

**DETERMINATION OF CALCIUM AND
PHOSPHORUS IN STAPLE FOODS IN
RICKETS PREVALENT AREAS IN
KADUNA STATE**

BY

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DECLARATION

I hereby declare that this research work was carried out by me under the joint supervision of Prof. M Garba and Dr.M.T Odunola. It has not been accepted in any previous publication for a higher degree else where. The works of other researchers and investigators are acknowledged and referred to accordingly.

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CERTIFICATION

The thesis entitled “Determination of calcium and phosphorus in staple foods in rickets prevalent areas (Gonin Gora, Jankasa and Kaso) in Kaduna State” by TURAKI ZIK KALIK meets the regulation governing the award of the degree of Masters of Science of Ahmadu Bello University, and is approved for its contribution to knowledge and literary presentation.

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DEDICATION

This research work is dedicated to my late father, my mother, beloved wife (Asabe Turaki) and children (Tatu and Kelly) then to the entire Turaki's family.

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ABSTRACT

The beginning of the 20th century witnessed the epidemic of nutritional rickets among children in many countries of Asia, North America, Northern Europe and Africa. It was observed that a low dietary intake of calcium among rural children is a major contributor to rickets disease. Nutritional rickets remain a problem in many countries of the world despite a decline in the prevalence of the condition in many developed countries. Prevalence of rickets remain high in Nigeria among infants and young children and it appears to be a consequence of calcium malnutrition. In Nigeria, 2.4% of households have been reported to have children with rickets, while the prevalence of the bone disorder has been reported to be as high as 14.9% especially central Nigeria. It is therefore imperative to evaluate the mineral content of some common foods consumed by rickets disease prevalence areas of Kaduna state namely: Gonin Gora, Jankasa and Kaso. This study was aimed at determining the calcium and phosphorus content of some staple in the communities. Atomic Absorption (SHIMADZU MODEL 650) was used to determine the levels of calcium and phosphorus in the food samples which were kindly provided by the occupants of the communities. Wet digestion method was adopted for all the food sample preparations. The results obtained showed that calcium levels in all foods were low with mean values of 0.05611 ± 0.02 S.E.M., 0.0687 ± 0.04 S.E.M and 0.1272 ± 0.06 S.E.M in Gonin Gora, Jankasa and Kaso respectively which were less than the 0.1300mg/l allowable limit. Phosphorous levels where high with mean values of 0.5382 ± 0.09 S.E.M and 0.4308 ± 0.01 S.E.M greater than 0.4126mg/l in Goni gora and Kaso respectively. However Phosphorus 0.2617mg/l in Jankasa was lower than 0.4126 allowable limit. Rickets among rural children has been reported to be attributed to low dietary calcium intake. The low levels of calcium in foods and or the low calcium intake with high phosphorus intake could be the major causes of the disease in these settlements especially during the period of the children growth.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 Rickets

This is a bone disease of children. It results in progressive softening and weakening of the bone structure as a result of loss of calcium and phosphate from the bone, which eventually causes destruction of the supportive gland matrix. (Blok *et al.*, 1998; Rowe, 2001; Rajakumar, 2003). The beginning of the 20th century witnessed the epidemic of nutritional rickets among children in many countries of Asia, North America, and northern Europe (Pettifor, 1978). Nutritional rickets is most common disease associated with severe bone mineral deficiency in very small children (USDA, 2007). It has been reported that with the discovery of the ultraviolet light (sunlight) in the treatment of vitamin D₃ deficiency rickets and the isolation of vitamin D, effective and inexpensive methods of treating nutritional rickets became available. In spite of these developments, rickets remains a major public health problem in many developing countries (Dux *et al.*, 2001; Muhe *et al.*, 1997) and its prevalence is reported to be on the increase in several developed countries (Finberg, 1979; Iqbal *et al.*, 1994; Rowe, 2001; Delucia *et al.*, 2003).

For long, it was generally accepted that nutritional rickets is caused by vitamin D deficiency alone and that dietary calcium deficiency might only exacerbate the disease in the presence of vitamin D deficiency (Irwin and Kienholz, 1973). More recent studies on the pathogenesis of rickets have deficiency of calcium plays a significant role. Considering the suggested that, among older children in developing countries in particular, dietary importance of

calcium, vitamin D and phosphorus in bone mineralization, USDA (2007) recommended that an adequate supply of these minerals be incorporated into the context of diet as a whole.

1.2 Vitamin D deficiency as a cause of rickets

Supply of vitamin D is required for adequate absorption of calcium as it is involved in maintaining bone mineral homeostasis and regulating renal excretion. Vitamin D is primarily derived naturally from cutaneous synthesis with exposure to ultraviolet radiation from sunlight (Greer *et al.*, 1984). In populations with limited exposure to sun because of environment, clothing or housing conditions, vitamin D is primarily the aetiology of most cases of nutritional rickets (Fitzpatrick *et al.*, 2000). In some parts of the world, rickets has been shown to be most prevalent at the ages of 3-18months (Salimpour, 1975; Molla *et al.*, 2000). In developing countries, especially in the rural and sub-urban communities, rickets is common in under-fives (Akpede *et al.*, 1999; Thacher *et al.*, 2000). Exclusive breast feeding, maternal vitamin D deficiency, living in temperate climates, lack of sun light exposure and darkly pigmented skin have been identified as factors that are important in the pathogenesis of rickets. There are reports that in the Middle East and other more-tropical climates, social and religious customs that prevent sun light exposure appears to be important (Molla *et al.*, 2000; Bassir *et al.*, 2001; Atiq *et al.*, 1998). In Latin America population, vitamin D is likely to be as a result of exposure to sunlight and resultant UV-B conversion in the skin of provitamin D to Vitamin D. Vitamin D status has been reported to be greater in light-skinned than in dark-skinned children during the summer in temperate countries (Oliveri *et al.*, 1993). Increased urbanization with resultant decreased time spent outdoors and increased air pollution may also

contribute to lower sunshine exposure in children. In general, skin absorption of UV-B is related to melanin and is therefore lower in darker-skinned populations. Rickets is not widely reported in Latin America. A much higher incidence of rickets was reported in southern Argentina than in northern areas associated with up to 52% incidence of very low hydroxyl vitamin D concentrations in southern Argentina compared with 9% incidence in Buenos Aires (Oliveri *et al.*, 1990).

It is observed that breast milk normally contains insufficient concentrations of vitamin D or its metabolites (Specker *et al.*, 1985; Hollis *et al.*, 1981). To increase maternal breast milk concentrations of vitamin D to levels that maintain the vitamin D status of the breast-fed infant, high dose of maternal vitamin D supplements of up to 2000 IU/d is needed (Ala-Houhala *et al.*, 1986). Research work conducted by Specker *et al.* (1985) revealed that the vitamin D status of breast-fed infants is correlated with sun light exposure rather than the vitamin D content of maternal breast milk.

Breast-fed infants are generally protected from vitamin D deficiency rickets during the first few months of life, because vitamin D metabolites, especially 25-hydroxyvitamin D [25(OH)D], do cross the placenta, such that neonatal 25(OH)D concentrations are approximately two-thirds of maternal values (Hilman and Haddad, 1974). Studies carried out in Middle East, North America and northern Europe have indicated the prevalence of low circulating concentrations of 25(OH)D during pregnancy (Henriksen *et al.*, 1995; Daaboul *et al.*, 1997; Grover and Morley, 2001 Datta *et al.*, 2002). Factors found to be important include increased skin pigmentation, immigration from non-European countries to countries of high latitude, limited skin exposure as a result of religious and social customs and vegetarian diets. Congenital rickets has been

observed in such situations, although its occurrence is rare (Mohapatra *et al.*, 2003; Anatlilotaki *et al.*, 2003), and neonatal hypocalcaemia is more frequent among neonates born to mothers with low 25(OH)D concentrations than among those born to mothers with normal vitamin D status (Zeghoud *et al.*, 1997). The development of clinical vitamin D deficiency rickets is dependent not only on vitamin D deficiency [circulating concentrations of 25(OH)D] but also on the duration of the deficiency, on the rate of child's growth (which influences calcium demands), and on dietary calcium content. Several studies have documented spontaneous healing of radiologically evident rickets during summer months and seasonal fluctuations in serum parathyroid hormone in northern Europe, North and South America (Gupta *et al.*, 1974; Guillemant *et al.*, 1995).

The seasonal changes in 25(OH)D concentrations, the lag period between the decrease in 25(OH)D concentrations and the development of biochemical, radiologic, or clinical rickets, and the influence of diet on the development of rickets have made it difficult to define a clear division between vitamin D deficiency and sufficiency on the basis of serum 25(OH)D concentrations. Nevertheless, there is widespread agreement in the paediatric literature that vitamin D deficiency should be defined as 25(OH)D concentrations of <10-12ng/ml (Greer, 2003; Shaw and Pal, 2002). In past decade, considerable discussion took place regarding vitamin D sufficiency and what should be considered normal serum concentration of 25(OH)D (Vieth, 1999; Chapuy *et al.*, 1997). Vitamin D insufficiency has been used to indicate serum 25(OH)D concentrations between those associated with vitamin D deficiency and those considered to be optimal. Vitamin D deficiency is associated with mild elevation of parathyroid hormone concentrations, although values remain within the

reference range (Jesudason *et al.*, 2002). Among young infants, it appears that parathyroid hormone concentrations increases only when 25(OH)D concentrations are in the vitamin D₃-deficient range (Zeghoud *et al.*, 1997). Studies with adolescents found that parathyroid hormone concentrations increased when 25(OH)D concentrations decreased below 12-16ng/mL (Guillemant *et al.*, 2001; Outila *et al.*, 2001), whereas Docio *et al.*(1998) suggested that perturbation in calcium homeostasis occur among prepubertal children when 25(OH)D concentrations are between 12 and 20ng/mL. Therefore, it appears that if the concept of vitamin D insufficiency is valid for children, values are very close to upper limit of what is defined as vitamin D deficiency, a pattern that is very different from that for adults (Tangpricha *et al.*, 2002).

1.3 Dietary calcium deficiency as a cause of rickets

Low dietary calcium intakes are thought to produce osteoporosis, rather than osteomalacia, in the adults through secondary hyperparathyroidism and increased bone turnover. Similar view was held for children until the 1970s, when several case reports of rickets among infants attributable to extremely low dietary calcium intakes in the presence of adequate vitamin D were published (Koof *et al.*, 1977; Legius *et al.*, 1989). At that time, it was suggested in South Africa that rickets among rural children was attributable to low dietary calcium intakes and not vitamin D₃ deficiency (Pettifor, 2004). These children were presented with active rickets at the ages 6-16 years (Pettifor *et al.*, 1978). In most Tropical and sub-Tropical countries, vitamin D deficiency is considered unlikely as a cause of rickets because children spend considerable part of the day playing outside in the sunshine; this was confirmed by normal serum 25(OH)D

concentrations and elevated 1,25(OH)₂D concentrations. Also, the radiologic, histologic, and biochemical features of rickets improved with calcium supplements in these sunny areas (Pettifor *et al.*, 1981; Marie *et al.*, 1982). Since then, several reports from Nigeria highlighted the role of low dietary calcium intakes in the pathogenesis of rickets among children in that region (Oginni *et al.*, 1996; Okonofua *et al.*, 1991; Thacher *et al.*, 1997; Oginni *et al.*, 2003), although vitamin D deficiency was also proposed (Laditan and Adeniyi, 1975; Ekanem *et al.* 1995). In other parts of the world like where rickets has been reported to be principally due to dietary calcium deficiency include Bangladesh and India (Fischer *et al.*, 1999; Balasubramnian *et al.*, 2003). It has been reported that unlike in South Africa, the Nigerian children with rickets tended to be younger, with a mean age of presentation of four years and to live in urban environments (Pettifor, 2004). Thacher *et al.* (Thacher *et al.*, 1999) showed that calcium supplements alone or in combination with vitamin D were equally effective in treating the disease and were more effective than vitamin D alone. Not only were 25(OH)D concentrations normal for most children at presentation but also among those treated with calcium alone. These findings, thus, highlight the importance of low calcium intakes in the pathogenesis of the disease.

Diets in South Africa and Nigeria characteristically have high content of unrefined cereal, which raises the possibility of dietary constituents such as phytates impairing calcium absorption (Pettifor, 2004).

It was also reported that in both South Africa and Nigeria, there is a high prevalence of history of bone deformities and clinical rickets among family members and the first degree relatives of children with active rickets (Thacher *et al.*, 2000). Although it is possible that similar environmental and socio-economic

factors among family members might be responsible for the disease, this finding does not raise the possibility that genetic factors might also play a role. In developing countries where calcium intakes are characteristically low and the population relies heavily on cereal-based staples, with few or no dairy products, dietary calcium deficiency appears to be the major cause of rickets among children outside the infant age group (Pettifor, 2004).

1.4 Relationship between vitamin D and dietary calcium intakes in the development of rickets

It has been known for years that low calcium intakes exacerbate vitamin D deficiency rickets (Pettifor, 2004). The deleterious effects of low dietary calcium intakes in the development of rickets among vitamin D deficient animals were demonstrated by Mellanby (Pettifor, 2004). More recently, similar effect with the addition of unrefined maize to vitamin D deficient diets for baboons was demonstrated (Sly *et al.*, 1984). However the mechanisms by which the exacerbation of the development of rickets due to low calcium intakes occurs are not clear. One of the most studied communities with high prevalence of rickets among humans has been the Asian community in the United Kingdom; these communities have been noted for years to be associated with high prevalence of rickets and osteomalacia (Dunningan *et al.*, 1962; Ford *et al.*, 1972; Dent *et al.*, 1973; Goel *et al.*, 1976; Ford *et al.*, 1976). Some of the proposed pathogenic mechanisms put forward to explain the development of rickets include lack of sun light exposure, increased skin pigmentation, lack of dietary vitamin D intake, genetic predisposition, low calcium diets and high phytates contents of diets. It has been demonstrated in rats model that elevation of 1, 25(OH)₂D

concentrations through feeding of the rats with low-calcium or high-phytate diets resulted in 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 (Clements *et al.*, 1987). It has also been shown that infusion of 1,25(OH)₂D led to reduction in serum 25(OH)D half-life and a 7-fold increase in 24,25-dihydroxyvitamin D production by the kidney (Halloran *et al.*, 1986). In human studies, the half-life of 25(OH)D was reduced by nearly 40% among patients with partial gastrectomies, secondary hyperthyroidism, and elevated 1,2(OH)₂D concentrations (Davies *et al.*, 1997), and a similar findings were noted among patients with intestinal malabsorption (Batchelor *et al.*, 1982) and subjects consuming high-fiber diets (Batchelor and Compston, 1983). It was proposed based on these findings that 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 of vitamin D status (Clements, 1989).

The role of low dietary calcium intakes in the pathogenesis of vitamin D deficiency is probably greater than originally recognized. This has , thus been proposed as a mechanism of rickets in children in India (Balasubramania *et al.*, 2003) and among toddlers in the United States (DeLucia *et al.*, 2003) and probably accounts for the lower 25(OH)D concentrations among rachitic subjects, compared with control subjects in Nigeria (Thacher *et al.*, 1999). There is a general consensus that calcium is well absorbed from human milk, with values for net calcium retention of 50% of intake (Fomon and Nelson, 1993; Abrams *et al.*, 1997). However, the efficiency of calcium absorption from any food source is likely to depend more on the total amount added and its interaction

with other food components such as vitamin D, phytate, oxalate and dietary fiber than on the relative solubility of the type of calcium salt added as fortificants (Heaney *et al.*, 1990a). If calcium absorption is low, then each of these factors significantly affects the efficiency of calcium absorption (Heaney *et al.*, 1990b). In Nigerian children with rickets, the capacity to absorb calcium is not impaired; however, fractional calcium absorption increases after the resolution of active disease. Calcium absorption may be inadequate to meet the skeletal demands of children with rickets during active phase of the disease (Graff *et al.*, 2004). Other studies carried out in Nigeria indicated that there is no relationship between protein deficiency and development of rickets (Walter *et al.*, 1997). Lead toxicity was common in Nigeria, with 70% of children having elevated levels of the metal in their bloodstream. It was however observed that this potentially competing divalent cation was not related to calcium deficiency or rickets (Fischer *et al.*, 2000). Results of studies conducted in Nigeria also revealed that their calcium absorption did not vary between rachitic children and non-rachitic ones (Fischer, 2007). The study further revealed that genetic factors were however found to have link with rickets (Fischer, 2007), while a newly described mutation seems to explain the disease in a sub-set of patients (Levine *et al.*, 2007).

Rickets therefore remained common in many parts of the world and calcium deficiency, not vitamin D deficiency, was the important cause of the disease (Thacher, *et al.*, 2000; Graff *et al.*, 2004; Thacher *et al.*, 2006).

1.5 Phosphorus requirement in infants

The principal sources of phosphorus in infants include cow's milk, cereal foods, meat and some soda beverages. Because of the ubiquity of these foods, intakes of phosphorus are generally high relative to requirements. In most instances, phosphorus intake far exceeds that of calcium (USDA, 2001). The functional consequence of these high intakes, especially in the presence of low calcium intakes, remains a topic of considerable controversy (Sax, 2001). It has been suggested that high phosphorus intakes contribute to hypocalcaemia and fractures in children and that further control studies are needed to evaluate these relationships (Wyshak, 2000). Fortification of diets of children with phosphorus is therefore highly unlikely.

1.6 BELOW ARE CALCIUM AND PHOSPHORUS TABLES.

TABLE 1.6.1

The table below shows a number of phosphorus rich foods along with their phosphorus content in milligrams (mg). (USDA 2007)

Food	Serving	Phosphorus (mg)
Milk, skin	8 ounces	247
Yogurt, Plain non fat	8 ounces	383
Cheese, Mozzarella part skin	1 ounce	131

Egg	1 large, cooked	104
Beef	3 ounces, cooked	173
Chicken	3 ounces, cooked	155
Turkey	3 ounces, cooked	173
Fish, halibut	3 ounces, cooked	242
Fish, salmen	3 ounces, cooked	252
Bread, whole wheat	1 slice	64
Bread enriched white	1 slice	24
Carbonated cola Drink	12 ounces	44
Almonds	1 ounce	139
Peanuts	1 ounce	101
Lentils	1/2cup, cooked	356

Table 1.6.2 (USDA 2007)

Recommended Adequate Intake by the IOM for Ca²⁺	
Age Group	Mg/day
0 to 6 months	210
7 to 12 months	270
1 to 3 years	500
4 to 8 years	800
9 to 13 years	1300
14 to 18 years	1300
19 to 50 years	1000
51+ years	1200

Table 1.6.3 (USDA 2007)

Tolerable Upper Intake Level (UL) for Phosphate	
Age-Group	UL (Mg/day)
Infant 0-12 months	Not possible to establish
Children 1 – 3 years	3,000
Children 4 – 8 years	3,000
Children 9 – 13 years	4,000
Adolescents 14 -18years	4,000
Adults 19 – 70years	4,000
Adults 70 years and older	3,000
Pregnancy	3,500
Breast feeding	4,000

1.7 Scope and Objective

Visitors to these villages with a population of about four thousand people can not fail to notice the strange features in children here. They all appear to be suffering from a disease condition called Rickets the cause of which medical experts attribute to nutritional disorder characterized by softening and weakening of bones in children resulting in skeletal deformities. Giving birth in these villages is normal but the feelings that run through parents can better be imagined. However, to a discerning mind or visitor to the area the reason is not far fetched as children born here end up with deformities. The prevalence rate of the disease in these villages is almost 100% as there is hardly a family without a child afflicted with the disease. Children in these villages with the disease were not born with these deformities; it began when they started walking. Driven by some kind of superstition, men here see their wives and mothers of the children as being possessed by some evil spirit. This belief as an explanation for the medical condition of the children is strongly rooted amongst the Gbaygi people, such that when new babies are born, parents are seldom happy. What hits them is the thought of what will become of their children. According to Kitz (1997), when the case was first reported in 1997 only about twelve (12) cases were known. Today, over two hundred and fifty (250) cases are known and the fear is that in a few years time the may double.

Information on the mineral contents of staple locally consumed foods in areas where rickets remain prevalent may provide clues on the quantity of such minerals to be provided as fortificants or supplements in diets of such people. The study is centred on the evaluation of calcium and phosphorus contents of

some foods in rickets prevalent areas of Kaduna state using Atomic Absorption Spectrophotometre (AAS). This study would help shed more light on the role of calcium and phosphorus deficiency in the development of rickets so that a community based programme could be developed for execution in some of these areas.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Statement of research problem

Nutritional rickets remains a problem in many countries of the world, despite a decline in the prevalence of the condition in many developed countries since the development of vitamin D and the role of ultraviolet light in prevention. The disease has continued to be a source of unhappiness in many communities, especially among infants who are exclusively breast-fed, infants and children of dark skinned immigrants living in temperate climates, infants and their mothers in the Middle East and infants and children in many developing countries in the tropics and subtropics such as Nigeria, South Africa and Bangladesh (Levine *et al.*, 2000, 2007; Pettifor, 2004; Thacher *et al.*, 2006). In Nigeria, 2.4% of households have been reported to have children with rickets while the prevalence of the bone disorder has been reported to be as high as 14.9%, especially in central Nigeria (Akpede *et al.*, 1999; Fischer, 2007).

Although vitamin D deficiency has been reported to be the major cause of rickets in many countries of the world, its role in the development of rickets in the Tropics and sub-Tropics is of limited value (Pettifor, 2004). Inadequate exposure to ultraviolet light, religion and customs have also been ruled out in the development of rickets in Tropical countries like Nigeria (Okonofua *et al.*, 1991; Thacher *et al.*, 1997; Pettifor, 2004). Impaired intestinal absorption of calcium, which has been considered to be a possible cause of rickets in some countries, contributes little in its development among Nigerian children (Pam *et al.*, 2004). Also, results of studies conducted in Northern Nigeria indicated that there was no

link between protein deficiency and development of rickets (Walter *et al.*, 1997). Fischer (2007) reported that the calcium intake of Nigerian children is uniformly low and vitamin D deficiency was not an aetiologic factor since the children have adequate levels of the vitamin. The same author also reported that although Nigerian children have high levels of lead, a divalent ion that competes with calcium, in their bloodstream, this was discovered not to be a factor in the development of rickets.

Prevalence of rickets remains high in Nigeria among infants and young children and appears to be a consequence of calcium malnutrition (Laditan and Adeniyi, 1975; Okonofua *et al.*, 1991; Ekanem *et al.*, 1995; Thacher *et al.*, 1997). Although there are sufficient evidences to suggest that genetic factors could be playing a part in the development of rickets in some communities in Nigeria (Fischer *et al.*, 2007), the general consensus is that calcium intakes, coupled with high phosphorus intakes, is considered to be the major aetiologic factor. Determining dietary calcium requirements requires the evaluation of the total amount of calcium needed by the skeleton during the ages of interest (USDA, 2007). Conversely, the currently available data are inadequate to establish the precise amounts of these nutrients that would be required for such supplement even in developed countries (Wyatt and Tejas, 2000); while in developing countries like Nigeria, there is dearth of information on the mineral contents of the diets that form the staple foods. It is however known that the bulk of the diets in these developing countries are cereal foods, which have high phosphorus content thereby exacerbating the development of rickets, especially, in young actively growing children (Pam *et al.*, 2004 Fischer, 2007).

Since the possible role of impairment of intestinal calcium absorption, vitamin D deficiency, religions and customs have been ruled out as the cause of rickets in Nigerian children (Thacher *et al.*, 1997; Akpede *et al.*, 1999; Pam *et al.*, 2004), it becomes imperative to evaluate the nutrient content of some of the common foods consumed by the average Nigerians with the view to ascertaining the mineral(calcium and phosphorus) content of such foods so that community-based intervention programme could be designed for implementation to curtail the incidence of the disease.

2.2 Justification

Nutritional rickets has remained a common occurrence among Nigerian children (Akpede *et al.*, 1991; Graff *et al.*, 2004; Pam *et al.*, 2004) and has been shown to respond adequately to calcium supplementation. Calcium intake in foods may be too low to meet the skeletal demands of children during the active phase of the disease (Pam *et al.*, 2004). The extent of calcium deficiency in developing countries is uncertain, but it is known that calcium consumption is extremely low in many locations where dairy products and fish are not eaten (Allen, 2001). Information on the mineral contents of locally consumed foods in areas where rickets remains prevalent may provide clues on the quantity of such minerals to be provided as fortificants or supplements in diets of such people, hence, the need to embark on studies to evaluate mineral content of some of these foods in rickets prevalent areas. The study would help shed more light on the role of calcium deficiency in the development of rickets so that a community-based programme could be developed for execution in some of these areas.

2.3 The Subjects Of This Study

2.3.1 Their Background

Jankasa and Kaso are rural land locked villages located in the remote interior of Chukun Local Government Area of Kaduna State Nigeria. They are twenty five kilometers (25kms) South of Kaduna town on the Kaduna-Kachia road. While, Gonin-gora, unlike Jankasa and Kaso, is just a few kilometers South of Kaduna town on the Kaduna-Abuja road. The natives of these villages are Gbaygi, Kadara, Fulani and Hausa. Though there are very few minority tribes scattered all over the region.

In terms of origin, the Hausa and Fulani claimed to be descended from the Fulani (Bororo) origin who were scattered all over and originated from North Africa. Some of the Bororo lost their cattle and settled in these areas for farming. According to them, cattle were the up-keep and as they dwindled, another means of livelihood had to be found. These communities believe they would have been bigger than they are except for the effects of tribal feuds, harsh climates and tribal wars combined to keep the population down. Their population began to grow with the arrival of the British and Missionaries who brought peace, medical help and educational services. As seen, the people live in a non- mountainous, dry and plain land. Evidently, the cause of this was the need for more land for farming and better homes. Each of these unparallel inhabitants have their own language. The Gbaygi, speaks Gbaygi language, Kadara speaks Kadara language, Fulani speaks Fulanchi. Hausa speaks Hausa which is the most used language. Communication in Hausa is done through out these areas. As the case is in traditional African society where everybody is a worker, Gbaygi and Kadara are all farmers, hunters and herdsman. There is no other way of earning a living from

these communities. Fulani still keep their cattle as a means of livelihood but not in large number. They milk cows and process the milk for commercial purpose. While the Hausa's, are both farmers and traders. Some of them buy farm produce and transport it to a distance for sale to earn interest.

2.3.2 Their Religion and Culture

The religion of these people is a combined belief in Allah (a Supreme God) with belief in gods, the spirit of ancestors and fetishes. About ten percent (10%) of the population practice animism while about sixty percent (60%) are Christian and the remainder practice Islam.

At the base of their traditional religion is the concept of the “ultimate Reality”. The conception of God or the Supreme Being came into being to explain the origin of man and the world. At the base of Gbagyi and Kadara religious philosophy, “God is the abstract idea, the cause”. He created the Earth and Man. He is an all knowing and all seeing God. He is “transcendent,” living in heaven from where he rules the universe. The foundation of this religion and others rests on the mystery of the origin of man and the universe. These people religion relates the beliefs in deities to daily life (which has led to the creation of still lesser gods or spirits which control moral and social life), and enterprises such as agriculture, hunting, marriages etc. They also believe in the spirits of dead ancestors. The dead ancestors living in the spirit world serve as “solicitors” and “advocators” on behalf of their living relatives. Despite the infiltration of Christianity and Islamic ideas into the Area, the religious concepts are still deeply entrenched in the minds of the inhabitants of these areas including those who have accepted Christianity or Islam. These Christians or Muslim can not be

regarded as consummate converts. They accepted Christianity or Islam because it affords them an additional channel of reaching God.

As part of their religious believes and culture these people especially the Gbaygi and Kadara care for each other (provided he/she is willing to work). People work for one another. The work is essentially distributive. They practice aid (gai-ya), a term for aid where people take turns helping each other working in the fields. It also means the host either is just married or is building a new house and needs assistance from the community. This type of occasion requires entertainment after a full day of working hours.

2.3.3 Their Foods

Whatever the culture, Tradition, Religious belief of these of people, the fact is that they eat food to live. The food provides energy, vitamins, minerals, fat which are all vital to the body. Different foods contain different nutrients. No single food contains all the nutrient required by body in amount the body needs. Therefore, these people have to take varieties of foods to what is needed by their bodies for their daily activities. They have different varieties of what forms their staple foods. Amongst many are: Milk, cheese, dried legumes, cabbage ,lettuce, onions, and Spinach, whole grains, green leafy, fruits, fish, meat, grains, bread, cereals, sea food, legumes, Bananas, Beans, potatoes, However, important nutritional decisions need to be made for the good health of individuals, or group such as the very young and the aged. The young age stands at a great risk of health hazard as manifested now in the communities when wrong nutritional decisions are taken. Fortunately, the case with these people is not that of decision, poverty, or lack of nutritional knowledge because they are blessed with all sort of

foods, most of which are fresh and carries the nutrients undistorted. Most vegetables are fetched directly from their farms. The milk known as nono (caw milk) is used fresh. With all these, one wonders why the scourge of RICKETS in these communities hence the need for this study.

2.4 Theory of Atomic Absorption Spectroscopy

This is a quantitative method of analysis that is used to analyse many metals and non metals in solution (Ana, 2005). In this method, electromagnetic radiation (EMR) is absorbed or emitted by atoms and measured. All atoms can absorb EMR, and the wavelengths at which is absorbed or emitted is exclusive for a particular element. The science of AAS has yielded three techniques for analytical uses namely: Atomic Absorption, Atomic emission and Atomic fluorescence. In all these processes, it is important to understand every atom and the processes involved in each technique.

2.4.1 Atomic Absorption

The atom of an element is made up of nucleus which is surrounded by electrons and every element has a specific number of electrons which move around the atomic nucleus in an orderly way. The most stable state of every atom is known as the “ground state”, which has stable electronic configuration and thus lowest energy. If energy of right magnitude is applied to an atom , it absorbs the energy and an outer electron will be excited (i.e less stable configuration). Since this state is unstable, the atom will spontaneously and immediately come back to its initial, stable orbital position. As a result of this, the light energy equivalent to the amount initially absorbed will be emitted. The wavelength of radiation energy has a direct relation to the electronic transition which occurred. Since, every

element has unique electronic structure, the wavelength of light emitted is also a unique property of individual element. The process of excitation and emission to ground state is involved in all the three fields mentioned above. The energy absorbed or emitted is measured and used for analytical purposes. The capability of an atom to absorb very specific wavelength of light is utilized in atomic absorption spectroscopy technique. Thus it offers several advantages over other methods.

2.4.2 The process of Atomic Absorption Spectrophotometry

The amount of light measured which is in resonant wavelength absorbed as light passes through a cloud of atoms is the quantity of interest. As the number of atoms in the light path increases, so also the amount of light absorbed increases predictably. By measuring the amount of light absorbed, a quantitative determination of the amount of analyze element present can be made. And the use of special light source and careful selection of wavelength allow the specific quantitative determination of individual element in the presence of others.

The amount of atoms required for atomic absorption measurement is produced by supplying enough thermal energy to the sample to dissociate the chemical compounds into free atoms (this is achieved by aspirating a solution of the sample into the flame aligned in the light beam). The ease, speed, precision and accurate determination has made this atomic absorption technique method the most popular in the determination of metals.

2.4.3 Quantitative Analysis by Atomic Absorption

Light at the resonance wavelength of initial intensity is focused on a flame cell containing ground state atoms. The initial light intensity is decreased

by an amount determined by the atom concentration in the flame cell. The light is then directed into the detector where the reduced intensity is measured. When comparing the reduced intensity (I) to initial intensity (I₀) of light, the amount of light absorbed can be measured or determined.

Transmittance (T) is the fraction of initial light which passes through the flame cell to fall on the detector. It is defined as the ratio of the final intensity to initial intensity.

$$\text{i.e. } T = \frac{I}{I_0}$$

while the absorbance (A) is the mathematical quantity given by:

$$A = \log \left\langle \frac{I_0}{I} \right\rangle .$$

This follows a linear relationship with concentration C.

Therefore, A = abc

Where: A = Absorbance

a = absorption coefficient

b = length of light intercepted by absorption species

c = concentration of the absorbing species.

The equation is known as the BEER'S LAW, which states that the absorbance is directly proportional to the concentration of the absorbing species for a given set of instrumental conditions.

The directly proportional behaviour between absorbance (A) and concentration (C) is observed in atomic absorption. This can clearly be seen on a graph when absorbance of standard solutions containing known concentrations of analyte are measured and the absorbance data are plotted against concentration .

After such, calibration is achieved or established, the absorbance of solutions of unknown concentrations may be measured and the concentration determined from the calibration curve (Beaty and Kerber,1993).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 MATERIALS

3.1.1 CHEMICALS

The reagents used include the following:

Sulphuric acid (BDH) Chemicals England.

Nitric Acid (BDH) Chemicals England

Perchloric acid (Analar grade) May and Baker, England.

Ammonium molybdate (Analar grade) May and Baker, England

Ammonium metavanadate (Analar grade) BDH Chemicals England

Hydrochloric acid (BDH) Chemicals England

Hydrochloric acid (BDH) Chemicals England

Ionization buffer: Lanthanum Chloride, Potassium Chloride (Aqua Regia).

3.1.2 EQUIPMENT AND GLASSWARES

Atomic Absorption spectrophotometer Shimadzu Model 650.

Block digester

Fume cupboard

Spectronic-20

Acetylene burner

Mortar

Analytical Weighing Balance Machine, (Mettler AE 240).

Kjeldahl Distillation Apparatus

Hot plate

Retort stand

P^H meter (Fisher Brand Model 300).

Vacuum dryer.

Spatula

Storage plastic bottles

Digestion tube

Pipets

Glass beads

Beakers 50ml, 100ml
Volumetric flasks 25ml, 50ml
Whatmann's filter paper
Wire loop
Glass vials
Quartz Crucible
Polyethene bags
Conical flask
Glass Petri-dishes
Glass spreader
Measuring cylinder:50ml
Test tubes

3.2 METHODS

3.2.1 Description of Sampling Areas

The study is conducted in Kaduna state which is strategically located in the North Central of Northern Nigeria. The state from North-East borders Kano, Jigawa, Bauchi, Plateau, Nasarawa, FCT, Niger and Zamfara.

The study areas/sampling points are areas most affected by the scourge of the disease, Rickets. These areas include: Jankasa, Kaso and Gonin Gora all in the southern part of Kaduna Metropolis. Jankasa and Kaso are rural land locked villages approximately 25 kilometers along Kaduna- Kachia road while Gonin-Gora is just some few kilometers from the metropolis along Kaduna-Abuja road. The inhabitants of these villages are: Gbaygi, Kadara, Hausa, Fulani and some few minority tribes. Sketch of the areas under study is presented in figure 1.

Fig 1. The Map of Kaduna State and the Study Areas for Sampling.

3.2.2 **SAMPLE COLLECTION AND PRESERVATION.**

The water sample collection and preservation method as described by Department of Water Affairs and Forestry (DWAF) 1992, and the vegetable/foods samples collection and preservation methods as described by Awofolu et al, (2005) were adopted in this study. The treatment of both water and vegetables/foods samples were done following the procedures described by Laboratory Procedure for Fertilizer and Water Analysis (LPFWA) 2004.

The address and place of collection, Date of collection, Name of sample and time of collection were recorded at the collection sites: Samples were all collected in the Months of October and November 2006.

3.2.3 **SAMPLING OF DRINKING WATER/ LIGUID FOODS.**

Drinking water samples were collected from different wells. The liquid foods were also collected from different homes. Both the water and liquid foods were all kept cooled en route to the laboratory and stored at 4⁰C.

After preservation, pending analysis. The pH of the water were determined directly with glass electrode pH Meter. Plastic rubbers were used for the collection of these water samples.

3.2.4 **SAMPLING OF VEGETABLE/FOODS**

Vegetables samples cabbage (*Brassica Oleracea L var capitata*); Lettuce (*Lactuca sativa L*);Maize (*Zea mays*);Okro (*Hibiscus esculentus*) ;Onion (*Allium cepa*); Spinach(*Amarantus spp*) samples were all collected directly in farms within the areas of study. The samples were collected in polythene bags labelled, and taken to the laboratory. The food samples were thoroughly rinsed with the

package water and air-dried at room temperature in the laboratory. The dried samples were ground into fine particles using mortar and pestle.

3.2.5 **PREPARATION OF CALIBRATION CURVES FOR Ca^{2+} , and PO_4^{2-}**

Six working standard solutions of Calcium of concentrations: 0.000mg/l, 1.000mg/l, 2.000mg/l, 3.000mg/l, 4.000mg/l, 5.000mg/l were used for curve calibration in Calcium.

Similarly, Six working standard solutions of Phosphorus of concentrations 0.000mg/l, to 5.000mg/l were also prepared and used for curve calibration in phosphorus concentration determination.

3.3 **SAMPLE TREATMENT FOR AAS ANALYSIS.**

Using pipette, 5ml of drinking water sample was drawn into a 25ml volumetric flask. A 10ml of a buffer i.e Lanthanium and potassium Chloride (A developer) was added, shaken and made up to the volume with distilled water. The solution of the mixture was kept for 30minutes before aspirating into the Machine. All the samples were treated in the same way. Both the working standards and the test samples were measured at 420 nm wavelength for Calcium. The concentration of Calcium was determined from a plot of Absorbance and Concentration as described in calibration curve method (3.2.5).

Calcium and Phosphorus sample solution were prepared each respectively by dissolving 5g of dried ground Calcium/Phosphorus sample in a digestion tube

and 20ml of digestion acid mixture (Nitric, Sulphuric and Perchloric in the ratio of 32:1:4) and three glass beads were added. The resultant solution was then heated at low temperature to avoid fuming and lost of volatile minerals. When the initial reaction subsided, the temperature of the digestion block was slowly increased from 180°C to 200°C. This digestion was continued at this temperature with occasional shaking until there was no visible particles and the colour of the digestion acid was cleared. The temperature was allow to rise from heating source to 240°C and evaporation of the digestion acid ensured. This was confirmed by the formation of white fumes within the digestion tube. When this digestion was completed, the tube was removed from the heating source and the content was filtered through acid washed filter paper in a 100ml volumetric flask using distilled water. From the content obtained, standard solution of each was prepared by dilution.

3.3.1 PREPARATION OF TRI-ACID SOLUTION (Aqua-Regia)

650 ml of Nitric acid, 80ml of perchloric acid and 20 ml of concentrated sulphuric acid were mixed in a 1000ml plastic beaker and stirred until thoroughly mixed (LPFWA,2004)

3.3.2 PREPARATION OF BUFFER SOLUTION.

1. A 50g quantity of ammonium metavanadate was dissolved in 500ml distilled water.
2. A 2.5g quantity of ammonium molybdate was dissolved in 500ml of distilled water.

The reagents were mixed slowly in a 2000ml volumetric flask. Perchloric acid was then added. The solution of the mixture was shaken and made up to mark volume with distilled water.

3.4 **PRE-TREATMENT AND SAMPLE ANALYSIS.**

INSTRUMENTATION.

The concentration of Calcium was determined under the following

Flame type: Air/Acetylene

Lamp: Ca Hollow Cathode

Wavelength: 420nm

3.4.1 **PRE-TREATMENT OF WATER.**

A 10ml of well water samples and liquid foods each was placed into 50ml clean beaker and a 10ml of aqua regia (mixture of Nitric, Sulphuric and Perchloric acids) was added to it. This mixture was heated over a hot plate until reduced to about 5ml. This was transferred into 50ml volumetric flask and made up to the mark with distilled water.

3.4.2 **Determination of Calcium and Phosphorus content.**

Calcium Analysis: To 5ml standard of food sample solution obtained from 3.3 above was added a known concentration of lanthanum (as lanthanum chloride), potassium chloride and 0.1M Hydrochloric acid solutions. This serves as a buffer because calcium react with phosphorus in flame to form calcium complex which is a very stable ionic complex. The resulting solution was aspirated into the AAS machine, and the absorbance was measured at conditions described in 3.4. The

concentrations of calcium was obtained using calibration curve plot. The data obtained from the calibration curve in 3.2.5 shown in our subsequent chapter (4) in Tables **2, 3, and 4**.

Phosphorus Analysis.

Phosphorus concentration was determined in the same manner as Calcium. The reagent 1 molybdate vandate was used as a complexing agent. When the reagent was added to the sample solutions, a coloured complex (yellow/orange) was formed. With the absorbance, the concentration of phosphorus was determine as stated under sample treatment for AAS analysis

CHAPTER FOUR

4.0 RESULTS.

4.1 Calcium concentration in water and food samples.

The results obtained for Calcium concentrations are shown in Tables 2, 3 and 4, Figures 2, 3 and 4 for Gonin Gora, Jankasa and Kaso respectively. Calcium Concentration in all foods in the study areas were found to range from 0.00 to 1.30. The highest concentration of 1.300 was found in Soya Beans in Kaso area and Calcium was not detected in Kunu sample from Gonin Gora (Tables 2, and 4) respectively. Despite the different nature of the samples, the Calcium concentrations have similar pattern.

In Gonin Gora, the concentration was found to be high compared to that of other study areas (Table 2).

4.2 Phosphorus concentration in water and food samples.

The result obtained for Phosphorus concentrations are shown in tables 5, 6 and 7. Figures 5, 6 and 7 for Gonin Gora, Jankasa and Kaso respectively.

The concentrations were found to range from 0.007 to 5.426. The lowest concentration (0.007), in orange and the highest concentration (5.426), in Fish were recorded in Gonin Gora (Table 5 and Figure 5). Jankasa and Kaso samples showed high concentration of Phosphorus (Tables 6 and 7 respectively).

Phosphorus levels were higher in all the study areas compared with Calcium level. It was significant ($p < 0.05$) high for Kaso.

Table 2. Calcium levels (mg/l) for liquid and mg/kg for solids of the different food samples from Gonin Gora settlement of Kaduna state.

S/N	SAMPLE	CONC. OF CALCIUM
1.	Tuwo	0.069
2.	Gari	0.034
3.	Kuka	0.156
4.	Okro	0.044
5.	Karkashi	0.045
6.	Honey	0.001
7.	Fish	0.223
8.	Beef meat	0.000
9.	Yoghurt	0.003
10.	Nono	0.348
11.	Orange	0.001
12.	Yakuwa	0.048
13.	Water	0.006
14.	Yam	0.005
15.	Kunu	0.000
16.	Fura	0.032
17.	Spinach	0.024
18.	Cabbage	0.015
19.	Potatoes	0.012

Mean = 0.05611 ± 0.0209

P = 0.4052

Table 3. Calcium levels (mg/l) for liquid and mg/kg for solids of the different food samples from Jankasa settlement of Kaduna state.

S/N	SAMPLE	CONC. OF CALCIUM
1.	White Kaura	0.018
2.	Yellow Kaura	0.008
3.	Red Kaura	0.012
4.	Millet	0.021
5.	Tuwo	0.042
6.	Acha	0.008
7.	Water	0.012
8.	Beans	0.051
9.	Patte	0.045
10.	Nono	0.871
11.	Groundnut	0.030
12.	Fura	0.053
13.	Maize	0.007
14.	Yam	0.004
15.	Sweet Potatoes	0.021
16.	Kuli Kuli	0.016
17.	Bread	0.002
18.	Onion	0.032
19.	Lettuce	0.054

Mean = 0.0687 ± 0.0447

P = 0.8119

Table 4. Calcium levels (mg/l) for liquid and mg/kg for solids of the different food samples from Kaso settlement in Kaduna state

S/N	SAMPLE	CONC. OF CALCIUM
1.	White Kaura	0.003
2.	Red Kaura	0.002
3.	White Beans	0.058
4.	Black Beans	0.047
5.	Yam	0.013
6.	Water	0.013
7.	Gurjiya	0.021
8.	Sweet potatoes	0.009
9.	Tuwo	0.068
10.	Tomatoes	0.006
11.	Dadawa	0.022
12.	Patte	0.189
13.	Rice	0.010
14.	Soya Beans	1.300
15.	Kindirmo	0.623
16.	Gauta	0.015
17.	Sesame Seed	0.245
18.	Millet	0.010
19.	Bread	0.002
20.	Eggs	0.002
21.	Eguisi	0.014

Mean = 0.1272 ± 0.0662

P = 0.3720

Table 5. Phosphorus levels (mg/l) for liquid and mg/kg for solids of the different food samples from Gonin-Gora settlement of Kaduna state.

S/N	SAMPLE	CONC. OF PHOSPHORUS
1.	Tuwo	0.310
2.	Gari	0.034
3.	Kuka	0.184
4.	Okro	1.064
5.	Karkashi	0.451
6.	Honey	0.036
7.	Fish	5.426
8.	Beef meat	1.097
9.	Yoghurt	0.036
10.	Orange	0.007
11.	Nono	0.068
12.	Orange	0.080
13.	Yakuwa	0.067
14.	Water	0.053
15.	Yam	0.080
16.	Kunu	0.098
17.	Fura	0.881
18.	Spinach	0.056
19.	Cabbage	0.193

Mean = 0.5382 ± 0.2833

P = 0.0976

Table 6. Phosphorus levels (mg/l) for liquid and mg/kg for solids of the different food samples from Jankasa Settlement of Kaduna state.

S/N	SAMPLE	CONC. OF PHOSPHORUS
1.	White Kaura	0.172
2.	Yellow Kaura	0.550
3.	Red Kaura	0.398
4.	Millet	0.423
5.	Tuwo	0.300
6.	Acha	0.400
7.	Water	0.016
8.	Beans	0.333
9.	Patte	0.037
10.	Nono	0.026
11.	Groundnut	0.262
12.	Fura	0.110
13.	Maize	0.360
14.	Yam	0.000
15.	Sweet Potatoes	0.062
16.	Kuli Kuli	0.348
17.	Bread	0.077
18.	Onion	0.489
19.	Lettuce .	0.873

Mean = 0.2617 ± 0.0541

P = 0.0224

Table 7. Phosphorus levels (mg/l) for liquid and mg/kg for solids of the different food samples in Kaso settlement of Kaduna state.

S/N	SAMPLE	CONC. OF PHOSPHORUS
1.	White Kaura	0.360
2.	Red Kaura	0.470
3.	White Beans	0.260
4.	Black Beans	0.228
5.	Yam	0.073
6.	Water	0.009
7.	Gurjiya	0.234
8.	Sweet Potatoes	0.065
9.	Tuwo	0.320
10.	Tomatoes	0.765
11.	Dadawa	1.264
12.	Patte	0.390
13.	Rice	0.173
14.	Soya Beans	0.338
15.	Kindirmo	0.982
16.	Gauta	0.376
17.	Sesame Seed	0.338
18.	Millet	0.430
19.	Bread	0.088
20.	Eggs	0.400
21.	Eguisi	1.484

Mean = 0.4308 ± 0.0844

P = 0.0081

Calcium concentration in Food in Gonin Gora Area

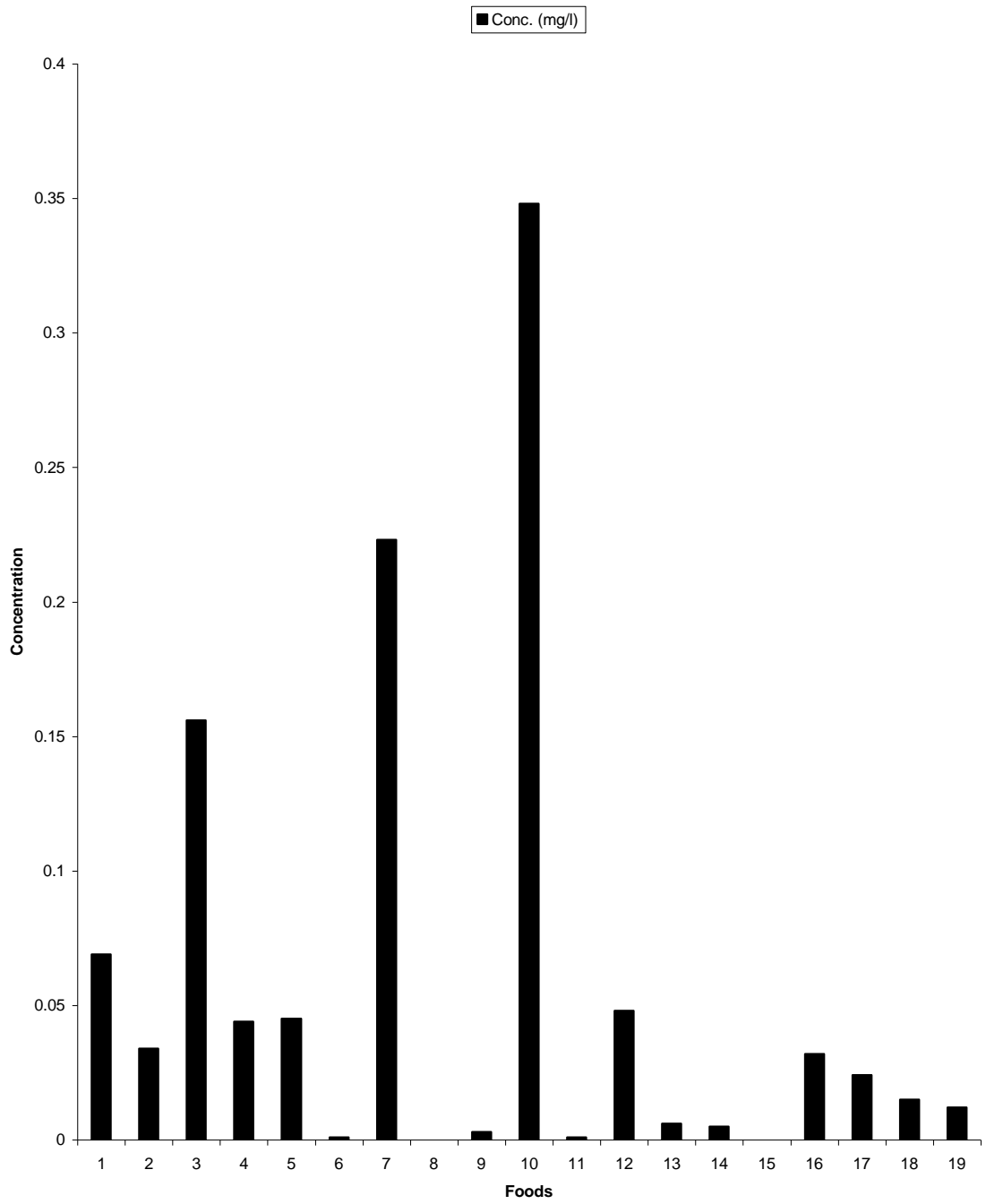


Figure 2

Calcium concentration in Foods in Jankasa Area

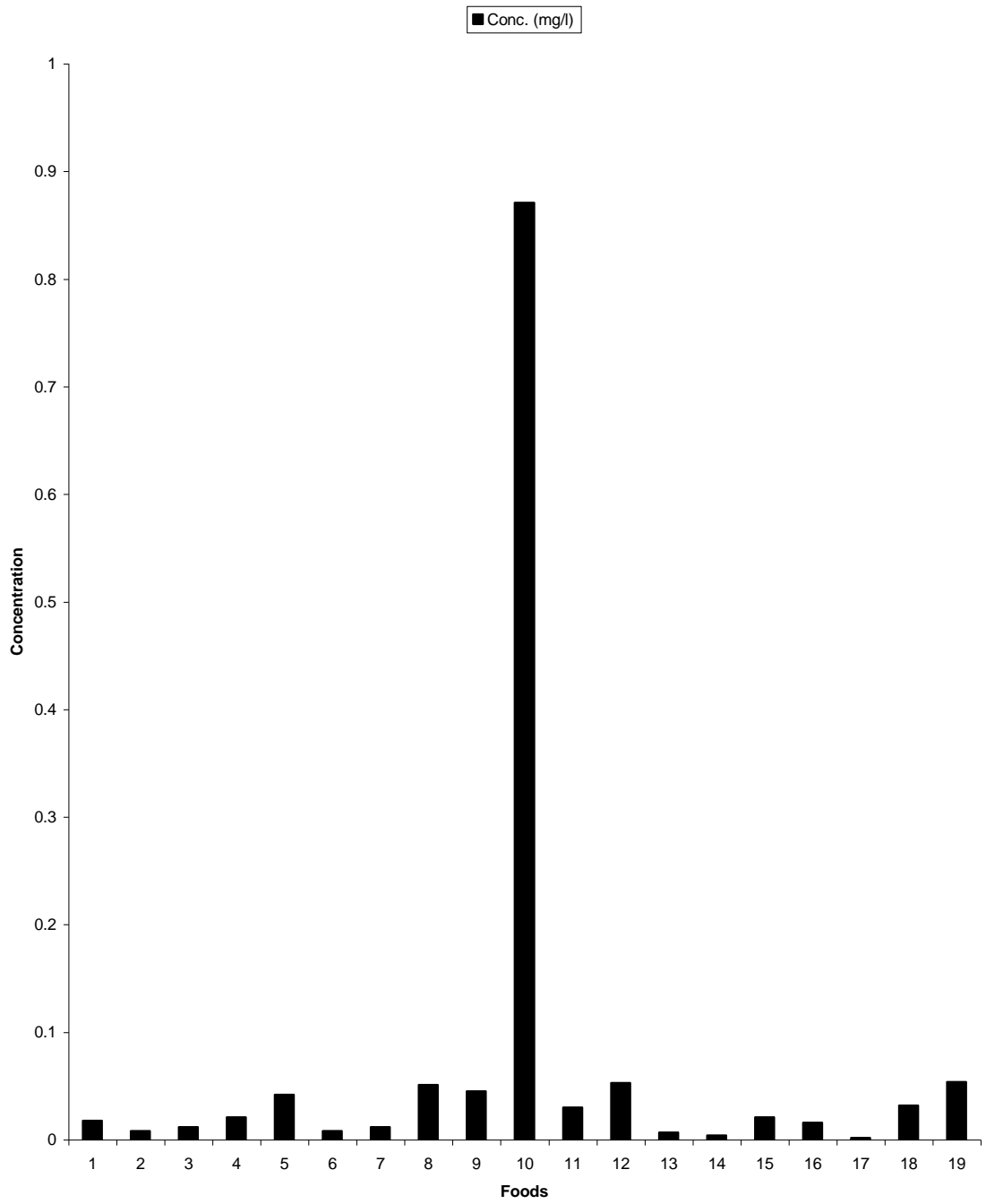


Figure 3

Calcium concentration in Foods in Kaso Area

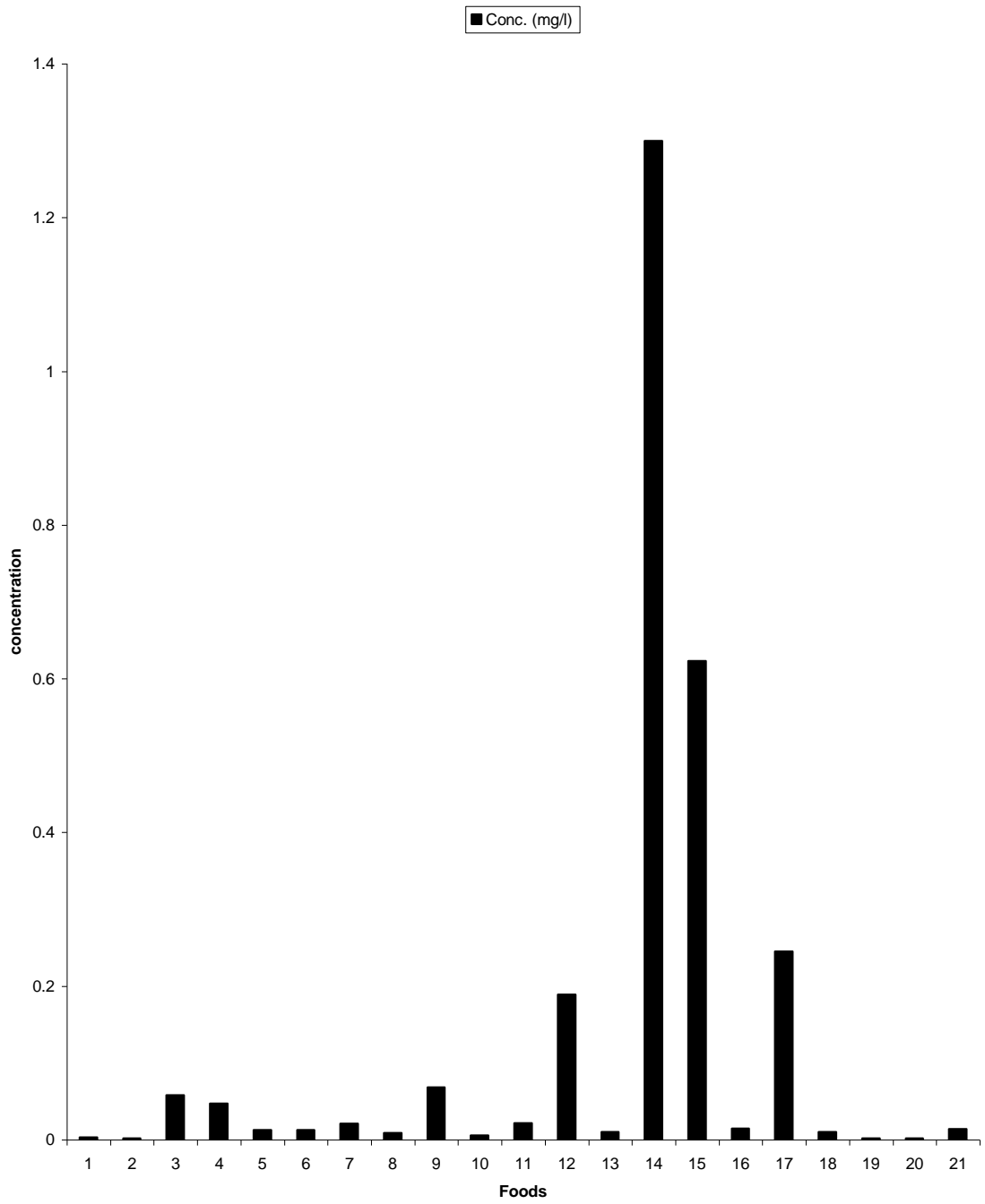


Figure 3

Phosphorus concentration in Foods in Gonin Gora Area

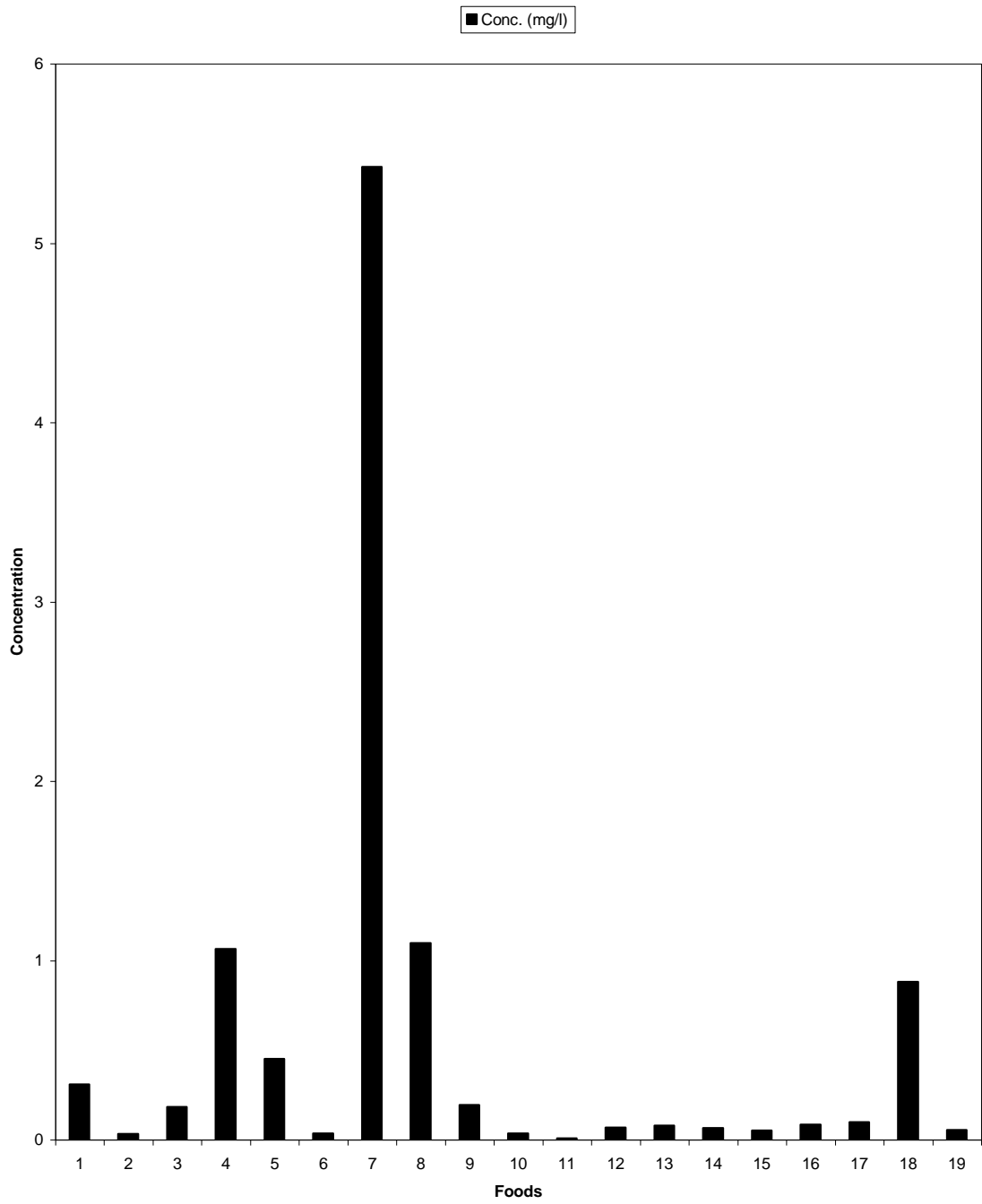


Figure 5

Phosphorus concentration in Foods in Jankasa Area

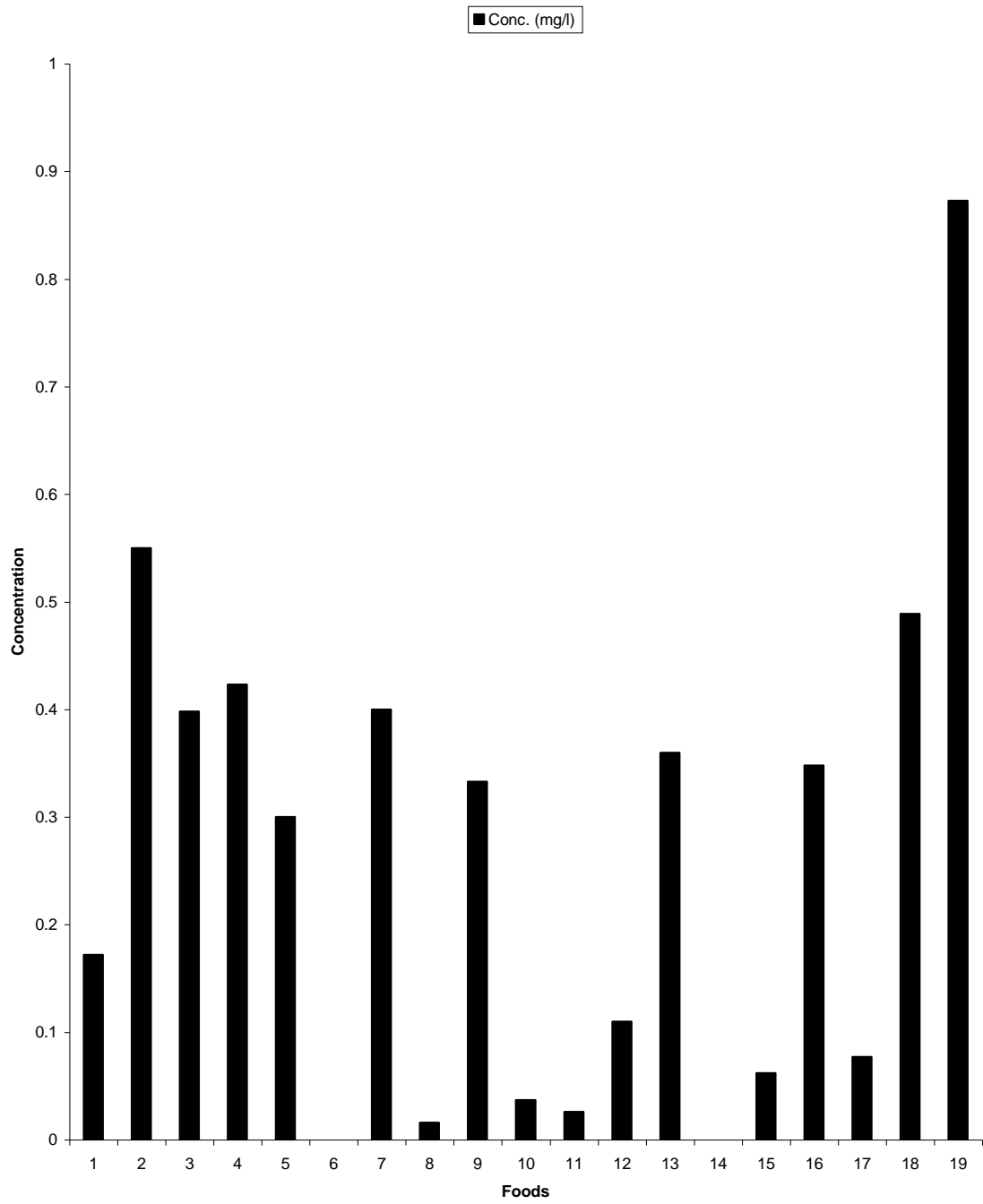


Figure 6

Phosphorus concentraion in Foods in Kaso Area

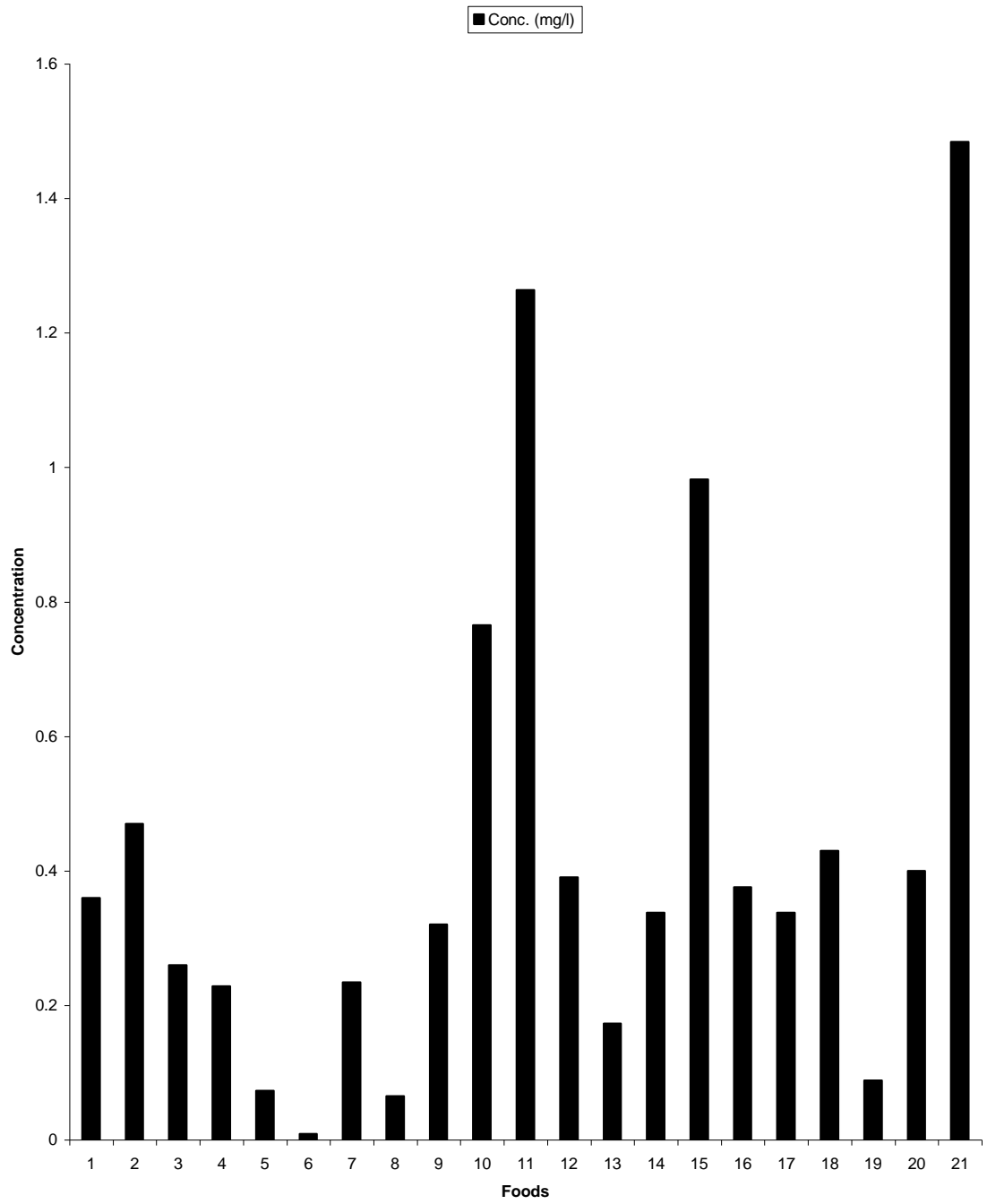


Figure 7

CHAPTER FIVE

5.0 DISCUSSIONS

5.1 Calcium Concentrations in Water and Food.

The levels of Calcium in water and food analyzed followed a similar pattern in all the study areas.

The mean value of calcium concentration in Gonin Gora the area was 0.0561 ± 0.0209 (S.M.E) mg/l or mg/kg which is lower than allowable limit of 0.130 mg/l or mg/kg (USDA 2007). This low intake of calcium could contribute to the development of the disease.

From the mean concentration, Jankasa showed a value of 0.0687 ± 0.0447 (S.M.E) which is also less than the allowable limit indicating a high risk factor for the disease in the area.

From the results of Analysis of Variance (ANOVA) calcium level in the study areas were found to be insignificantly low but below allowable limit of 0.130 mg/l or mg/kg (USDA 2007). The rickets problem is however minimal in Gonin Gora compared with Jankasa and Kaso. Gonin Gora has higher level of calcium in most of their food due to closeness of the village to Kaduna town which improved the standard of the diet in the community.

The mean value of calcium level in Kaso was 0.1272 ± 0.0662 (S.M.E) which is higher than that of Gonin Gora and Jankasa since soya beans with high calcium concentration (1.300 mg/l or mg/kg) formed part of their staple food thus affecting the mean value of calcium levels. Despite the high mean value of calcium levels the area was most affected with the

disease than other communities. Almost each family has a victim of the disease.

In Jankasa the result of ANOVA showed the mean value of 0.0687 ± 0.0447 which is also below the allowable limit of 0.130 mg/l or mg/kg (USDA 2007). The health condition in Jankasa area is better than Kaso but most of the houses also have at least a victim of the rickets disease.

5.2 Phosphorus Concentration in Water and Food

The levels of phosphorus in the foods were found to be generally higher than the levels of calcium (Tables 5, 6 and 7) and (Figures 5, 6 and 7) for Gonin Gora, Kaso and Jankasa respectively. The levels of phosphorus are insignificantly low in Gonin Gora $P = 0.0976$, Kaso $P = 0.0081$ but significantly low in Jankasa $P = 0.0224$.

The functional consequences of this high intake of phosphorus in the presence of low calcium remains a topic of controversy (Sax, 2001). Wyshak (2000) reported that high phosphorus intake contributed to hypocalcaemia and fractures in children. The high phosphorus intake in the presence of low calcium could also be responsible for the problem.

CHAPTER SIX

6.0 SUMMARY, CONCLUSION AND RECOMMENDATIONS

6.1 SUMMARY

Atomic Absorption Spectrophotometer was used to determine the levels of calcium and phosphorus in staple food in Gonin Gora , Jankasa and Kaso rickets prevalent areas in Kaduna State. The results showed high levels of phosphorus and low levels of calcium which could be the major cause of the disease in these areas. Further studies have been recommended to get at other causes of problem.

6.2 CONCLUSION

Most staple food in the three study settlements (Gonin Gora, Kaso and Jankasa) are low in calcium but high in phosphorus. The highest mean concentration of calcium was found to be 0.1272 ± 0.0662 (S.E.M) in Kaso.

The low calcium intake among infants and children has been to be attributed to the development of Rickets (Koof et al, 1972; and Legius et al, 1989). Rickets among rural children has been reported to be attributed to low dietary calcium intakes (Pettifor, 2004). The levels of phosphorus were 0.5382 ± 0.2833 S.E.M and 0.4308 ± 0.0844 S.M.E above allowable limit of 0.400 mg/l or mg/kg (USDA 2007) in Gonin Gora and Kaso respectively. But found to be 0.2617 ± 0.0541 S.M.E in Jankasa below allowable limit.

The low levels of calcium in the foods and/or the low calcium intake with high phosphorus intake could be the major cause of the disease in these settlements especially during the period of the children growth.

6.3 Recommendations

1. The community based programme on nutrition and diet capacity building should be adopted in the communities/settlements.
2. Farmers in these settlements should be encouraged to cultivate soya beans for their consumption.
3. Consumption of calcium rich foods i.e. soya beans, nono, kindirmo should be encouraged in these areas.
4. Calcium oral supplements should be administered to victims for a period of time to see if their condition could be reversed.
5. Further studies on the following can be conducted in the settlements:-

- Comparative studies on calcium and phosphorus serum levels of children who are victims and non victims should be conducted.
- The blood level vitamins of the children should be determine *IN VIVO*
- There should be a mass enlightenment campaign in the communities because most men believe their wives are responsible for this problem.
- Determination of some vitamin levels in these children would be necessary.

All these studies would help to discover the root of the problem.

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Appendix 1

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**ANOVA FOR
JANKASA AREA**

Anova: Two-Factor Without
Replication

<i>SUMMARY</i>	<i>Count</i>	<i>Sum</i>	<i>Average</i>	<i>Variance</i>
Row 1	2	0.19	0.095	0.011858
Row 2	2	0.558	0.279	0.146882
Row 3	2	0.41	0.205	0.074498
Row 4	2	0.444	0.222	0.080802
Row 5	2	0.342	0.171	0.033282
Row 6	2	0.008	0.004	0.000032
Row 7	2	0.412	0.206	0.075272
Row 8	2	0.067	0.0335	0.0006125
Row 9	2	0.378	0.189	0.041472
Row 10	2	0.908	0.454	0.347778
Row 11	2	0.056	0.028	0.000008
Row 12	2	0.163	0.0815	0.0016245
Row 13	2	0.367	0.1835	0.0623045
Row 14	2	0.004	0.002	0.000008
Row 15	2	0.083	0.0415	0.0008405
Row 16	2	0.364	0.182	0.055112
Row 17	2	0.079	0.0395	0.0028125
Row 18	2	0.521	0.2605	0.1044245
Row 19	2	0.927	0.4635	0.3353805
Calcium Conc (mg/l)	19	1.307	0.068789474	0.038039953
Phosphorus Conc (mg/l)	19	4.974	0.261789474	0.055794064

ANOVA

<i>Source of Variation</i>	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>P-value</i>	<i>F crit</i>
Calcium Conc (mg/l)	0.6678743	18	0.037104129	0.654049027	0.811917275	2.217197134
Phosphorus Conc (mg/l)	0.3538655	1	0.3538655	6.237725949	0.02242218	4.413873405
Error	1.021138	18	0.056729889			
Total	2.0428778	37				

Appendix 2

ANOVA FOR KASO AREA

Anova: Two-Factor Without Replication

<i>SUMMARY</i>	<i>Count</i>	<i>Sum</i>	<i>Average</i>	<i>Variance</i>
Row 1	2	0.363	0.1815	0.0637245
Row 2	2	0.472	0.236	0.109512
Row 3	2	0.318	0.159	0.020402
Row 4	2	0.275	0.1375	0.0163805
Row 5	2	0.086	0.043	0.0018
Row 6	2	0.022	0.011	0.000008
Row 7	2	0.255	0.1275	0.0226845
Row 8	2	0.074	0.037	0.001568
Row 9	2	0.388	0.194	0.031752
Row 10	2	0.771	0.3855	0.2880405
Row 11	2	1.286	0.643	0.771282
Row 12	2	0.579	0.2895	0.0202005
Row 13	2	0.183	0.0915	0.0132845
Row 14	2	1.638	0.819	0.462722
Row 15	2	1.605	0.8025	0.0644405
Row 16	2	0.391	0.1955	0.0651605
Row 17	2	0.583	0.2915	0.0043245
Row 18	2	0.44	0.22	0.0882
Row 19	2	0.09	0.045	0.003698
Row 20	2	0.402	0.201	0.079202
Row 21	2	1.498	0.749	1.08045
Calcium Conc (mg/l)	21	2.672	0.127238095	0.09230569
Phosphorus Conc (mg/l)	21	9.047	0.430809524	0.149668962

ANOVA

<i>Source of Variation</i>	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>P-value</i>
Calcium Conc (mg/l)	2.598290476	20	0.129914524	1.159328706	0.372086826
Phosphorus Conc (mg/l)	0.967633929	1	0.967633929	8.634952868	0.008123175
Error	2.241202571	20	0.112060129		
Total	5.807126976	41			

Appendix 3

ANOVA FOR GONIN GORA AREA

Anova: Two-Factor Without Replication

<i>SUMMARY</i>	<i>Count</i>	<i>Sum</i>	<i>Average</i>	<i>Variance</i>
Row 1	2	0.379	0.1895	0.0290405
Row 2	2	0.068	0.034	0
Row 3	2	0.34	0.17	0.000392
Row 4	2	1.108	0.554	0.5202
Row 5	2	0.496	0.248	0.082418
Row 6	2	0.037	0.0185	0.0006125
Row 7	2	5.649	2.8245	13.5356045
Row 8	2	1.097	0.5485	0.6017045
Row 9	2	0.196	0.098	0.01805
Row 10	2	0.384	0.192	0.048672
Row 11	2	0.008	0.004	0.000018
Row 12	2	0.116	0.058	0.0002
Row 13	2	0.086	0.043	0.002738
Row 14	2	0.072	0.036	0.001922
Row 15	2	0.053	0.0265	0.0014045
Row 16	2	0.117	0.0585	0.0014045
Row 17	2	0.122	0.061	0.002738
Row 18	2	0.896	0.448	0.374978
Row 19	2	0.068	0.034	0.000968
Calcium Conc (mg/l)	19	1.066	0.056105263	0.008310211
Phosphorus Conc (mg/l)	19	10.226	0.538210526	1.525671731

ANOVA

<i>Source of Variation</i>	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>P-value</i>
Calcium Conc (mg/l)	14.59665205	18	0.810925114	1.121523348	0.405215117
Phosphorus Conc (mg/l)	2.208042105	1	2.208042105	3.053760121	0.097588493
Error	13.01502289	18	0.723056827		
Total	29.81971705	37			

