lack of adequate exposure to sunlight was found to be a risk factor for rickets as many of the children were either not exposed to sunlight at all or were exposed while covered so did not sun rays on the skin for production of vitamin D.

The other risk factor was feeding of children. Mothers preferred spinach to feed the children but spinach fixes calcium and therefore this nutrient is unavailable to the body which also affects presence of vitamin D.
Mixed flour feeding was a risk factor because the porridge cooks for a short time but most of the mixed foods did not cook to make the nutrients available.

Hereditary factors did not show an association with rickets in this study unlike what is documented in most literature review. The reason being that, very few of the respondents could recognize the signs and symptoms of rickets.

5.2 Recommendations

Pediatricians and obstetricians and Nutritionists are responsible for educating parents (and expecting parents) about measures to prevent rickets. For the expecting mother, this includes recommending proper nutrition measures when expectant and appropriate nutritional counseling. For the new mother, it is important for her to be advised about vitamin D supplementation for exclusively breastfed babies. Proper exposure to sunlight for babies as the child gets older. Appropriate nutritional counseling when weaning and reasonable knowledge on type of foods to use during weaning.

The government through ministry of health to recommend fortification of formula milk with vitamin D for mothers who voluntarily do not breastfeed

The government to put a policy in place through Ministry of health for exposure to sunlight of children at least once a day in the morning hours with least covering of cloths.

All mothers who are expectant to receive serious training at Antenatal clinics from trained nutritionists on appropriate feeding of babies during weaning.

Consumption of spinach and mixed flours should be done in moderation especially for children who are less than two years to reduce the incidence of rickets.

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APPENDICES Appendix 1

Participant Consent Form

Title of Research Project: Risks and causative factors of Rickets in inpatient children aged between 6-59 months; A case study of Kenyatta National hospital.
Name of Researcher:
Participant Identification Number for this project: Please tick in box

1 confirm that I have read and understand the information sheet/letter (delete as applicable) dated *[insert date]* explaining the above research project and I have had the opportunity to ask questions about the project. ☐

I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason and without there being any negative consequences. In addition, should I not wish to answer any particular question or questions, I am free to decline. *Insert contact number here of lead researcher/member of research team (as appropriate).* ☐

I understand that my responses will be kept strictly confidential; I give permission for members of the research team to have access to my anonym's responses. I understand that my name will not be linked with the research materials, and I will not be identified or identifiable in the report or reports that result from the research. ☐

4. I agree for the data collected from me to be used in future research

I agree to take part in the above research project. ☐

Name of Participant (*or legal representative*)	Date	Signature
Name of person taking consent (*If different from lead researcher*)	Date	Signature

To be signed and dated in presence of the participant

Researcher	Date	Signature

Appendix 2

STUDY QUESTIONNAIRE

DIETRY ASSESSMENT OF HOSPITALIZED CHILDREN PATIENTS A CASE STUDY OF KENYATTA NATIONAL HOSPITAL NAIROBI.

SECTION A; PATIENTS INFORMATION

The information in this section will be obtained by asking the patient directly and from the patient's medical records i.e. patient's file and Doctor's Comments

Study No…….. Date of interview …….

Hospital…… IP/NO……

Medical ward ……..

1 Name of Patient… ……..

2 Patients Age ……

- Patients Sex

1= male 2=Female

3. Care givers other characteristic

SECTION A

Key code are provided below

Residence	Ethnicity	Occupation	Education	Marital status	Age	Gender	Family Size
				1= Single 2= Married 3=Divorced/Separated 4=Widowed			

Occupation of care givers

1= -house wives

2 Trader/Businessman/Woman

3=Professional /Accountant/Lawyer

4=Government Employee

5= Servant /House boy/Maid

6=Casual laborer

7=Farmer

8=Student

9=Unemployed

Education

1=None

2=Primary

3=Technical training after primary

4=Secondary

5=Post Secondary certificate

6= University/Professional

SECTION C

Q23 Anthropometry for children aged 6 -59 months or (65 -109.9 cm) in the household

SERIAL NO	Q16 Child Birth Order	Q 17 Weight (0.1 kg)			Q18 Height (0.1 cm)			Q 19 Bilateral oedema 1=YES 2= NO	Q 20 MUAC (cm)		
		1	2	Aver	1	2	Aver		1	2	Aver

DIETARY INTAKE

24 hr Household Dietary Diversity

Twenty four-hour recalls for food consumption in the house hold normally when baby is at home

Food group Consumed: What foods groups did members of the household consume no	Did a member of your household consume food from any these food groups in the last 24 hours? *1=Yes* 0= *No*
Type of food	2=no
Cereals and Cereals products (e.g. maize, spaghetti, rice, bread)?	
Milk and milk and milk products (e.g. goat/cow fermented milk, milk powder)?	
Sugar and h	
Oils/fats (e.g. Cooking fat or oil, coconut milk, butter, ghee, margarine)?	
Meat, poultry, Meat, Poultry, offal (e.g. goat, beef; chicken or their products)?	
Pulses/legume Pulses/Legumes, nuts (e.g. beans, lentils, green grams, cowpeas; peanut,)?	
Roots and tube Roots and Tubers (e.g. sweet potatoes, cassava, arrowroot Irish potatoes)?	
Green vegetables, tomatoes, carrots, onions)?	
Fruits (e.g. Water melons, mangoes, grapes, bananas, lemon)?	
Eggs?	
Fish and sea foods (e.g. fried/boiled/roasted fish, lobsters)?	
Miscellaneous *(e.g. spices, chocolates, sweets, beverages, etc)?*	

Q22 How many food groups have the family consumed?
1=every day 2. =In two days
3 =in three days 4= Per a Week
Q23 How many meals did the household consume in 24 hours.)

Q24 Who feeds the baby normally at home?
1 =mother 2=hose help 3=sister 4=brother 5=others

SECTION E: Food frequency (types of food consumed)

	Q25.frequency per week	Q26.frequency consumed per 2wks	Q27 Rare Consumed	Q28 never consumed	Q29 Food source
Animal products					

Q40What time of the day usually? 1=morning 2=mid day 3=even

SECTION H: FREQUENCY OF FOODS RICH IN CALCIUM

	Q40. consumed Daily	Q41 consumed four times a week	Q42 Consumed three times a week	Q43 Consumed twice a week	44. Consumed once a week	45. Not consumed
Cereals						
1.Finger millet						
2.cassava						
3.wheat						
4.Sweet potatoes						
5.Bread						
Animal Product						
6.Beef						
7. chicken						
8..Omena						
9.eggs						
10.[redacted] and [redacted] Products						

Legumes						
12.Peas						
13.beans						
14.chick pea						
15.Cowpeas						
16.Soya bean						
17.pigeon pea						
Vegetables						
18.Amaranth leaves						
19.Cassava leaves						
20.Pumpkin leaves						

SECTION I: COMPLIMENTARY FEEDING

Q 46 What mixed flours did you feed the baby

.....

Q 47 how often did you give the food?

1= once a day

2=twice a day

3=3 times a day

4=4 times a day

Q 48 Approximately how long did you cook the food ?

1= 5 minutes 2 = 7 minutes

3 = 10 minutes 4 = 30 minutes

Q49 How often do you give the baby spinach?

1= daily

2= Twice a day'

3= Once in two days

4= Twice a week

5= once a week.

APPENDIX 3

KEY INFORMANT QUESTIONNAIRE

RESEARCH TOPIC: RISK AND CAUSATIVE FACTORS OI RICKETS

NUTRITIONIST

- What is your name?

- Which ward do you work?

.....

- In the course of your work how oflen do you encounter children suffering from rickets

.....
.....
.....
.....
.....
.....
.....
.....
.....

What in your opinion is the cause of the rise in the cases of rickets?

.....
.....

Appendix 4

PIE CHARTS REPRESENTING DIFFERENT VARIABLES

Figure 1

Birth condition

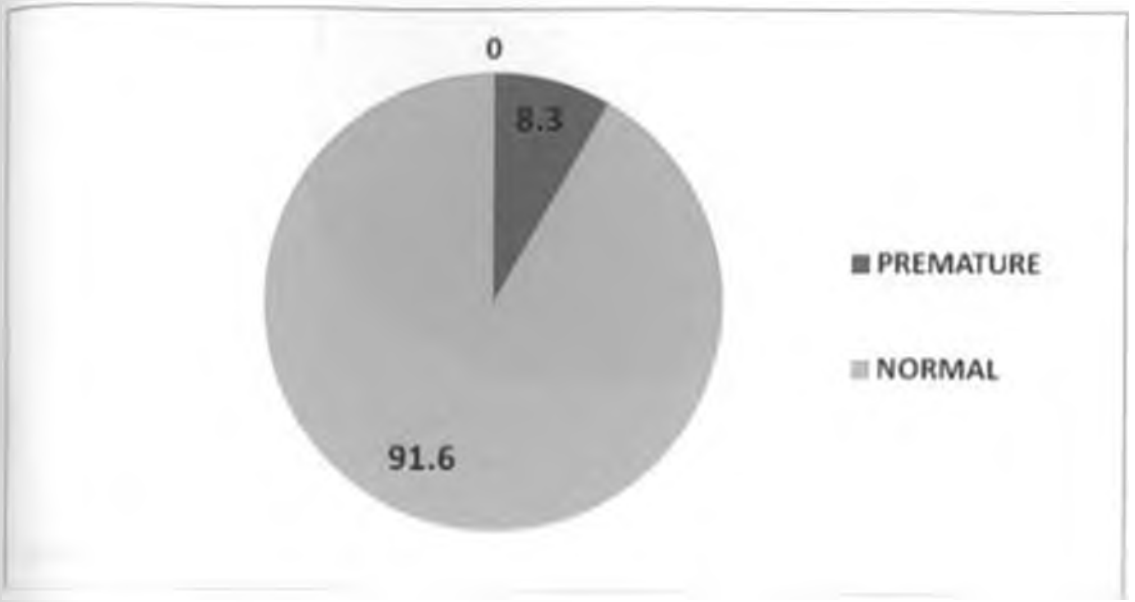


Figure 2

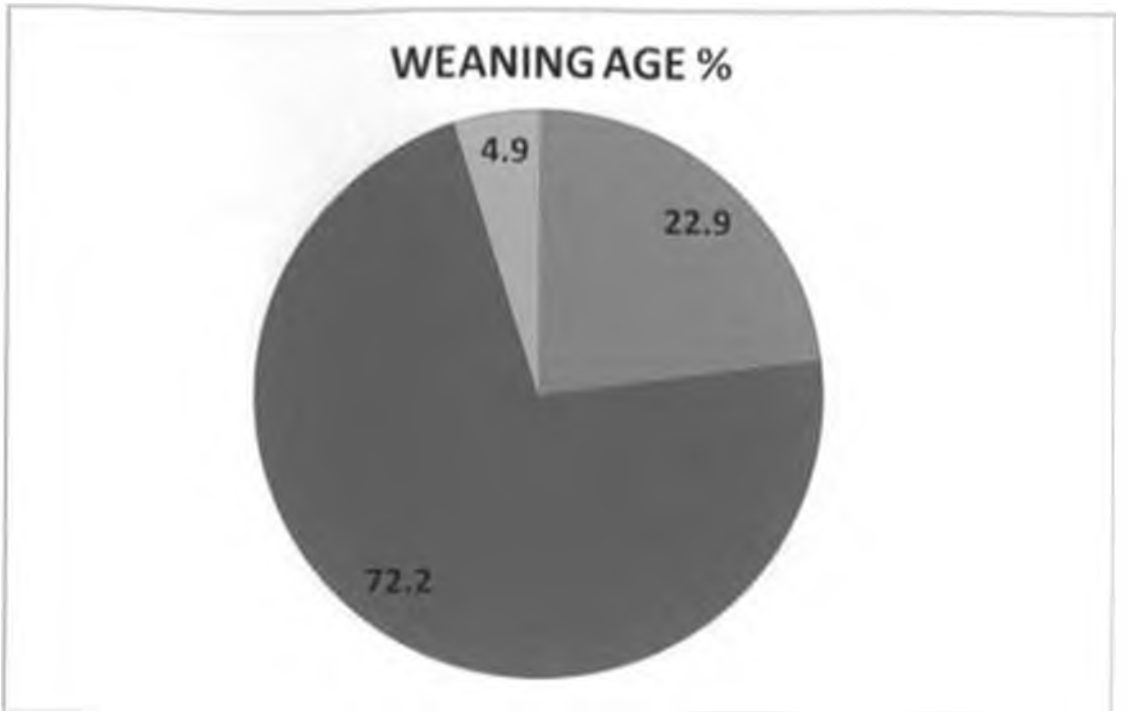


Figure 3

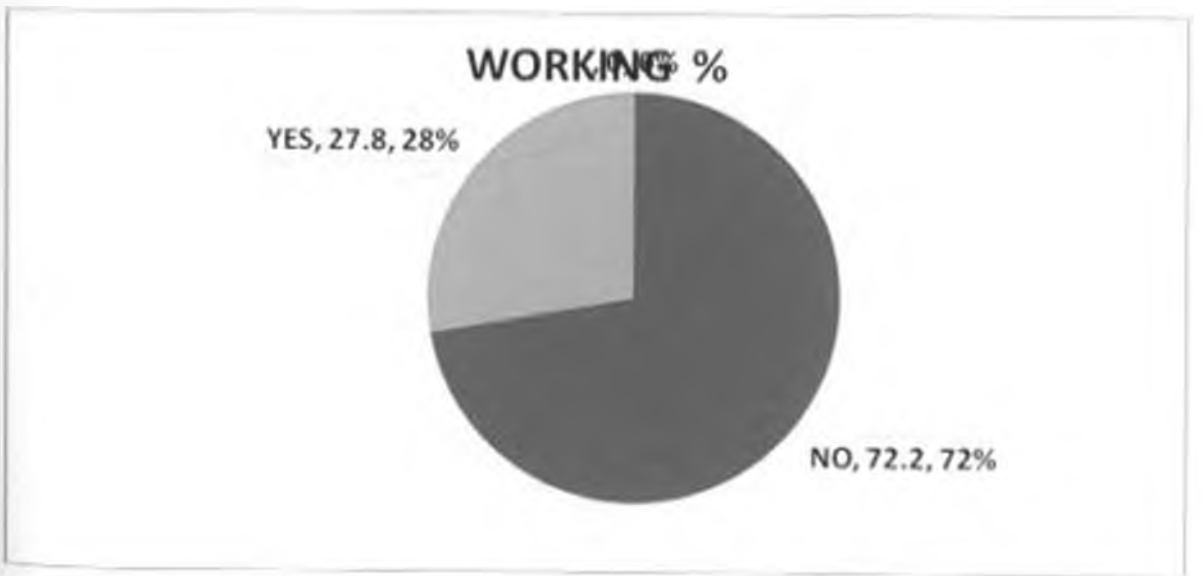


Figure 4

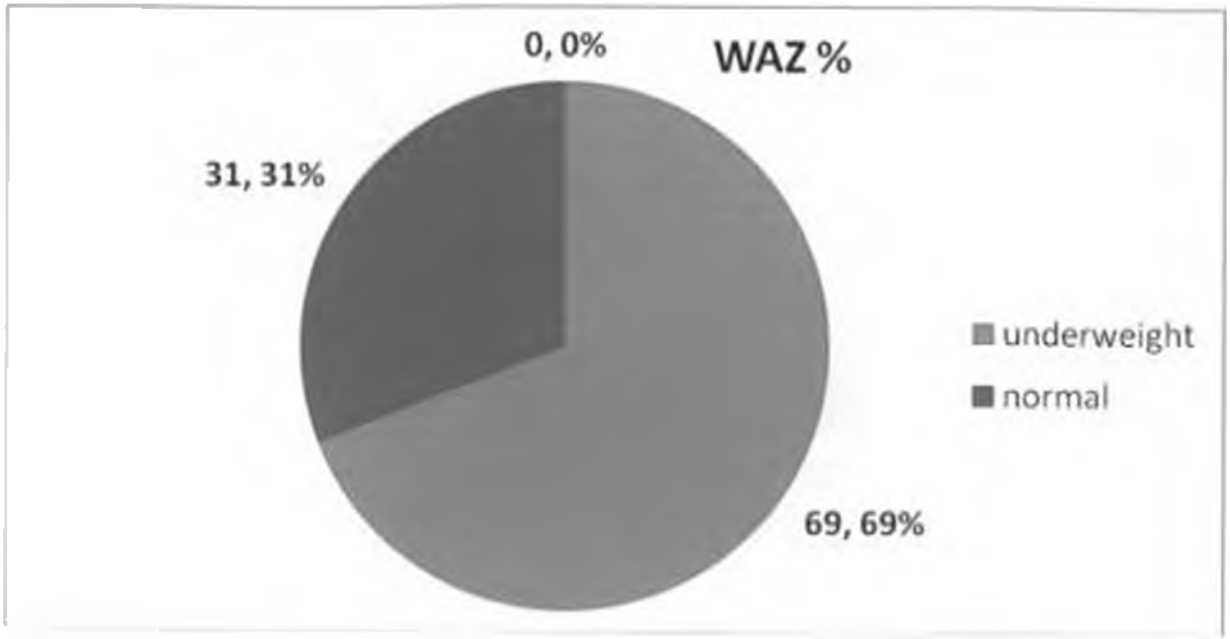


Figure 5

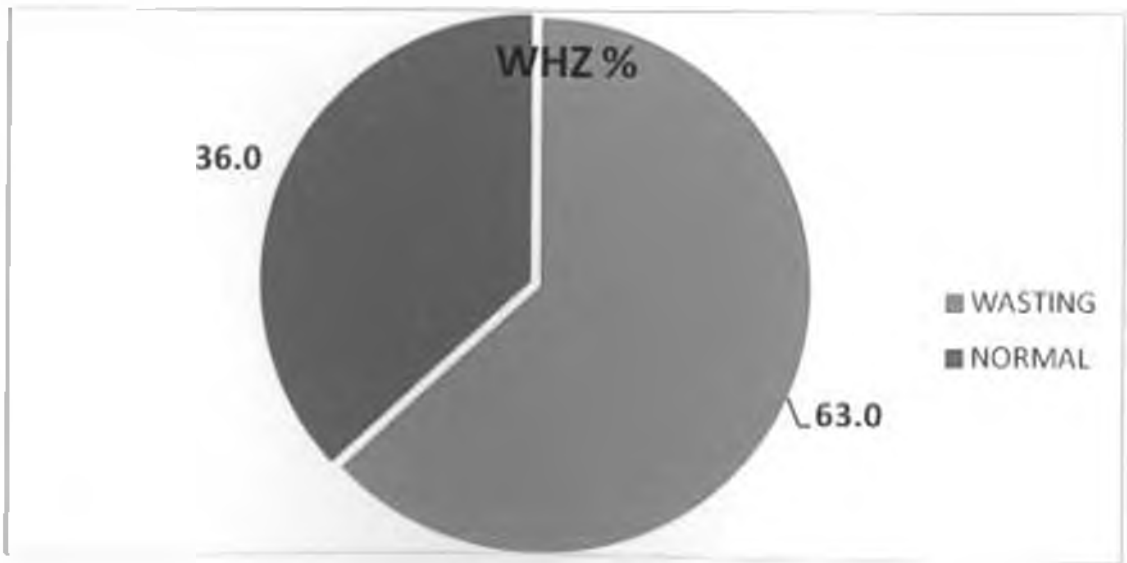
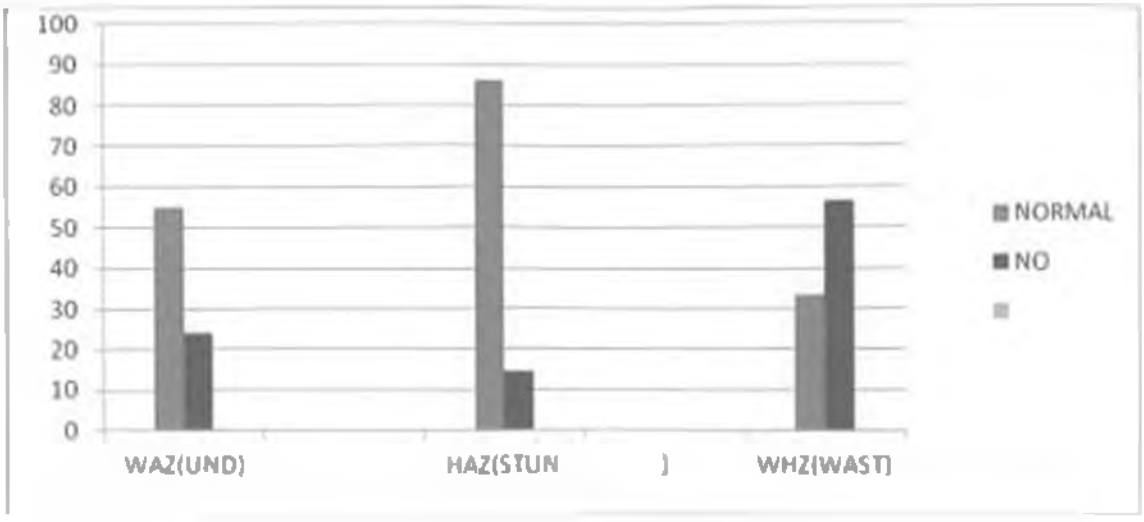


Figure 6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80
70
60
50
40
30
20
10
0
WAZ(UND)
HAZ(STUN)
WHZ(WAST)
NORMAL
NO