Estimation of Selenium and Chromium Content of Selected Foods.

BY

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I INTRODUCTION

About 4 per cent of the body weight is made up of the elements usually designated as minerals. These may be defined as those elements which remain largely as ash when plant or animal tissues are burned (Robinson, 1972). Approximately 50 mineral elements are found in the body, some are present in relatively large amounts and are called macronutrients. Many of the elements are present in such minute amounts that they are generally referred to as trace elements or micronutrients (Lewis, 1976).

In addition to the major mineral elements human body requires minute quantities of certain elements known as trace elements. These are as follows: Copper, Manganese, Molybdenum, Zinc, selenium, chromium, Fluoride, Iodine, Nickel, Tin, Silicon and Vanadium. The exact role these elements play in the body is often as an integral part of vitamins, enzymes or hormones. Very minute amounts of these elements are required in the diet and any normal diet will contain sufficient for the body's need (Fox and Gameron, 1984).

Selenium was found to be an essential trace element in 1957 when it was discovered that animals deficient in selenium had increased susceptibility to liver necrosis (Harvey, et al., 1985). Selenium deficiency was found to be

associated with glutathione peroxidase deficiency (Cohen and Chovaniec, 1985). The effect of selenium deficiency on the responses to Candida albicans infection were examined in mice (Boyne and Athur, 1986). The deficiencies of selenium and vitamin E enhanced the acute toxicity of drugs which are activated by oxygen (Dell and Oison, 1984).

Selenium deficiency can be prevented by feeding inorganic selenium compounds such as sodium selenite or organic selenium compounds such as selenomethionine. Both selenium and vitamin E are known to protect cellular constituents against lipid peroxidation, tissue damage and have been known to inhibit mutagenesis (Shamberger, 1979).

It was found that while 5ppm or more of selenium in the diet depressed the growth rate of chicks, 2ppm slightly stimulated growth. Schwarz and Foltz (1957) announced that selenium was an integral part of factor 3 and that selenium salt would themselves protect rat against the dietary necrotic liver degeneration prevented by factor 3' (Zingaro and Cooper, 1974). Selenium levels in blood varied inversely with the serum total bilirubin and directly with albumin. Because selenium is essential for glutathione peroxidase activity, a deficiency may result in increased tissue lipid oxidations. Selenium deficiency in alcoholic may cause enhanced hepatic damage. Selenium deficiency has been also identified with congestive cardiomyopathy, anemia and

pancreatic fibrosis (Dennis, 1984). Selenium acts a chemopreventive agent against mammary carcinogenesis (Thompson, 1984). as antioxidant and has the ability to cure illnesses such as cancer and heart diseases (Wenck and Baren, 1980).

'Keshan disease' is the best known selenium deficiency disease of China. "Kaschin-Beck disease" affecting mainly the joints of both animals and humans, has been recognised in China and is also preventive with dietary selenium supplements (Xianmao et al., 1985). Foods may contain excess or deficiency of selenium, thus leading to signs of toxicity or of deficiency disease. "Alkali disease" in cattle occurs when grains and forages containing high levels of selenium are ingested (Robinson, 1972). Selenium is stimulating and probably essential for the growth of certain indicator plants such as Astragallus racemosus (Zingaro and Copper, 1974).

The selenium content of American and Canadian wheat and rye was very much higher than that in grain from other sources. The best sources of selenium are foods of high protein content such as meats, South Dakota cheeses, breads, cereals, soya beans preparation, poultry, egg and sea foods (Uchida and Shimoishi, 1984 and Pennigton et al., 1986). Meat dairy products and wheat grain are the best dietary

sources of selenium (Seymour and Halper, 1979). The average daily intake of selenium is 60-150mcg (µg) (Lindeman, 1982).

The trivalent chromium is an essential trace element for the mammal which must be provided by the diet. It is also known that dietary chromium is mostly in an inactive form, which must be converted to active form, known as the "glucose tolerence factor" before exerting any physiological role which is thought to be as a cofactor for insulin action (Gardiner, 1984). It may be also possible that chromium has a role in lipid metabolism. Invitro, chromium (Cr^{3}III) has been found to bind to DNA and induce abnormal synthesis of RNA (Bunker, 1984).

The chromium deficiency state in the mammals has been associated with an insufficient dietary intake of chromium, a weak gastrointestinal absorption, an excessive urinary excretion, aging, diabetes mellitus, pregnancy and protein calorie malnutrition (Gurson and Saner, 1982). Its deficiency in animals leads to an impairment of glucose tolerence and severe restriction leads to a syndrome indistinguishable from diabetes mellitus (Arnold, 1978). It develops an impaired growth and an increased incidence of atheromatous lesions in aorta (Halpern, 1979). Chromium in the oxidation state of '+6' has been said to be carcinogenic (Draper, 1979).

Meat, cheese, whole grains and condiments are good sources of available chromium. It appears to be less available from the leafy vegetables and is very low from polished rice, refined flours and sugars (Anderson and Dibble, 1976). Food and Nutrition board recommended 50mcg to 200mcg as a safe and adequate intake of chromium by adults and adolescents (1985). The amount of chromium in plant foods depends on the chromium in the soil and processing and refinment reduces the chromium content of foods (Briggs and Calloway, 1973).

Since selenium and chromium are found to be important minerals in nutrition, it is necessary to know the amounts present in the foods consumed by man. However, very little information is available on the same in India and abroad. Hence in this study an attempt has been made to evaluate selected meat, milk, vegetables, sea foods and eggs for their selenium and chromium contents.

Review of Literature

II REVIEW OF LITERATURE

The review of literature pertaining to the study "The selenium and chromium content of selected foods" is discussed under the following headings:

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A. Selenium

1. History of selenium

Arnold de Villanova was the first to observe and describe the element which came to be known as selenium. But he called it as sulphur rubeum. Later in 1817, Berzelius discovered selenium as a red deposit in the flues and chambers of sulphuric acid plant. The name selenium has been discovered from the Greek term 'selene' that is, the moon (Anil Kumar, 1983). This element was feared as a poison until its essentiality was demonstrated in 1956. Although selenium was used in cancer therapy during World War I, well documented evidence of its anticarcinogenic properties was reported by Clayon and Baumann only in 1949 (Draper, 1979).

2. Occurrence of selenium

The occurrence of selenium in soils is a topic of considerable interest, particularly in view of its toxic effects on cattle through plants which can accumulate the elements from seleniferous soils (Kakin and Davidson, 1967). Selenium in soil may occur as selenites, selenates, elemental selenium and as selenium in association with pyrite and other minerals. According to Sindeova (1964) soils have a selenium content of around 0.01ppm. The soils with the greater selenium content occur in low-lying areas which are poorly drained and in high organic residues. The amount of selenium in soil and thereby in food varies greatly between countries. In Sweden the soil has low selenium content (Akesson and Okerson, 1985). In the case of surface waters much higher concentrations of selenium have been found, notably in seleniferous region, the maxium value noted being 400ng/litre (Scott and Voegeii, 1961).

According to Rosenfold and Beath (1984) there are 24 species and varieties of the plant Astragalus which grow in seleniferous soils and for which selenium is an essential nutrient. Such plants have been designated as selenium indicator plants. Grain and forage crops from seleniferous region have shown a high selenium content; notably wheat (115ppm), barely (137ppm), corn (40ppm) and grass (280ppm).

In a study conducted in Belgium on the selenium content of the beer of different brands the selenium levels ranged from 0.2 to 15.2ng/ml. The selenium content of the Finnish dried milk is 34 ± 6 µg/kg (Nurrtame and Koivistoinen, 1983). The selenium content of skim milk in Finland is around 43µg/kg. The selenium value is highest in colostrum (median 30ng/ml) of lactating mothers. Subsequently selenium content declines significantly during the first month of lactation then comes to a plateau (Median 17 to 18ng/ml).

The selenium contentration in the wool of sheep is 125-570 ng/g (Kumpulainen and Railtila; 1984). In a study conducted in Spain, 27 samples of vegetable origin were analysed and the amount of selenium found, ranged between undetectable to 7.6×10^{-2} µg/g. The value of selenium in meat is between 0.01 and 0.85 µg/g (Domingue and Motario, 1983). In Germany the median content of selenium in food was 27ng/g (Leven Smiddelen, 1982). Meat and fish are the main sources of selenium and they contain less than 1.0µg/g. Under normal dietary conditions hen's egg contains a total of 10-12 µg selenium, most of which is present in the yolk. The total amount and the proportion present in the yolk and white are markedly and rapidly influenced by the selenium status of the hen's diet and by chemical form or forms in which dietary selenium is supplied (Lavender and Wood, 1977). At highly toxic selenium intakes extremely high selenium levels can occur in hen's eggs particularly in the white

(Levander et al., 1975). Hadjimarkos and Shearer (1973) have found 2.0µg selenium per dl in 39 samples of cow's milk and Millar and Sheppard (1973) found 1.3 µg selenium/dl) in human milk and 0.5µg selenium/dl in bovine milk. Thus, this element must be considered a normal component of human milk (Bourne and Grenada, 1981).

A study conducted in central South Dokato Showed that the best sources of selenium were foods of high protein content such as meat, South Dokata Cheeses, semolina products, breads, break fast cereals, oats, poultry, eggs and some other sea foods (Maynard, 1973). The study on milk samples gave an overall average and standard deviation of 6±12µg/lit. The selenium level was found to be 0.009 ppm in Torula Yeasts. Brewar yeast contained 0.1-1.0ppm selenium (Ewan, 1968). Tissues from lambs contain 0.01 to 0.05 ppm selenium. Heart and skeletal muscle of dystrophic animals contain about 0.02 ppm of selenium. In sea foods the selenium levels are between 0.11 and 0.97 µg/g. The normal serum selenium concentration varies from 12 to 88 µg/lit (Dutta and Miller, 1983).

3. Biological role of selenium

a. Selenium and vitamin E

Selenium and Vitamin E deficiency in animal causes hepatic necrosis, degeneration, necrosis of cardiac,liver and skeletal muscles, vascular lesions, edema, yellow discoloration of body fat and anemia (Amemiya, 1985). Very few

experimental studies of selenium deficiency alone have been reported. Selenium deficiency causes cataracts in the second generation in rats and selenium toxicity also causes cataracts in rats during suckling period. Vitamin E deficiency induces increased lipofusion granules in the retinal pigment epithelium (Robinson and Kuwabara, 1979). Selenium is an essential trace element in the eye and the iris contains the highest concentration followed by retina and lens (Shearer et al., 1980). Vitamin E and Selenium are micronutrients that play an important role in preventing lipid peroxidation (Stone et al., 1986).

Selenium reduces or prevents the effect of vitamin E deficiency in animals particularly in liver necrosis (Krehl, 1970). Selenium has been reported to be a growth factor for chicks and prevents dietary liver necrosis and muscle degeneration in animals deficient in vitamin E (Halsted, 1981). Majaj et al. (1982) have demonstrated the activity of vitamin E in stimulating reticulocytosis in malnourished infants with macrocytic anemia.

b. Relationship of selenium to proteins

The high proportion of selenium in the common plant and in animal tissues is closely associated with protein. The selenium remains with the protein during precipitation with trichloro acetic acid or dialysis, under general

conditions but much of it is sufficiently labile and is removed by more drastic procedures especially under alkaline conditions.

Two generalisations can be made regarding the chemical form of selenium in proteins. First, the covalently bound selenium is linked to either carbon or sulphur. Secondly the presence of selenium in a protein is always related to the presence of sulphur. The selenium atom is either incorporated in place of sulphur atom in a sulphur amino acid or is attached to the sulphur atoms of cysteine residues (Zingaro and Cooper, 1974).

Because of the well known interrelationships between protein and selenium, the effectiveness of selenium administration along with protein in improving anemia is associated with kwashiorkor (Lindeman, 1982).

c. Effects of other trace elements of selenium utilization

The possibe antagonistic effects of different dietary concentrations of copper, cadmium, iron, molybdenum and manganese on selenium utilisation by the rat were studied by the measurement of the absorption and organ distribution of dietary selenium as (Se^{75}) selenite and by effects on organ glutathione peroxidase (GSH-Px) activity. Although a high concentration of copper (200mg/kg) in the diet did not alter the percentage absorption and total body retention of doses of $^{75}Se\,O_3^{2-}$ by rats, after such treatment tissue

^{75}Se distribution was changed and the total selenium was low in some tissues. After copper treatment GSH-Px activity of liver,testes, kidney and whole blood was also lower.

Several studies with rats and chick show that lower concentration of inorganic elements may modify selenium metabolism. Thus competition between $SeO4^{2-}$ and MoO_4^{2-} for a common intestinal transport mechanism have been demonstrated by using sections of rat and chick small intestine. The possibility that dietarymolybdenum could also influence the metabolic fat of selenium is suggested from many similarities in the chemical properties of Mo/se and Mo/sulphur complexes. Several studies with rats illustrate both the protective effect of selenium against cadmium toxicity and deranged metabolism of selenium following cadmium administration.

Iron deficiency anemia has been reported to decrease whole blood glutathione peroxidase activity in rats and in pigs induce signs of selenium and vitamin E deficiency.

A copper selenium interaction has been demonstrated in chicks and rats in which copper was used to counteract selenium toxicity. The tolerance of high doses of copper has also been shown to be reduced when rats become selenium deficient, but whether this reflects a direct interference with selenium metabolism is not clear.

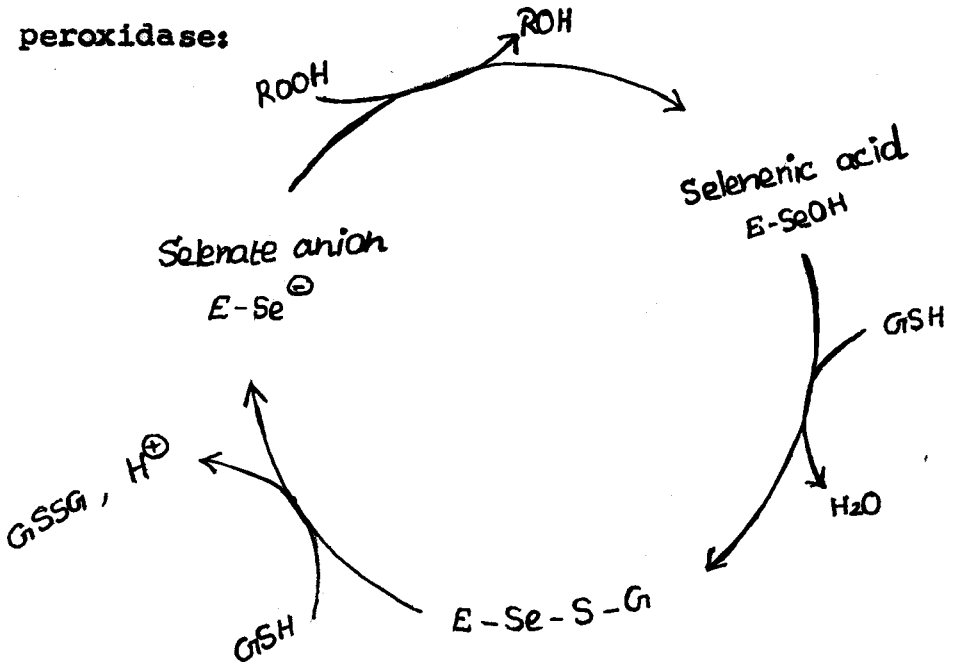
Decreased dietary manganese offered to pigs and increased manganese offered to horses have both been reported to decrease body retention of selenium suggesting interaction between the elements (Rahim et al., 1986).

d. Selenium and glutathione peroxidase activity

Selenium is a constituent of glutathione peroxidase, the enzyme in red blood cells which prevents their oxidative haemolysis by hydrogen peroxide (Diplock, 1974). Selenium acts an an antioxidant (Oison et al., 1984).

Atherosclerosis is a complex multifactorial disorder in which environmental, nutritional, metabolic and genetic factors may interact in the development of the disease. The damaging process of lipid peroxidation may be an important factor in the etiology of atherosclerosis. Selenium plays an important role in preventing lipid peroxidation. Selenium is an essential prosthetic group for glutathione peroxidase which reduces lipid peroxides to the corresponding lipid alcohols. Rats deficient in dietary selenium develop low glutathione peroxidase level in plasma and red blood cells and in most other cells (Rngdal et al., 1985; Nicholas, 1986).

The following figure presents a hypothetical mechanism for the hydroperoxide reduction catalysed by Glutathione peroxidase:



where GSH is reduced glutathione and ROOH is any of a wide variety of hydroperoxides (Robert Olson, 1984).

Selenium deficiency impairs the ability of neutrophils to kill ingested cells of the yeast Candida albicans. These defects in function have been associated with changes in the enzymic and structural components of the selenium deficient neutrophil. Impaired efficiency of scavenging of hydrogen peroxide by decreased glutathione peroxidase activity in the cytoplasm of stimulated neutrophils is thought to allow peroxides diffusing from the phagosome to inhibit their ability to oxidize glucose via the hexose monophosphate

shunt. Selenium deficiency will also impair other immune functions in the animal such as immunoglobulin formation and lymphocyte proliferative response to mitogens (Boyne and Authur, 1986). Aurothioglucose and penicillamine competitively inhibit Se-GSH-Px in inverse proportion to the concentration of hydrogen peroxide and reduced glutathione respectively (Mercuro and Combs, 1985).

In 1976, a non-selenium dependent GSH-Px activity was found in rat liver which did not decrease in selenium deficiency and was subsequently found to be one or more glutathione-s-transferases. This activity was shown to increase in selenium deficient rat livers, in other rat tissues and also in mouse liver, after prolonged deficiency. The non selenium dependent activity differs in that it cannot use hydrogen peroxide but only organic hydroperoxidases (example: tertiary butyl hydroperoxide) and thus it should be possible to distinguish it from the selenium dependent GSH-Px in the coupled assay (Thomson et al., 1985).

e. Selenium and plant metabolism

The selenium content of a plant is primarily determined by the species and whether it is grown in seleniferous or non-seleniferous soil. Seleniferous soils are defined by the vegetation they support rather than by their selenium content. Another important factor that affects the selenium content

of a given species is the sulphur/selenium nutrition. The selenium content increases with the level of selenium available and decreases with the sulphur supply. For example plants can tolerate much higher concentrations of selenium in the presence of high contentration of sulphur (Robb and Pierpoint, 1983).

f. Selenium in red blood cells and serum

Decreases in red blood cell selenium concentration were found in normal subjects older than 60 without concurrent significant changes in serum selenium, although differential results showed that decreased concentration of selenium in both red blood cells and serum occured with alcoholic cirrhosis, malignancies and chronic renal failure. Red blood cell selenium concentrations were also decreased in patients with stable chronic diseases. Decreased serum selenium concentrations were positively correlated with albumin concentration in patients with cirrhosis. There was no correlation between serum selenium and bilirubin concentration in patients with liver disease or between serum selenium and creatinine concentrations in patients with chronic renal failure whose urinary excretion of selenium was far below control levels (Henning, 1984).

4. Recommended dietary intake

Food and Nutrition Board (1980) suggested a range of 50 mcg to 200 mcg per day as adequate and safe intake of selenium by adults. Recommendation for other age groups were derived from the range on the basis of expected food consumption. The main dietary sources of selenium are fish and eggs (29.5% of the total) cereals (28%) and milk (18.7%) (Mutaten, 1985).

The estimated safe and adequate daily dietary intake of selenium in mg: infants - 0.01-0.06; adolescents 0.02-0.20; adults - 0.05-0.20 (Davidson and Passonore, 1983).

Selenium is excreted mainly in the urine and feces but on high intakes, substantial amount may be lost in the breath. The urinary excretion correlates positively with the dietary intake and blood level of selenium. Excretion increases during catabolic stress, such as surgery. Plasma selenium concentration responds to dietary changes more rapidly than erythrocyte selenium and is considered a sensitive index to short term changes in selenium status, whereas red blood cell selenium and the GSH-Px activity are more indicative of long term selenium nutrition (Anderson et al., 1976).

5. Toxic effects of selenium

a. Chronic poisoning

Increased amount of selenium in the diet leads to alkali disease. Chronic selenium toxicity symptoms are: depression, garlic odour of breath, coated tongue, loss of nails and hair, hemolytic anemia and damage to the kidney, liver and spleen. Selenium exhibits significant toxic effects in reproduction, calcification and dental caries. Selenium is teratogenic and causes abnormal development of embryos in experimental and domestic animals. There are reports of teratogenic effects in humans, which include miscarriages and the birth of clubfooted infants to female laboratory technicians exposed to selenium (Luckey and Venugopal, 1977).

b. Acute poisoning

Selenosis is caused by both organic and inorganic forms of selenium. Symptoms of acute selenium toxicity in man are nervousness, fever, vomiting, somnolence, lowering of blood pressure, tetanic and clonic spasms and death due to respiratory failure (Luckey and Venugopal, 1977). In the final stages of acute selenium poisoning the affected animal suffers from salivation, grating of teeth, paralysis and blindness. Death often results from respiratory failure (Swaminathan, 1982).

6. Selenium and cancer

Low selenium content in the soil results in low selenium content in the food and water, which is associated with increased mortality from cancers of the esophagus, stomach and rectum. Smaller increases in mortality are found in cancers of the pharynx, large intestine, bladder and kidney, with no change in mortality from cancers of the lung, lymphnodes, pancreas and prostate and some forms of leukemia. Cancer patients with the lowest blood selenium levels show the greatest likelihood of primary neoplasms, disseminated tumours and decreased survival. Low serum selenium levels could indicate a change prior to cancer initiation or a dietary change associated with cancer or may be caused by therapy. Not all the studies have found differences between pre and post therapy blood selenium levels. Vitamin E and C, selenium and β-carotene all have antioxidant properties (Watson and Leonard, 1986).

a. Selenium and mammary tumours

Schrazer (1979) took specially bred mice that normally develop spontaneous mammary tumours and fed them two ppm of selenium in their drinking water. Instead of the usual cancer incidence of 80 to 100%, only 10% developed tumours. The daughter whose mother developed breast cancer or any individuals subsisting on the typical American diet should

increase their consumption of cereals and sea foods at the expense of meat, fat, sugar and potatoes and Schrauzer recommended any where from 150-250 mcg of supplemental selenium a day (Alfin Slater and Krishchevsky, 1980). The supplement must be of highest quality. Organically bound selenium is best, where the selenium is in a form found normally in food (John Feltman, 1979).

b. Selenium and Colon tumours

Selenium has been shown experimentally to alter the distribution of colonic cancers. In animals not receiving selenium supplementation, right colon tumours were noted to predominate over left colon tumours at a ratio of approximately 2:1. With supplementation of selenium to 8 ppm there was a more than 90% reduction, specifically of right colon tumours and no significant change in the number of left colon tumours (Pence and Budding, 1985).

B. Chromium

1. History of chromium

The metal chromium was discovered by Vanquelin in 1797. It's name is derived from the Greek word "Chroma" meaning colour, because it forms a large number of coloured salts (Mertz and Dekkar, 1971). In 1959 Schwarz made the important discovery that very small amounts of chromium were necessary in the diet for normal metabolism of blood glucose in the rat (Bogert and Briggs, 1973).

2. Occurrence of chromium

Chromium occurs in traces in human and animal tissues. Recent studies have shown that chromium may be a co-factor for insulin. The chromium content of an adult human body is estimated to be 6mg. Most adult tissues contain 0.02-0.4 ppm of chromium on dry basis. The blood contains about 0.009-0.055 ppm (Swaminathan, 1974).

Chromium is present in cereals, legumes, wheat, rye, oats, barley, grain, peas and beans. The estimated values of chromium ranged between 0.064-0.095 µg/kg (Tokeykologiezna, 1983).

The following table (Kirppatrick and Coffin, 1971) gives the amount of chromium present in different foods:

Food type	Chromium content (ppm)
Milk dairy products	0.05
Meat, fish, poultry	0.06
Cereals	0.06
Potatoes	0.04
Leafy vegetables	0.09
Legumes	0.06
Root vegetables	0.09
Garden fruits	0.05
Fruits	0.05
Oils and fats	0.03
Sugars	0.34
Drinks	0.03

Chromium in beers of eight brands ranges from 0.5-5.6 ng/ml (Robberecht and Deelstra, 1984). The chromium content of the refined sugar is low (Sirichakwal and Young, 1985). In a study conducted in New Southwales, Australia, the birds from the industrialised region had no significant heavy metal contamination. But accumulation of chromium, copper and manganese was found in the salt glands (Hulbert and Horning, 1981). The mean serum concentration of normal human subjects is 0.16 ng/ml with a standard deviation of 0.83 ng/ml and range of 0.0382-0.352 ng/ml (Versieck and Hoste, 1978). The total amount of chromium in wine averaged 100mg/litre with low values of 10-50mg and maximum values of 400mg (Shapcott and Hubert, 1979).

3. Biological role of chromium

a. In carbohydrate metabolism

Numerous animal studies have shown that a chromium deficient diet results in an impaired glucose tolerance. Schwarts and Mertz (1981) postulated that this was dueto a deficiency of glucose tolerence factor (GTF), a non-protein organic complex that contains chromium as metal activator. The average American diet contains only 5-115µg chromium per day and this is poorly absorbed. Tissue (Hepatic) chromium concentration declines remarkably with age (10 fold), suggesting that chromium deficiency may be common in the adult population.

Doisg et al. (1980) showed that insulin requiring diabetics absorbed 2-4 times more chromium than did normals. They also excreted more chromium in the urine. Their hypothesis was that diabetics are chromium deficient and develop an adaptive increase in chromium absorption. Interestingly, intravenous glucose or insulin, produces a rapid fall in serum chromium concentration (normal 1-5µg/ml) in normal subjects. High carbohydrate intake increases urinary chromium excretion (normal 3-5µg/day) possibly predisposing individuals to chromium deficiency.

In genetically diabetic mice, GTF, present in high concentration in brewer's yeast reduced abnormal blood glucose concentration to normal. Inorganic trivalent chromium has no effect. Doisy et al. (1982) reviewed their studies and those of three other groups showing that maturity-on-set diabetics, middle aged and elderly subjects with impaired glucose tolerence showed improvement with addition of either 150µg of inorganic trivalent chromium or GTF to their diet. Furthermore, those patients treated with GTF had much lower serum insulin concentration. Their hypothesis was that GTF acts in concert with insulin to aid movement of glucose into the cell and that availability of chromium is the limiting factors in the production of GTF. Finally they also showed that serum triglyceride and cholestrol levels are decreased with GTF (Lindeman, 1982).

Carbohydrate intolerance associated with protein malnutrition in humans is also improved by chromium supplementation. Nicotinic acid-chromium complex potentiates the activity of insulin (Podolskys et al., 1985). GTF extracted from brewer's yeast improves carbohydrate metabolism in genetically diabetic mice when given parenterally, where inorganic chromium administrated orally is ineffective (Robinowitz et al., 1983). Trivalent chromium is essential for carbohydrate metabolism in man. Trivalent chromium is a cofactor in several enzyme systems involved in glucose metabolism and may be necessary for insulin/receptor interaction (Dickerson et al., 1978).

Schraeder (1980) induced severe chromium deficiency in rats and the animals developed a syndrome resembling diabetes mellitus. It was rapidly cured when 5 ppm of chromium III was supplied in drinking water. Chromium III has also been reported to be effective in improving glucose tolerance in some patients suffering from diabetes mellitus, while it was not effective in few others (Swaminathan, 1982).

b. In lipid metabolism

The long-term effects of this suboptimal intake of chromium may be related to the decrease in tissue chromium with age and the increased incidence of diabetes and atherosclerosis observed in developed countries (Richard and Kozlovsky, 1985).

Chromium facilitates the uptake of cholesterol and fatty acids by the liver and the cholesterol content of the blood of rats that have been given chromium supplemented diet over a long period of time is significantly lower than that of their untreated litter mates. There is some evidence that the same effect can be achieved in human beings given chromium acetate in doses of 2mg daily. It seems that chromium also facilitates the utilisation of glycine, serine and methionine (James Crossland, 1980).

Deficiency of chromium increased cholesterol in rats. Supplementation of chromium to human diets decreased cholesterol and increased HDL (Mertz, 1982). Addition of 5ppm of chromium III in drinking water brought the cholesterol level to normal (Harold Draper, 1979).

c. In the development of plants

Chromium as sodium dichromate at 2.5 ppm increased the yields of cabbage and spinach on peat soil in Canada (Gupta, 1983). The chromium level of 100kg/ha completely inhibits the germination of spinach seeds. When chromium (VI) level of 5kg/ha was applied there was no reduction in the number of spinach plants, though the weed growth was significantly reduced. The result suggests that soil applied chromium (VI) may have a herbicidal property (Taiwal and Misra, 1985).

d. Biological uses of chromium

Chromium in trivalent oxidation state is an essential nutrient. However, chromate and dichromate are toxic and must be monitored (Frederic-Betpera and Bruno Jaselskis, 1981). Chromium may act as a protective agent against ischemic coronary heart disease. Chromium supplementation has been shown in several human studies to lower elevated serum cholesterol with magnitude of effects similar to that achieved by strict limitations of dietary fats (Tuman et al., 1974).

4. Nutritional assessment

Although malnourished children, multiparous women and subjects at and beyond middle age appear to be at risk of marginal chromium deficiency, no quantitative assessment of this risk can be made. Several studies indicate that the amount of dietary chromium is marginal under many conditions in Western Countries (Schelenz and Radional, 1977) except perhaps in Sweden.

5. Long-term effect of marginal chromium deficiency in man

Impaired glucose tolerence may precede the development of maturity-onset diabetes, but this is true for only a small proportion of the cases, and diabetes is a disease of many etiologies. Chromium supplementation, although

considered not to be a therapeutic agent in established diabetes, may in some cases prevent or delay the appearance of the disease. This hypothesis remains to be examined in long-term, prospective tials.

More importantly, chromium may be a protective agent against ischemic coronary heart disease. In rats fed diets deficient in chromium and protein, the capacity for the incorporation of certain amino acids in heart muscle was reduced (Swaminathan, 1982). Chromium supplementation has been shown in several human studies to lower elevated serum cholesterol with a magnitude of effects similar to that achieved by strict limitation of dietary fats (Doisy et al., 1976; Liu and Morris, 1978). Elevated serum cholesterol levels are, one of the several risk factors for cardiovascular disease. A second, strong risk factor, gaining increasing attention, consists of elevated fasting insulin levels and elevated insulin responses in glucose tolerance tests (Stout, 1977). Two independent studies, one in Finland and one in Australia, detected significantly increased risk of morbidity and mortality from coronary heart disease, in subjects with elevated insulin levels and responses, independent of glucose tolerance and of other risk factors (Schroeder, et al., 1970; Welborn and Wearm, 1979). Elevation of insulin levels is the first consistent and persistent sign of marginal and moderate chromium deficiency; it can be normalized by chromium supplementation (Newman et al., 1978).

6. Serum and urine levels of chromium

Fasting serum chromium levels were measured in healthy volunteers and in insulin dependent diabetics - Normal subjects - 0.075 ± 0.069 µg/dl; and insulin-dependent diabetics - 0.24 ± 0.17 µg/dl. The overnight fasting and 2 hour post prandial oxidized urinary chromium levels were measured on multiple days in four healthy normal individuals. In normal subjects, fasting non-oxidized chromium was found to be 0.21 ± 0.14 ng chromium per mg of creatinine and fasting oxidised chromium was found to be 0.39 ± 0.30 ng of chromium per mg of creatinine. In insulin-dependent diabetics the values were 0.60 ± 0.52 and 0.85 ± 0.57 ng of chromium per mg of creatinine respectively (Moody and Lindstrom, 1977).

Chromium excretion in urine is dependent not only on the chromium stores but also to some degree on the amount of volume excreted. The amounts of excreted chromium should be expressed on the basis of creatinine excretion, otherwise adjustments for urinary volume have to be made (Shapcott and Hubert, 1979).

7. Recommended dietary intake

According to Borel and Anderson (1984) 50 µg/day of chromium is adequate for daily diet. The majority of the diet samples contained between 10-40mg of chromium only. According to Sunden et al. (1980) the estimated safe and

adequate daily dietary intake of chromium in mg in infants = 0.01-0.06; adolescents = 0.02-0.20; adults = 0.05-0.20.

Chromium supplementation in elderly persons

The effects of measured amounts of brewer's yeast, chromic chloride or placebo were tested in 23 free-living elderly volunteers who were eating nutritious diets and were not at risk for chromium deficiency, as measured by the absence of a clinical response to supplementation. Results showed that age was not a factor leading to chromium deficiency (Henning et al., 1985).

8. Chromium in parental nutrition

Patients under TPN (Total Parental Nutrition) get intravenous solutions, which are composed of purified elemental nutritions. This may be a reason that trace element insufficiencies occur under TPN, as it has been reported for copper and zinc by several investigators (Wulfseeling et al., 1979).

In 1977 Jeejeebhoy et al. gave an account of chromium deficiency in a female under TPN for more than five years. They found decreased glucose tolerance, weight loss, high free fatty acids and peripheral neuropathy and high insulin levels. All symptoms reversed after 20µg of chromium were given daily.

9. Chromium requirements during intravenous nutrition

For adult patients receiving TPN for any length of time (more than a few days) chromium should be added to the infusion mixture, in an amount of around 20-30µg/day, which together with the pre-existing chromium will give the total amount recommended, 54.6µg/day. Any slight excess chromium administered will be excreted by the kidney. For premature babies or neonates receiving TPN for few days or so it does not seem necessary to add any additional chromium to the infusion fluids in current use (Fell et al., 1979).

10. Toxic effect of chromium

Large dose of hexavalent chromium salt leads to corrosive effects in the intestinal tract and to nephritis (Snell and Snell, 1967).

Chromium carcinogenesis is documented by Baetjer (1956). Chromium is involved primarily in lung cancer, especially among workers in chromium metallurgy and refining and in dichromate manufacture from chromite ores. The cancer is not of any single histologic type; squamous cell carcinomas, round cell carcinomas and adeno carcinomas are reported. Pulmonary cancer is reportedly induced by chronic inhalation for 4-20 years. Chromium does not induce cancer in other areas of the human body nor does it increase the incidence of cancer due to other carcinogenic agents. The

actual carcinogenic chfomium compound has not yet been identified, both Cr^{+6} and fine particles of insoluble chromate ore are considered to be the agents. Monochromate dust, fumes and mist have also been judged to be hazardous. In 1971 survey of workers exposed for about 20-25 years in a German plant involved in chromic oxide and chromic sulphate (Cr^{3+}) manufacture revealed no lung cancer among the workers (N.A.S., 1974). Thus observations about the involvement of either Cr^{3+} or Cr^{6+} as the carcinogenic agent in human bronchial cancer are controversial. Chromium compounds appear to have no carcinogenic potential by the oral route. Chromium dust and fumes are a great hazard and chronic exposure increases the incidence of lung tumours (Schepers, 1981). Intramuscular or subcutaneous injection of chromite suspension in lanolin into rodents induced bronchial cancer; before injection, the chromite was leached to remove soluble chromates (Poyne, 1980). Subsequently soluble and insoluble chromates were tested for carcinogenicity; soluble compounds had to be injected repeatedly to induce cancer with intratracheal deposition, soluble chromates were not effective in inducing cancer and the incidence was low (Hueper and Poyne, 1982). It is now accepted with both Cr^{3+} and Cr^{6+} compounds are active in inducing bronchial carcinomas, with Cr^{3+} playing a key role (Alfin-Slator and David, 1980).

The carcinogen in chromium induced bronchiogenic cancer is Cr^{3+}, apparently in the form of insoluble Cr_2O_3 that has been inhaled chronically over a long period. Although the chromium content of the lungs was much greater than normal, the amount of either soluble or insoluble chromium have no direct relationship to the incidence of cancer (Luckey and Venugopal, 1977).

Experimental Procedure

III EXPERIMENTAL PROCEDURE

The experimental procedure adopted for the present study is discussed under the following headings:

1. Selection of food samples
2. Digestion of the food sample
3. Analysis of selenium and chromium levels in the selected samples
4. Statistical Analysis

1. Selection of food samples

The following food samples were selected for the estimation of selenium and chromium:

Milk: Cow's milk, buffalow's milk and Dairy milk

Egg: Hen's egg and pigeon's egg

Meat: Goat, sheep, chicken and pork

Fish: (10 varieties) Mirgear, Rough, Kalappas, Valli, Kendai, Moy, Black fish, Irlai, Mathi, Shark and Kelithi

Vegetables: Cauliflower, beans, onion, cluster beans, potato, cabbage, carrot, plantain, beetroot, brinjal, greens and tomato

Milk and egg were collected from shops in Coimbatore. All types of meats were collected from slaughter houses in Coimbatore. Vegetables were collected from Uthagamandalam and Coimbatore. All types of fish samples were collected from the Tamil Nadu Government fish shops in Coimbatore

The experiments were repeated with five samples of each food item collected in different days.

2. Digestion of the food samples (A.O.A.C., 1977)

Placed 10g of sample with 3 glass beads into 100ml Kjeldhal flask. Added 30ml of HNO_3-$HClO_4$ (5+1) solution. Allowed the sample to digest over night at room temperature. Next day the flask was heated under low flame and continued heating through $HClO_4$ oxidation which is characterised by vigorous surface reaction and evolution of white fumes. The solution was heated till it became clear and colourless and dense white fumes of sulphur trioxide appeared. The solution was cooled. The solution was colourless or faintly yellow in colour. Then this digested soltion was transferred to 100ml volumetric flask containing 30ml of hydrochloric acid and 20ml of water. It was cooled and made upto 100ml with water. One blank was digested for every 10 samples, using the same amount of all reagents to give digested reagent blank.

3. Analysis of selenium and chromium levels in selected samples

Estimation of selenium (Snell and Snell, 1967)

Principle

Selenium in an acid solution such as in hydrochloric acid is reduced to the element with a suitable reagent such

as sodium metabisulphite and the resulting yellow colour is read colorimetrically at 420 nm.

Reagents

1. Selenium powder
2. Concentrated Nitric acid
3. Concentrated Hydrochloric acid
4. Saturated sodium metabisulphite solution

All the reagents were prepared in double distilled water.

Standard selenium solution

100ml of selenium powder was weighed and a few drops of concentrated Nitric acid was added and then evaporated to dryness. This method was repeated twice. Dissolved the dried salt in 10 ml of dilute HCl (1:9) and made upto 100ml with the same (10ml of HCl + 90ml of water) (A.O.A.C.,1980).

Working standard selenium solution

1.0ml of selenium stock solution was made upto 1000ml with distilled water, so that 1 ml of solution contained 1µg of selenium.

Procedure

0.4, 0.8, 1.2, 1.6 and 2.0ml of selenium working standard was pipetted out into clean dry test tubes. 1.0ml of glycerol was added to stabilise the solution. 0.8, 1.6,

2.4, 3.2 and 4.0ml of saturated metabisulphite solution was added to the standard tubes and heated for 7 minutes at 70°C. Made up the volume to 7.0ml in all the tubes with water and read the transmittance at 420nm.

2.0ml of the treated food samples was taken and subjected to the same procedure.

Estimation of chromium (Snell and Snell, 1967)

Principle

Under acidic condition hexavalent chromium reacts with δ- diphenyl carbazide solution to form a reddish violet coloured complex which can be determined either visually or photometrically.

Interferences

Iron above 1mg/lit interferes but can be eliminated by preliminary treatment with cupferron. Other common ions generally present in water do not interfere.

Reagents

Double distilled water was used for the preparation of all reagents.

Chromium stock solution

Dissolved 282mg dried potassium dichromate ($K_2Cr_2O_7$) in double distilled water and made upto 1000ml in a volumetric flask. 1ml contained 100µg of chromium.

Chromium working standard

Pipetted 10ml chromium stock solution into a 500ml volumetric flask and made upto the mark with distilled water. 1.0ml contained 2µg of chromium. 1.0ml of this was again diluted to 1000ml. 1ml of the resulting solution contained 0.002µg of chromium.

3. Sulphuric acid 5%

4. Phosphoric acid 85%

5. Diphenyl carbazide solution

Dissolved 500mg diphenyl carbazide and 8g phthalic anhydride in 200ml 95% ethyl alcohol. Transferred it to an amber bottle and stored in a refrigerator (Reagent is stable for several months in refrigerator).

Procedure

Placed appropriate volumes of chromium working standard in 50ml volumetric flasks, covering the range upto 25mg. Included a flask containing 25ml distilled water as blank.

Took 25ml of the digested, neutralized sample in a 50ml volumetric flask. If the sample was turbid centrifuged it before transferring to the flask.

To the blank, standards and sample added the following in order, mixing after each addition:

a. 10ml 5% H_2 SO_4

b. 0.4ml phosphoric acid

c. 4ml diphenyl carbazide solution

d. Made upto mark with distilled water, mixed well and set aside for 5 minutes

Measured the optimal densities of the blank, Standard and sample using a spectro-photometer at a wave-length of 540nm or colorimeter using green filter taking water as reference.

4. Statistical analysis

Arithmetic mean

Mean value is obtained by adding together all the items and by dividing this total by the number of items.

$$\bar{X} = \frac{\Sigma X}{N}$$

Standard deviation

$$\sigma = \sqrt{\frac{\Sigma (X - \bar{X})^2}{N-1}}$$

X = Individual observations

N = Number of samples

Results and Discussion

IV RESULTS AND DISCUSSION

The results pertaining to the study "Estimation of selenium and chromium in selected foods" is discussed under the following headings:

Estimation of selenium and chromium in selected varieties of

1. Egg
2. Milk
3. Meat
4. Fish
5. Vegetables

1. Egg

Table I shows the selenium and chromium contents of different varieties of egg. Fig.1a and 1b represents the same diagrammatically.

TABLE I

SELENIUM AND CHROMIUM CONTENTS IN TWO VARIETIES OF EGG IN µg/g

Variety	Selenium Mean ± S.D.	Chromium Mean ± S.D.
Hen	0.1613±0.744	0.084 ± 0.035
Pigeon	0.1158±0.525	0.040 ± 0.02

Figure. 1.a

EGG

SELENIUM IN µg/g

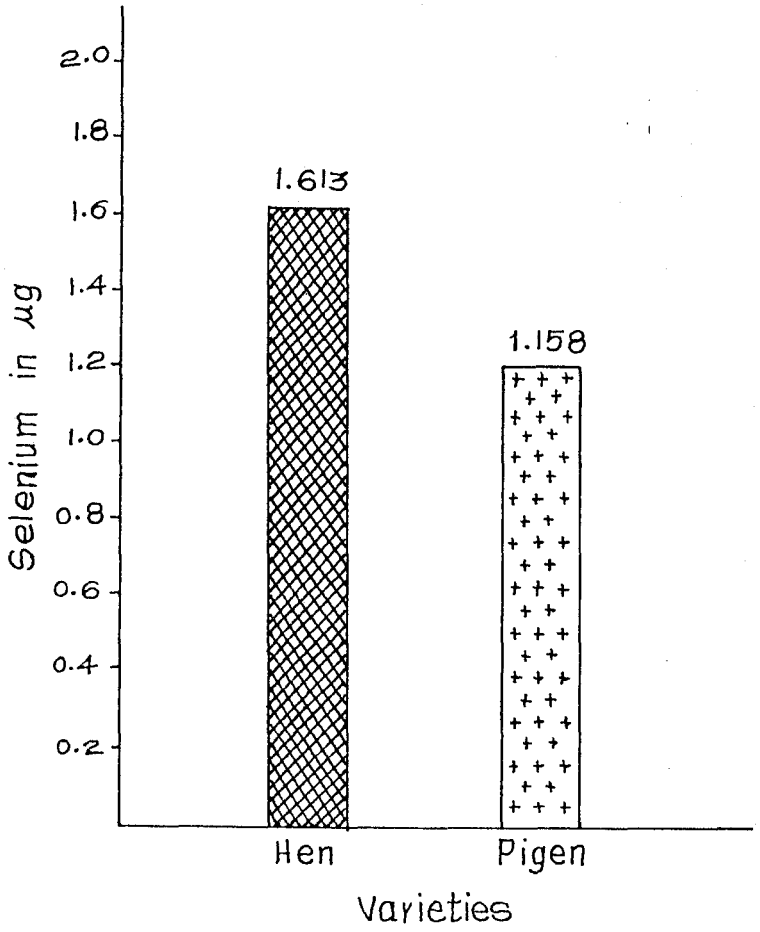
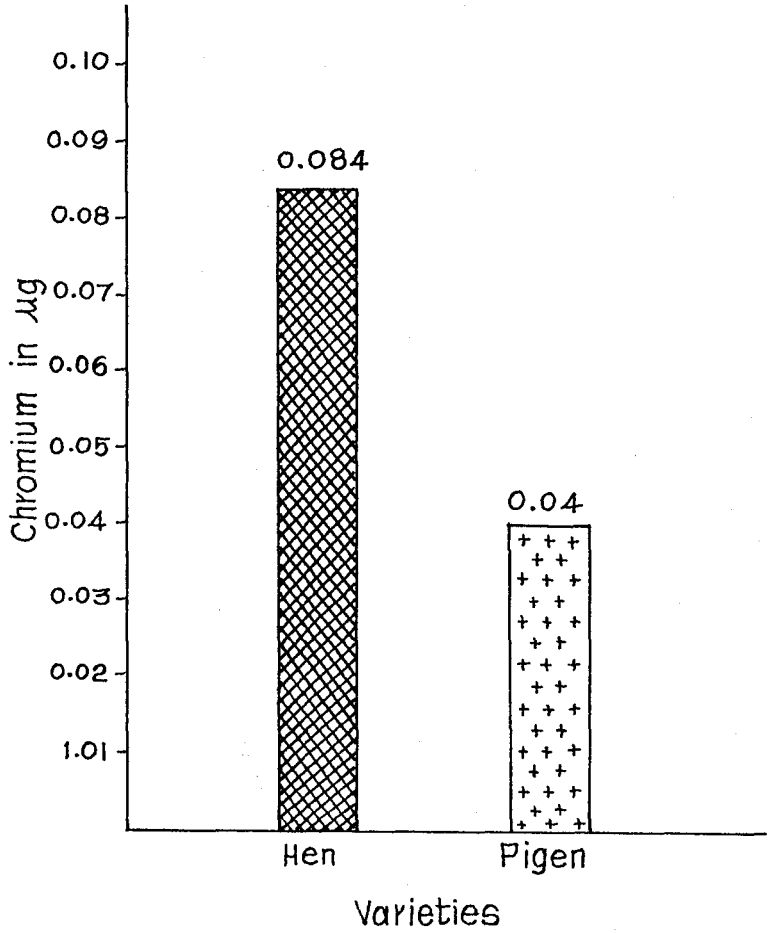


Figure. 1.b

EGG

CHROMIUM IN µg/g



It is seen from Table I that the hen's egg is richer in both selenium and chromium when compared to pigeon's egg.

According to Lavender et al. (1977) the amount of selenium present in egg is 0.1-0.2 µg/g. The present finding is in agreement with this above value. The amount of selenium present in the egg is influenced by the selenium status of the hen's diet and by chemical form or forms in which dietary selenium is supplied.

According to Alfin-Slater and David Kritchevsky (1980) the amount of chromium present in the egg ranges between 0.05-0.1 µg/g. The present study shows that in pigeon egg the chromium content (0.04 µg/g) is less than the above value.

2. Milk

Table II gives the selenium and chromium contents of different varieties of milk. Fig.2a and 2b diagrammatically represent the same.

TABLE II

SELENIUM AND CHROMIUM CONTENTS IN SELECTED VARIETIES OF MILK IN μg/dl

Variety	Selenium Mean ± S.D.	Chromium Mean ± S.D.
Cow's milk	1.9 ± 0.935	0.894 ± 0.435
Buffalow's milk	1.608 ± 0.744	1.234 ± 0.664
Dairy milk	1.648 ± 0.809	1.224 ± 0.661
Goat's milk	2.02 ± 0.929	0.978 ± 0.497

The mean selenium content of different types of milk generally consumed by man ranges from 1.608 ± 0.744 to 2.02 ± 0.929 μg/dl. Goat's milk has the highest value and the buffalow's milk samples have the lowest value.

The mean chromium content of different varieties of milk ranges from 0.894 ± 0.435 to 1.234 ± 0.664 μg/dl. Buffalow's milk has the highest value and the cow's milk has the lowest value.

The selenium content in the different varieties of milk in μg/dl increases in the following order:

Buffalow (1.608 ± 0.744) < Dairy (1.648 ± 0.809) < Cow (1.9 ± 0.935) < Goat (2.02 ± 0.929)

The chromium content in the different varieties of milk in μg/dl dncreases in the following order:

Figure. 2.a

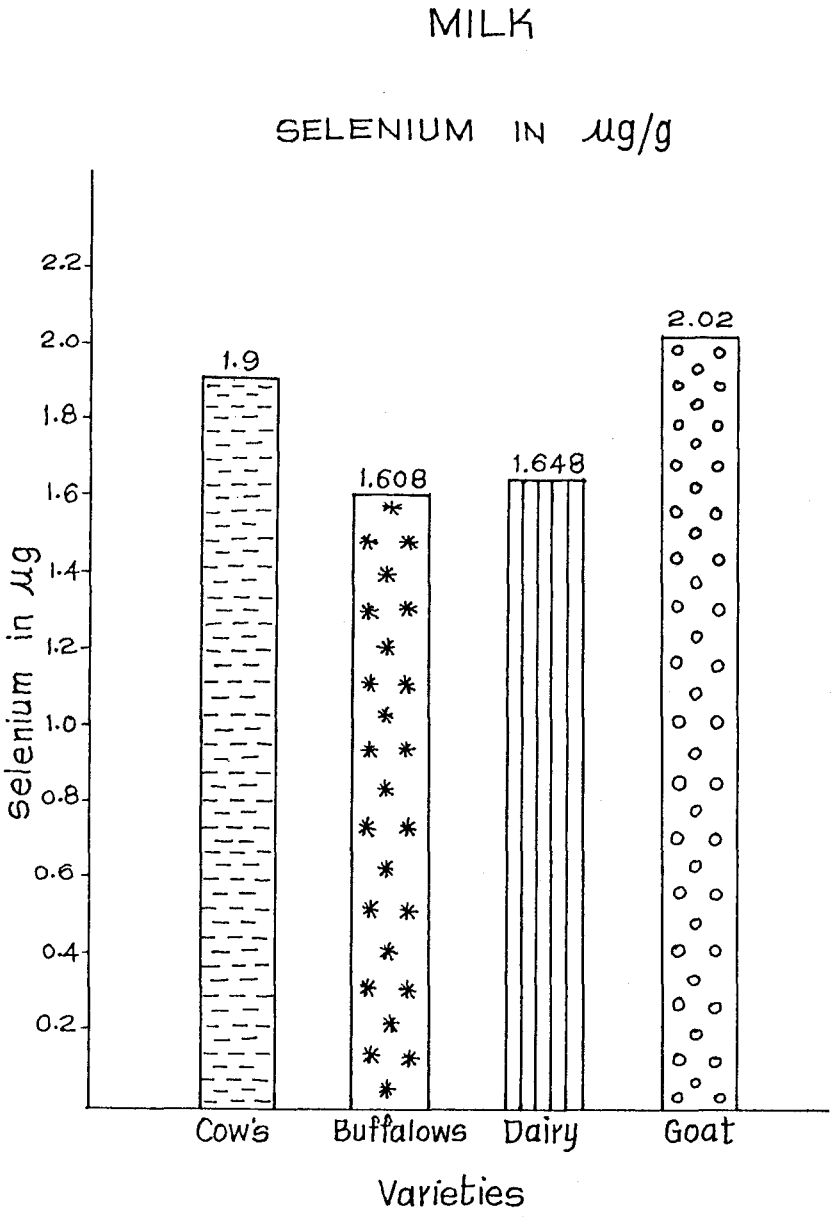
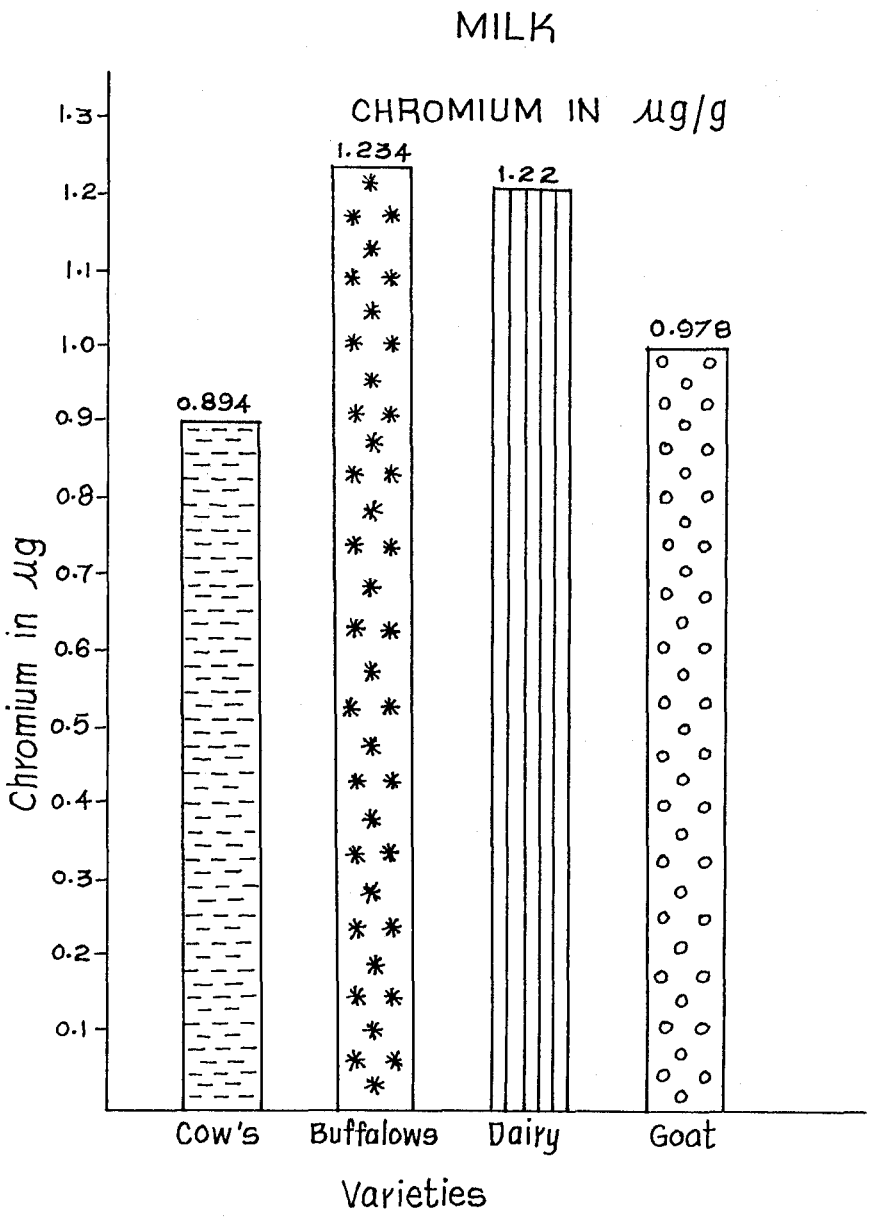


Figure. 2.b



Cow (0.894 ± 0.435) < Goat (0.978 ± 0.497) <

Dairy (1.224 ± 0.661) < Buffalow (1.234 ± 0.664)

As per the report of Bourne et al. (1981) the amount of selenium content in cow's milk ranges from 0-3µg/dl. According to Sheares (1973) the amount of selenium present in bovine milk ranges between 0.5-2.0µg/dl. The present finding shows that it ranges between 1.608-2.02 µg/dl. This increased amount may be due to the high amount of selenium present in the forage crops fed to the animals which is related to the selenium content of soil.

Bourne et al. (1981) report that the amount of chromium present in cow's milk ranges from 0-2µg/dl. In the present study chromium is present in all samples and varieties of milk studied and ranges from 0.894 to 1.234 µg/dl.

3. Meat

Table III gives the selenium and chromium contents of different varieties of meat. Fig.3a and 3b diagrammatically represent the same.

TABLE III

SELENIUM AND CHROMIUM CONTENTS IN SELECTED VARIETIES OF MEAT IN μg/g

Variety	Selenium Mean ± S.D.	Chromium Mean ± S.D.
Goat	0.849 ± 0.831	0.113 ± 0.056
Sheep	0.3814 ± 0.626	0.0986± 0.051
Pork	1.28 ± 1.001	0.128 ± 0.062
Chicken	0.323± 0.602	0.092 ± 0.054

The mean selenium content of different varieties of meat ranges from 0.323 ± 0.602 to 1.28 ± 1.001 μg/g. Pork has the highest and chicken has the lowest value.

The selenium content in the different varieties of meat in μg/g increases in the following order:

Chicken (0.323 ± 0.602) < Sheep (0.3814 ± 0.626) < Goat (0.849 ± 0.831) < Pork (1.28 ± 1.001)

The mean chromium content ranges from 0.092 ± 0.054 to 0.128 ± 0.062 μg/g. Pork sample has the highest and chicken the lowest value.

The chromium content in different varieties of meat in μg/g increases in the following order:

Figure. 3. a

MEAT

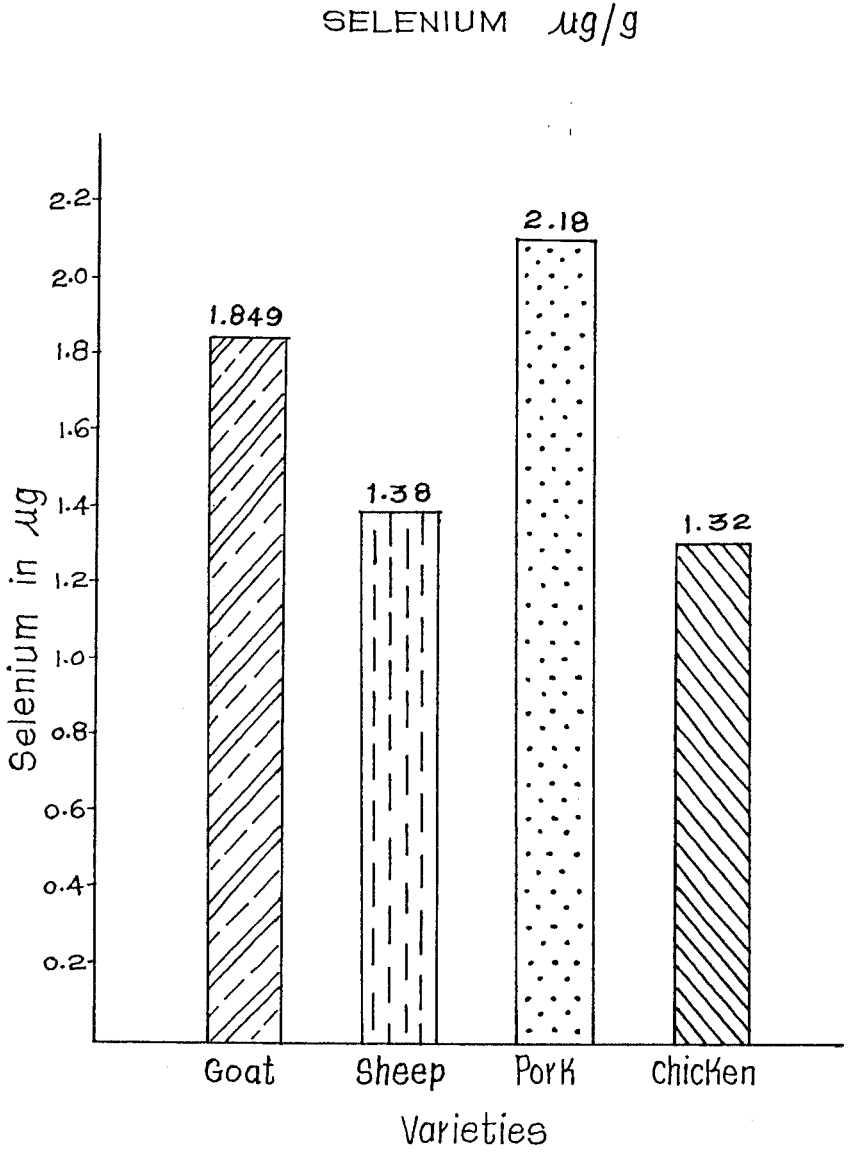
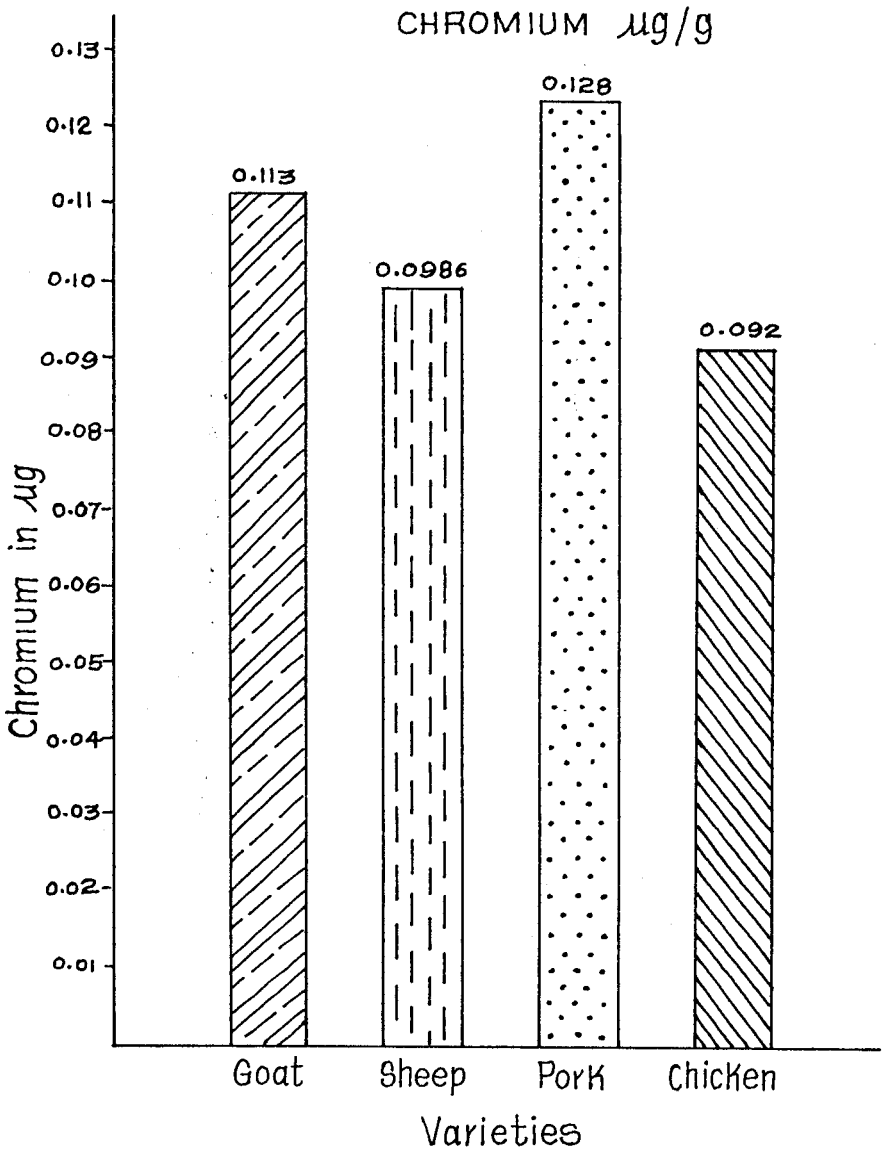


Figure. 3.b

MEAT



Chicken (0.092 ± 0.054) < Sheep (0.0986 ± 0.051) <
Goat (0.113 ± 0.056) < Pork (0.128 ± 0.062)

According to Domingue and Motario (1983) the amount of selenium present in meat ranges between 0.01-0.86 µg/g. The present finding shows the range between 0.323 to 1.28 µg/g. This increase in selenium content of meat is probably due to the higher selenium content of forage consumed by the animals.

The amount of selenium present in the crops mainly depends on the selenium present in the soil. Hence it may be considered that the soil is rich in selenium.

According to Alfin-Slater and David Kritchevsky (1980) and Kirkapatrick and Coffin (1971) the amount of chromium present in meat ranges between 0.05-0.1 µg/g. But the present finding shows that the chromium value of meat ranges between 0.092 to 0.128 µg/g. which is higher than the earlier reports. This indicates that either the forage consumed by the animals and chicken is richer in chromium or has chromium in a form easily absorbable.

4. Fish

Table IV presents the selenium and chromium contents of different varieties of fish. Fig.4a and 4b diagrammatically give the same.

TABLE IV

SELENIUM AND CHROMIUM CONTENTS IN SELECTED VARIETIES OF FISH IN µg/g

Varieties	Selenium Mean ± S.D.	Chromium Mean ± S.D.
Mirgear	0.3776 ± 0.616	0.172 ± 0.081
Rough	0.8816 ± 0.888	0.085 ± 0.037
Kalappas	0.615 ± 0.723	0.072 ± 0.033
Valli	0.9096 ± 0.853	0.222 ± 0.094
Kendai	1.1726 ± 0.989	0.103 ± 0.057
Moy	0.3992 ± 0.615	0.072 ± 0.032
Block fish	0.813 ± 0.836	0.12 ± 0.056
Irlai	0.3558 ± 0.612	0.105 ± 0.055
Mathi	1.0006 ± 0.987	0.104 ± 0.051
Shark	0.9626 ± 0.884	0.126 ± 0.055
Kelithi	0.3452 ± 0.604	0.11 ± 0.058

The mean selenium content of different varieties of fish ranges from 0.3452 ± 0.604 to 1.1726 ± 0.989 µg/g. Kendai has the highest value and kelithi has the lowest.

The selenium content of different varieties of fish in µg/g increases in the following order:

Figure. 4.a

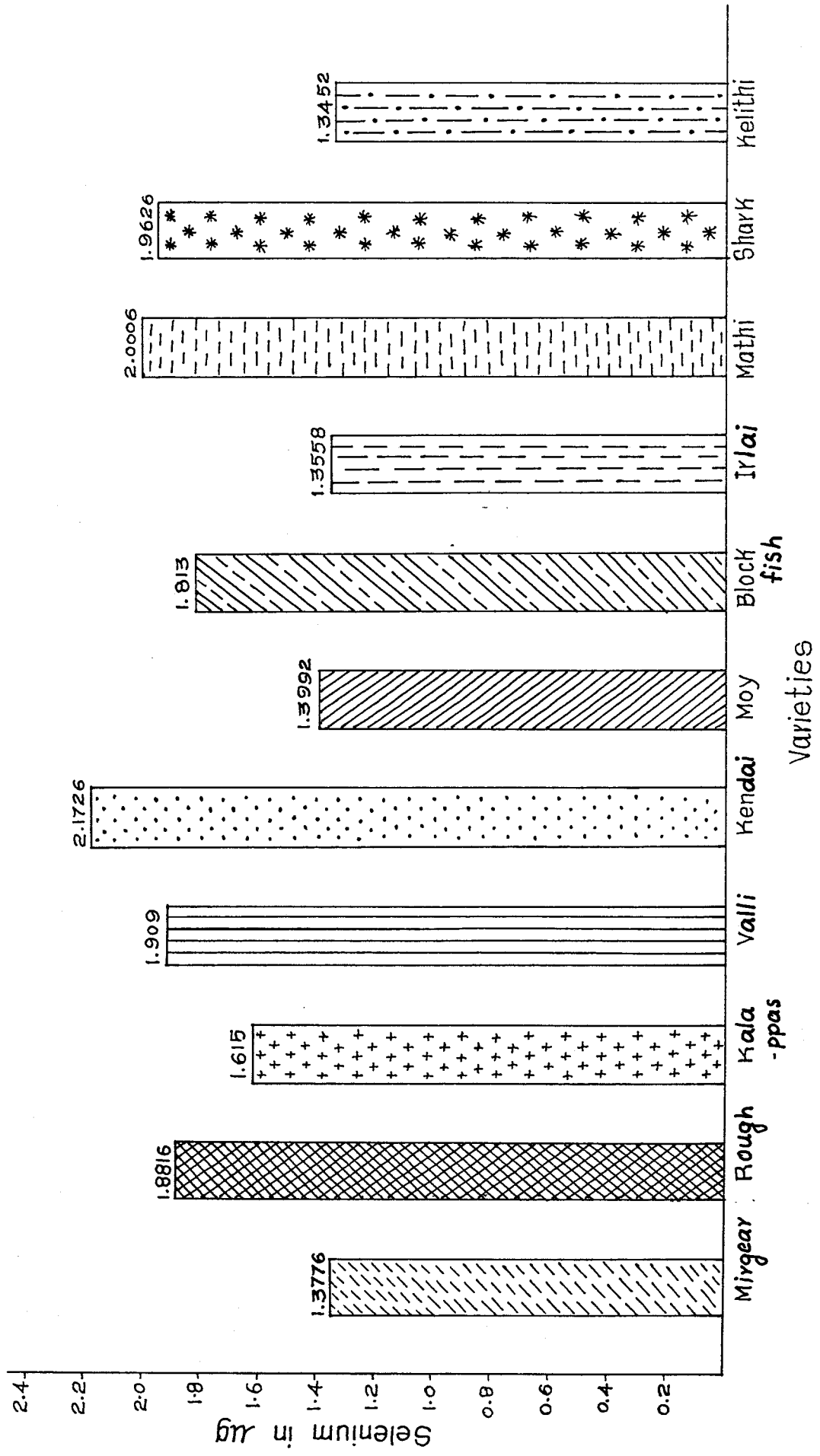
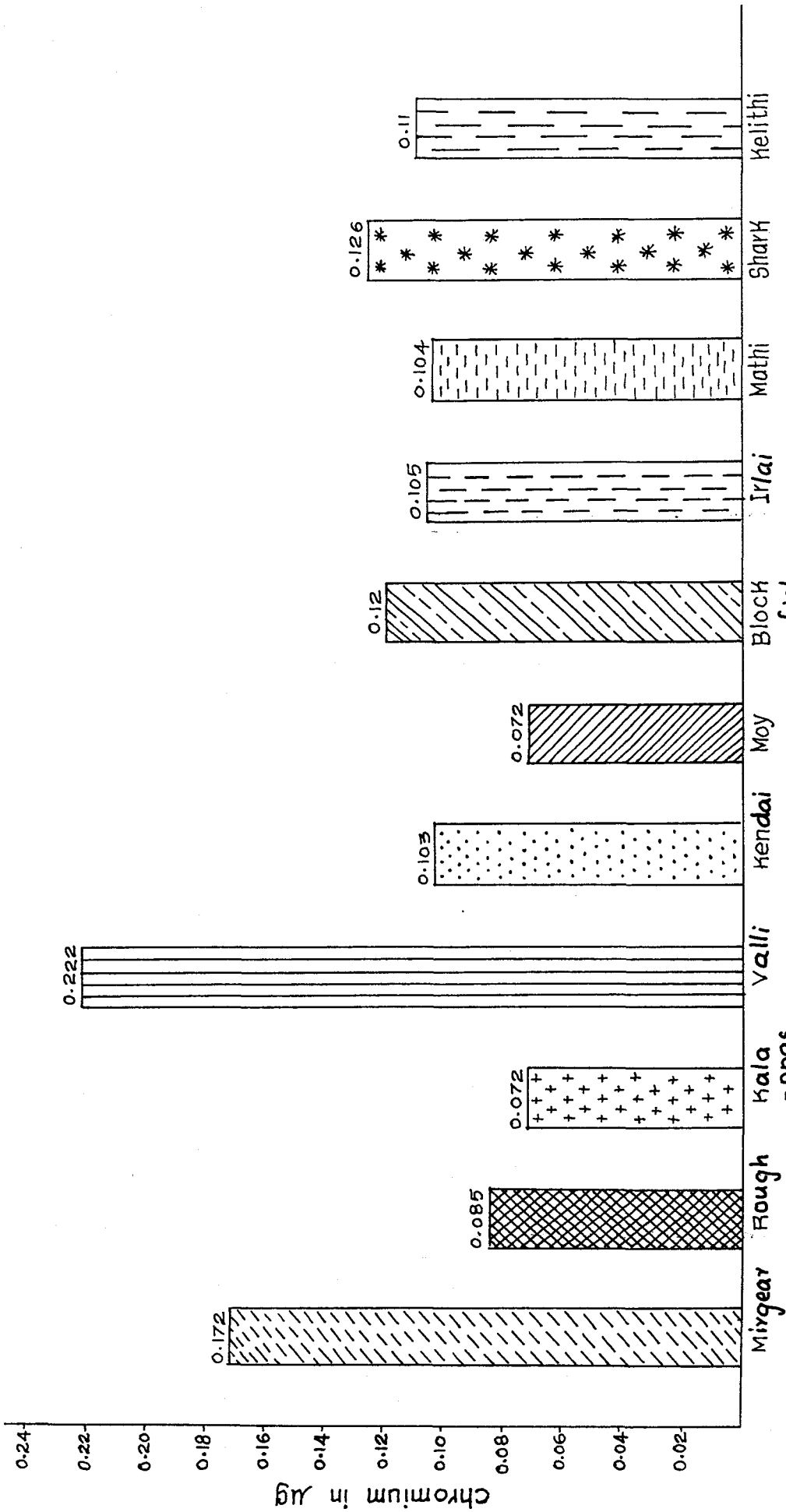

FISH
SELENIUM µg/g
Selenium in µg
2.4
2.2
2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
1.3776
1.8816
1.615
1.909
2.1726
1.3992
1.813
1.3558
2.0006
1.9626
1.3452
Mirgeay
Rough
Kala -ppas
Valli
Kendai
Moy
Block fish
Irlai
Mathi
Shark
Kelithi
Varieties

Figure. 4.b

FISH

CHROMIUM IN µg/g



Kelithi (0.3452 ± 0.604) < Irlai (0.3558 ± 0.612) <
Mirgear (0.3776 ± 0.616) < Moy (0.3992 ± 0.615) <
Kalappas (0.615 ± 0.323) < Black fish (0.813 ± 0.836) <
Rough (0.8816 ± 0.122) < Valli (0.9096 ± 0.543) <
Shark (0.9626 ± 0.884) < Mathi (1.006 ± 0.887) <
Kendai (1.1726 ± 0.989)

The mean chromium content of different varieties of fish ranges from 0.072 ± 0.032 to 0.222 ± 0.094 µg/g. Valli fish has the highest and Kalappas and Moy fish samples have the lowest value.

The chromium content of different varieties of fish in µg/g increases in the following order:

Moy (0.072 ± 0.032) = Kalappas (0.072 ± 0.033) <
Rough (0.085 ± 0.037) < Kendai (1.03 ± 0.057) <
Mathi (0.104 ± 0.051) < Irlai (0.105 ± 0.055) <
Kelithi (0.11 ± 0.058) < Black fish (0.12 ± 0;056) <
Shark (0.126 ± 0.055) < Mirgear (0.172 ± 0.081) <
Valli (0.222 ± 0.094)

According to Dutta and Miller (1983) the amount of selenium present in the fish ranges between 0.11-0.97 µg/g. Ringdal et al. (1985) reported the level of selenium in fish as 0.1-0.3 µg/g. As per the present finding the values are between 0.3452 and 1.1726 µg/g. Sea foods contain more amount of selenium compared to other foods.

According to Kirkpatrick and Coffin (1971) and Alfin-Slater (1980) the amount of chromium present in fish ranges between 0.06-0.1µg/g. In the present study the values are between 0.072 and 0.222 µg/g. This increase in value may be due to the higher content of chromium in the Bhavani Sagar Dam and Bay of Bengal from where the fishes are caught.

5. Vegetables

Table V indicates the selenium and chromium contents of different varieties of vegetables. Fig.5a and 5b represent the same diagrammatically.

TABLE V

SELENIUM AND CHROMIUM CONTENT OF SELECTED VARIETIES OF VEGETABLES IN μg/g

Varieties	Selenium Mean ± S.D.	Chromium Mean ± S.D.
Cauliflower	0.0034±0.0007	0.032 ± 0.019
Beans	0.0062±0.0008	0.044 ± 0.024
Onion	0.0408±0.020	0.025 ± 0.015
Cluster beans	0.019 ±0.0009	0.0304± 0.016
Potato	0.0434± 0.023	0.0316± 0.015
Cabbage	0.0342± 0.014	0.0304± 0.014
Carrot	0.0318± 0.015	0.0338± 0.018
Plantain	0.0362± 0.017	0.0338±0.012
Beet root	0.0466± 0.027	0.0284±0.016
Brinjal	0.0158± 0.0007	0.0428±0.019
Greens	0.008 ± 0.0003	0.0492± 0.024
Tomato	0.013 ± 0.0006	0.037 ± 0.016

The mean selenium content of the different vegetables ranges from 0.0034 ± 0.0007 to 0.0466 ± 0.027 μg/g. Beet root has the highest and cauliflower the lowest value.

The selenium content of different varieties of vegetables in μg/g increases in the following order:

Figure.5.a

VEGETABLES

SELENIUM IN µg/g

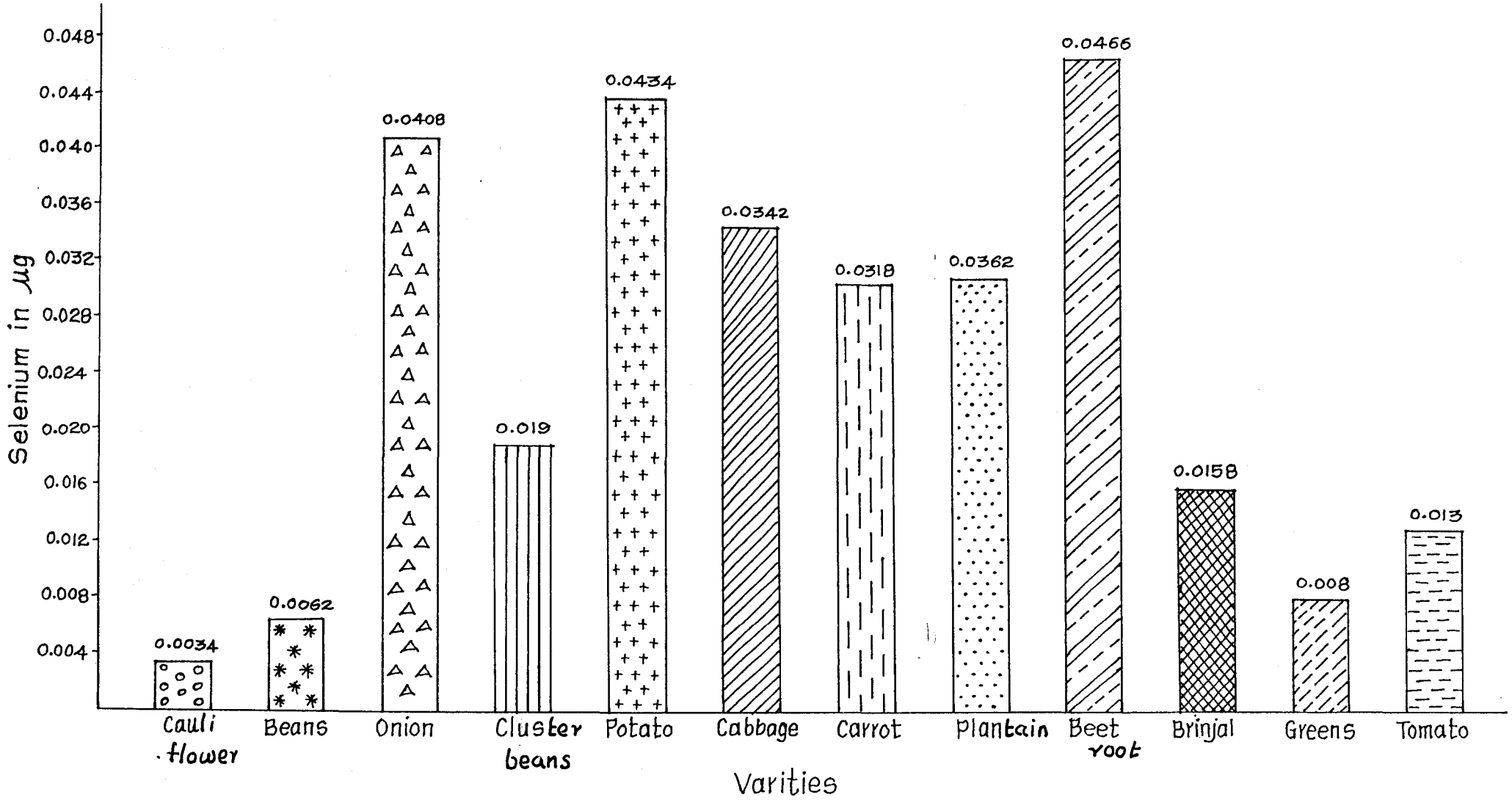
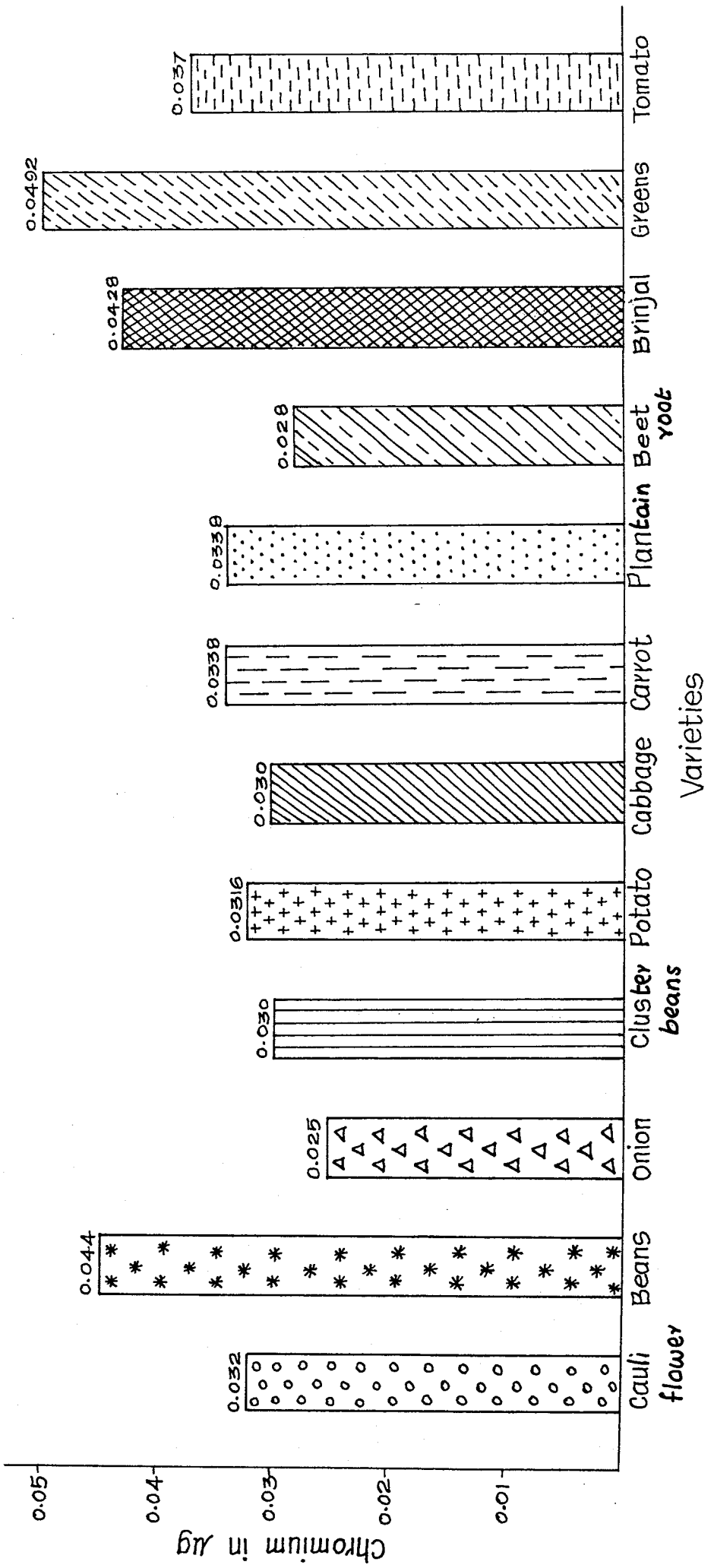


Figure.5.b


VEGETABLES
CHROMIUM IN µg/g
Chromium in µg
0.05
0.04
0.03
0.02
0.01
0.032
0.044
0.025
0.030
0.0316
0.030
0.0338
0.0338
0.028
0.0428
0.0492
0.037
Cauli flower
Beans
Onion
Cluster beans
Potato
Cabbage
Carrot
Plantain
Beet root
Brinjal
Greens
Tomato
Varieties

Cauliflower (0.0034 ± 0.0007) < Beans (0.0062 ± 0.0008) < Greens (0.008 ± 0.0003) < Tomato (0.013 ± 0.0006) < Brinjal (0.0158 ± 0.0007) < Cluster beans (0.019 ± 0.0009) < Carrot (0.0318 ± 0.015) < Cabbage (0.0342 ± 0.014) < Plaintain (0.0362 ± 0.017) < Potato (0.0434 ± 0.023) < Onion (0.0408 ± 0.02) < Beet root (0.0466 ± 0.021).

The mean chromium content ranges from 0.025 ± 0.015 to 0.0492 ± 0.024 µg/g. Onion has the lowest value and greens the highest value.

The chromium content of different varieties of vegetables in µg/g increases in the following order:

Onion (0.025 ± 0.015) < Beet root (0.028 ± 0.016) < Cluster beans (0.0304 ± 0.016) < Cabbage (0.0304 ± 0.014) < Potato (0.0316 ± 0.015) < Cauli flower (0.032 ± 0.019) < Carrot (0.0338 ± 0.018) < Plaintain (0.0338 ± 0.012) < Tomato (0.037 ± 0.016) < Brinjal (0.0428 ± 0.019) < Beans (0.044 ± 0.024) < Greens (0.0492 ± 0.024).

According to Penning et al. (1982) vegetables contain only trace amounts of selenium. Among the vegetables roots and tubers have higher amount of selenium compared to other vegetables. The present finding shows that the value ranges between 0.0034 and 0.0466 µg/g.

According to Alfin-Slater and Kritchevsky (1980) the amount of chromium present in vegetables ranges from 0.02-0.05 µg/g. In the present study the chromium value is found to be between 0.025 and 0.0492 µg/g.

The present study indicates the following:

1. Milk is a rich source of selenium and chromium.

2. Pork and fish have a high content of selenium.

3. Vegetables are poor sources of both selenium and chromium.

Summary and Conclusion

V. SUMMARY AND CONCLUSION

The trace element selenium acts as a chemopreventive agent against mammary carcinogenesis. Selenium has the ability to cure illnesses such as cancer and heart disease. Selenium deficiency is reported to produce the following: glutathione peroxidase deficiency, acute toxicity to drugs which are activated by oxygen; hepatic damage in alcoholics; congestive cardiomyopathy; anemia and pancreatic fibrosis. "Keshan disease" is the best known selenium deficiency disease in China. "Kaschin-Beck disease" affecting mainly the joints of both animals and humans, has been recognised in China and is also preventive with dietary selenium supplements.

Trivalent chromium is essential for normal carbohydrate and lipid metabolism in man. The trivalent chromium is a cofactor in several enzyme systems involved in glucose metabolism and may be necessary for insulin recepton interaction. The chromium deficiency state in the mammals has been associated with an insufficient dietary intake of chromium, a weak gastrointestinal absorption, as excessive urinary excretion, aging, diabetes mellitus, pregnancy and protein calorie malnutrition. Since selenium and chromium are important in nutrition an attempt has been made to evaluate the content of selenium and chromium in selected foods such as egg, milk, fish, meat and vegetables.

1. Egg

Two varieties of eggs (hen and pigeon) were analysed. The selenium content of Hen's egg: 0.1613 ± 0.744 µg/g;
Pigeon's egg: 0.1158 ± 0.525 µg/g;
The chromium content of Hen's egg: 0.084 ± 0.035 µg/g;
Pigeon's egg: 0.040 ± 0.02 µg/g;

2. Milk

Four varieties of milk were evaluated. Among the different varieties of milk studied goat milk had the highest amount of selenium and the buffalow's milk the lowest.

The selenium content in the different varieties of milk in µg/dl increased in the following order:

Buffalow (1.608 ± 0.744) < Dairy (1.648 ± 0.809) < Cow (1.9 ± 0.935) < Goat (2.02 ± 0.929)

Among the different varieties of milk studied, Buffalow's milk had the highest value of chromium and cow's milk the lowest.

The chromium content in the different varieties of milk in µg/dl increased in the following order:

Cow (0.894 ± 0.435) < Goat (0.978 ± 0.497) < Dairy (1.224 ± 0.661) < Buffalow (1.234 ± 0.664).

3. Meat

Four varieties of meat were evaluated. Among the different varieties of meat studied pork had the higheet amount of selenium and chicken the lowest.

The selenium content in the different varieties of meat in µg/g increased in the following order:

Chicken (0.323 ± 0.602) < Sheep (0.3814 ± 0.626) < Goat (0.849 ± 0.831) < Pork (1.28 ± 1.001)

Among the different varieties of meat studied pork had the highest amount of chromium and chicken the lowest.

The chromium content in different varieties of meat in µg/g increased in the following order.

Chicken (0.092 ± 0.054) < Sheep (0.0986 ± 0.051) Goat (0.113 ± 0.056) < Pork (0.128 ± 0.062).

4. Fish

Eleven varieties of fish were evaluated. Among the different varieties of fish studied Kendai had the highest level of selenium and kelithi the lowest.

The selenium content of different varieties of fish in µg/g increased in the following order:

Kelithi (0.3452 ± 0.604) < Irlai (0.3558 ± 0.612) <
Mirgear (0.3776 ± 0.616) < Moy (0.3992 ± 0.615) <
Kalappas (0.615 ± 0.323) < Black fish (0.813 ± 0.836) <
Rough (0.8816 ± 0.122) < Valli (0.9096 ± 0.543) <
Shark (0.9626 ± 0.884) < Mathi (1.0006 ± 0.887) <
Kendai (1.1726 ± 0.989).

Among the different varieties of fish studied valli had the highest level of chromium and Moy and Kalappas the lowest.

The chromium content of different varieties of fish in µg/g increased in the following order:

Moy (0.072 ± 0.032) = Kalappas (0.072 ± 0.033) <
Rough (0.085 ± 0.037) < Kendai (0.103 ± 0.057) <
Mathi (0.104 ± 0.051) < Irlai (0.105 ± 0.055) <
Kelithi (0.11 ± 0.058) < Black fish (0.12 ± 0.056) <
Shark (0.126 ± 0.055) < Mirgear (0.172 ± 0.081) <
Valli (0.222 ± 0.094).

5. Vegetables

Twelve varieties of vegetables were evaluated. Among the different varieties of vegetables Beet root had the highest level of selenium and cauliflower the lowest.

The selenium content of different varieties of vegetables in µg/g increased in the following order:

Cauliflower (0.0034 ± 0.0007) < Beans (0.0062 ± 0.0008) <
Greens (0.008 ± 0.0003) < Tomato (0.013 ± 0.0006) <
Brinjal (0.0158 ± 0.0007) < Cluster Beans (0.019 ± 0.0009) <
Carrot (0.0318 ± 0.015) < Cabbage (0.0342 ± 0.014) <
Plantain (0.0362 ± 0.017) < Potato (0.0434 ± 0.023) <
Onion (0.0408 ± 0.02) < Beet root (0.0466 ± 0.021).

Among the different varieties of vegetables studied greens had the highest level of chromium and onion had the lowest.

The chromium content of different varieties of vegetables in µg/g increased in following order:

Onion (0.025 ± 0.015) < Beet root (0.0284 ± 0.016) <
Cluster beans (0.0304 ± 0.016) < Cabbage (0.0304 ± 0.014) <
Potato (0.0316 ± 0.015) < Cauliflower (0.032 ± 0.019) <
Carrot (0.0338 ± 0.018) < Plantain (0.0338 ± 0.012) <
Tomato (0.037 ± 0.016) < Brinjal (0.0428 ± 0.019) <
Beans (0.044 ± 0.024) < Greens (0.0492 ± 0.024).

The present study indicates the following:

1. Milk is a rich source of selenium and chromium
2. Pork and fish have a high content of selenium
3. Vegetables are poor sources of both selenium and chromium

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