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# A Quantitative Assessment of Minerals of Toxicological Importance in Utah Fast Foods

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# A QUANTITATIVE ASSESSMENT OF MINERALS OF TOXICOLOGICAL IMPORTANCE IN UTAH FAST FOODS

by

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Lisa R. Williams

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# ABSTRACT

A Quantitative Assessment of Minerals
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X-ray flourescence (XRF) and atomic absorption spectrophotometry (AAS) measurements for manganese, iron, copper, and zinc were compared for 96 samples of 21 foods from different sources. Correlation coefficients were 0.94 for manganese, 0.99 for iron, 0.93 for copper, and 0.91 for zinc for XRF vs. AAS determinations.

Similiar comparisons were performed on 228 samples of fast foods purchased in Utah retail outlets. Correlation coefficients ranged from 0.91 for copper to 0.97 for iron and zinc. Comparisons of values generated by XRF for manganese, iron, copper, zinc, selenium, arsenic, and aluminum to values certified by the National Bureau of Standards indicated no significant differences by student's t tests.

The simultaneous multielement capabilities of XRF allowed for an extensive screening study for high levels of toxic minerals in the fast foods. Levels of selenium, arsenic, and aluminum in fast foods were determined by XRF. Inductively coupled plasma was used to screen for high cadmium levels since cadmium detection limits by XRF were too high to be of value.

(68 pages)

# INTRODUCTION

Nutritional toxicology is concerned with the scientific basis and consequences of regulatory decisions relating to control of toxicants in foods, e.g., setting legal tolerances for the maximum permissible levels of toxicants in order to assure optimal food safety. The levels of nutritional intake form a continuum from lethal deficiencies to lethal excesses. Optimal nutrient requirements of all organisms are for the level that will meet minimum nutrient needs but not in quantities large enough to be detrimental to health. A study of the full range of nutritional concerns cannot be complete without careful examination of the toxic excesses of minerals that can be found in some diets.

At the present time, the Food and Drug Administration (FDA) accords the highest priority for studies of toxic elements in foods to mercury, lead, cadmium, arsenic, and selenium (Jelinek and Corneliussen, 1977). In discussions of establishing Recommended Dietary Allowances (RDAs) for trace element intakes, the issue of whether a certain threshold would clearly delineate disease from health becomes the question. Selenium, for example, is an essential trace element that is toxic at high concentrations and that has demonstratable anticarcinogenesis properties at more moderate concentrations (Menkes et al., 1986). The difference between beneficial and toxic levels for selenium is small (Scott, 1973). Arsenic, aluminum, and cadmium have no known physiological function and therefore do not have established RDAs. They are, however, causally implicated in several known human disease states. To avoid health risks to certain population segments, their presence in any cell must be regarded as something to be minimized.

The FDA is charged with overseeing the foods and drugs on the market and those being introduced each year. It must test or order testing, assess the test results, and approve or disapprove of thousands of food products on the resulting evidence. The FDA is often under heavy pressure from food companies, which stand to gain or lose profits depending on the decisions, and the public, which wants information and protection. To

assess human consumption of a particular food substance, it is necessary to know the levels of the substance in the food and the daily intake of each food containing the substance.

It becomes a major challenge to provide up-to-date data on the mineral composition of the food supply. A gap is evident in nutritional data for epidemiological studies of restaurant foods, which are dominated by franchised fast food products. Food frequency questionnaires indicate that up to 40% of food budgets are spent on fast food products in some population segments, and these foods have not been characterized for toxic mineral levels (Nielson, 1985).

A priority goal of epidemiologists should be an improvement in the analytical methods needed to develop data on the composition of foods. Appropriate and validated methods should be used for determining the composition of foods, particularly the components whose dietary intake in the U.S., whether high or low, is of public health importance or scientific concern.

# OBJECTIVES

1. To establish and validate x-ray fluorescence as a non-destructive method of mineral analysis in dietary materials.
2. To generate useful new data on toxic mineral levels of fast foods to support current epidemiological research.

# LITERATURE REVIEW

## NEED FOR FAST FOODS DATA

### USDA Handbook No. 8

The primary source of food composition data since 1963 has been the USDA Handbook No. 8 (Gebhardt et al., 1978). These food composition tables contain more than 3000 food items and recipes. The food items are placed in 16 food groups including baby foods, dairy and egg products, vegetables, beef products, etc. Foods are categorically researched and nutrient profiles for each food item are determined. Currently, the USDA Handbook contains information on the following: food energy, vitamins, lipids, amino acids, and some minerals. Information is available on calcium, iron, magnesium, manganese, phosphorus, potassium, sodium, zinc, and copper content of foods (Gebhart et al., 1978). Information regarding minerals of toxicological importance, such as arsenic, selenium, cadmium, and aluminum, are not available.

The data used are based on values obtained from industry, government agencies, and other scientific and technical literature and are prepared using the facilities of the Nutrient Data Bank. Revision and updating of the major nutrient tables are constantly underway to provide up-to-date tabulations on food composition which can be used in national programs and government policies (Gebhardt et al., 1978). Food companies as well rely on nutrient data tables to increase the sales and acceptance of a product in any open market system (Rand et al., 1987).

### Fast foods

Fast foods are quickly becoming a major source of nutrition for many Americans. Priorities, in many cases, seem to be time and personal energy. Thus, we tend to rely on fast foods and eating out for convenience and pleasure. There is a spiraling use of fast foods, and the number of fast food chains has increased dramatically from only a handful

in the 1950s to tens of thousands today. Fast foods are those foods originating from the restaurant chains emphasizing quick preparation and service of food. On the average, about a fourth of the average teenager's total diet comes from snacks or fast foods (Hamilton et al., 1988). Busy parents find it easy to rely on fast foods to feed their children.

The first question to ask about fast foods is, "How often do I use them?" If a fast food restaurant is visited only once a week, then the food consumed there accounts for only about one meal out of 20 and has little impact on overall diet. The more often fast food places are visited, the more important are the food choices made there. The traditional fast food meal of hamburger, fries, and soft drink has now expanded to include pizza, tacos, breakfast items, salads, and other foods, which are becoming important components of the American diet.

For the most part, fast foods contribute substantial percentages of recommended intakes. However, consideration must be given to nutritional as well as toxicological levels of minerals contained in the food. Fast foods are not included as a group in USDA Handbook No. 8.

## SELENIUM

### Nutritional requirements

Selenium (Se), a mineral present in foodstuffs in trace quantities, is essential for human growth and development and for maintenance of health in adults. Selenium occurs in all cells and tissues of the body in concentrations that vary with the tissue and the amount and form of Se in the diet (Levander, 1986). The following values for human autopsy specimens have been reported: kidneys 0.61-1.84 (mean 1.09), liver 0.28-0.81 (mean 0.54) and muscle 0.11-0.38 (mean 0.24) ug Se/g of wet tissue (Schroeder et al., 1970). The concentration of Se in blood is highly responsive to changes in the diet level.

The Se levels in whole human blood from 210 donors at 19 sites in the U.S. were reported to range from 0.10 to 0.34 ug/ml (Allaway et al., 1968).

The present RDA for Se is 50-200 ug/day for adults (Lane et al., 1983). The per capita dietary intake of Se for the U.S. has been estimated at 132 ug/day (Watkinson, 1974). Certain areas of the U.S., such as Oregon, have been classified as "low Se", while other areas, such as South Dakota, have been classified as "high Se" (Christensen et al., 1988). In other parts of the world, extreme ranges are observed in China, with intakes from 30 ug/day to 4990 ug/day depending on the geographic origin of the food (Lane et al., 1983). For years, New Zealand and Finland have been known to have areas with low levels of Se in soils, plants, livestock, and human tissue (Luo et al., 1985). In addition to geographic origin, other factors that influence Se content of food include the class of food and the extent and type of processing and cooking. Ordinary cooking practices do not cause appreciable losses of the element (<14%) (Levander, 1975). Food is the major source of Se. Seafoods, organ meats, and muscle meats are generally good sources of Se (>0.2 ug/g). Grains and cereals contain variable amounts of Se depending on where they are grown. Seleniferous areas of China have levels as high as 6.9 ug/g in corn. Wheat and grains produced in other seleniferous areas, including South Dakota, are reported to have average concentrations of 0.4 to 1.0 ug/g (Lo and Sandi, 1980). Fruits and vegetables are mostly poor sources (<0.01 ug/g wet weight) (Levander, 1986). The human dietary requirement for Se is likely to be in the range of 0.1 to 0.2 ug/g (Levander, 1975).

Selenium has been implicated in many aspects of nutrition, toxicology, and medicine for much of this century. Although Se is an essential trace element, it is toxic at high concentrations (Menkes et al., 1986). Levels in the range of 2 to 10 ug/g produce a chronic toxicity, while levels above 10 ug/g produce drastic changes resulting in sudden death (Scott, 1973). The U.S. Department of Agriculture has placed the minimum dietary Se levels at which signs of toxicity will ultimately arise at 3-4 ug/g, but this will clearly

vary with the extent to which other dietary components with which Se interacts are present (Levander, 1986).

Bioavailability

Biological availability of Se ranges widely with the type of food and form of the element. Selenium in plant materials, such as corn and wheat, is readily available (79%), while in various fish and animal products, it is much less available (9-25%) (Combs and Combs, 1986). Selenium occurring naturally in foods is predominantly organic (e.g., selenoamino acids) rather than inorganic, and some inorganic Se compounds, such as selenous acid, are poorly retained (1-10%). Other inorganic forms of Se, such as selenite and selenate, are highly absorbable (92-100%) (Combs and Combs, 1986). Some of the Se supplements which are being marketed contain inorganic Se and cannot be recommended for human use because they contain the element in a reduced form not normally found in foods. Loading studies with human subjects revealed the Se supplements do not produce a rise of the blood Se even after 5 weeks of continuous supplementation (Schrauzer and Ishmael, 1974). Selenite is generally regarded as the most toxic chemical form of Se. Of almost equal magnitude are the selenoamino acids and selenate. Organic Se exhibits less toxicity. Selenium in its most toxic form is toxic when supplied at a level of 1-5 mg Se/kg body weight (Martin and Gerlach, 1972).

Selenium is rapidly absorbed and incorporated into the body pool. Approximately 95% of Se is absorbed over a range of dietary Se intakes from deficient to mildly toxic levels (Levander, 1986). The liver concentrates more Se than any other organ. Detoxifying organs tend to accumulate the highest quantities of Se. Selenium excretion is primarily by way of the kidney. Significant quantities can be eliminated through the lungs, however (Martin and Gerlach, 1972).

## Toxicity

Selenium toxicity is rare and not a major problem in humans (Lane et al., 1983). An individual who had been ingesting 600 ug/day for prolonged periods was tested and demonstrated normal health (Schrauzer and White, 1978). Mild toxicity symptoms would be expected to become observable after the prolonged ingestion of 2000-3000 ug/day. In certain high Se areas, such as Venezuala, such high Se intakes are not uncommon (Jaffe, 1976). In high Se areas of China with reported cases of chronic human selenosis, the daily intake averages 4990 ug/day (Levander, 1986). Acute Se poisoning produces central nervous system effects, liver and spleen damage, decayed teeth, gastrointestinal distress, loss of hair and nails, loss of fertility, and congenital defects (Goyer, 1986). Acute effects are found at a daily dietary Se intake of 8 ug/g food (Levander, 1986). A maximum daily intake of 500 ug/day has recently been suggested as the upper limit for the maintenance of good health (Schrauzer et al., 1977).

Twelve cases of human selenosis resulting from ingestion of overly potent Se tablets meant to be consumed as a health food supplement were reported to the FDA in 1984. The tablets contained 27-31 mg of Se, 75% in the form of organic Se. Symptoms reported were nausea, vomiting, nail changes, hair loss, cramps, diahrrea, and garlic breath. Liver function tests were normal (Levander, 1986).

## Deficiencies

The only documented naturally occurring human Se deficiency diseases are a cardiomyopathy called Keshan disease and a degenerative joint disease called Kashin Beck disease, which is found in Chinese women and children who consume less than 30 ug Se/day (Lane et al., 1983). The disease responds extremely well to Se supplementation, which reduces dramatically the number of deaths (Levander, 1986).

In animals, Se has been found to be necessary for the prevention of various diseases. These include liver necrosis in rats; exudative diathesis and pancreatic fibrosis in poultry; muscular dystrophy in lambs, calves, and other species; and hepatosis dietetica

in pigs (Levander, 1986).

Cancer

An inverse relationship exists between serum Se levels and subsequent incidence of cancer (Knekt et al., 1988). Some human epidemiology studies show that a low Se concentration in serum increases the risk of human cancer (cancer of the stomach, esophagus, colon, lung, prostate, and breast) (Miyamato et al., 1987). Some case-control studies have demonstrated lower blood Se levels among cancer patients (Knekt et al., 1988). Lung cancer patients have very low serum Se concentrations (Miyamato et al., 1987). These reports do not prove a causal relationship, but they support the hypothesis that the human mortalities from certain neoplastic diseases are controlled by dietary Se intakes (Watkinson, 1974). Although Se may not be effective in the treatment of advanced human carcinoma, Se may be one of several trace elements which retard or prevent tumor development (Schrauzer, 1976).

The epidemiological evidence that Se may produce cancer-protecting effects in humans is supported by the observed significant reduction of the incidence of spontaneous mammary tumors in Se-treated C3H mice (Schrauzer, 1976). The occurrence of mammary tumors in inbred tumor-prone mice dropped from 82 to 10% incidence when their water supply contained 2 ug/g Se (Shrauzer and Ishmael, 1974; Griffin, 1979; Shultz and Leklem, 1983; Lane and Medina, 1983; Temple and Basu, 1987). This work, typical of many animal studies, may have relevant implications for human cancer mortalities.

It should be mentioned that Se has been suspected of being a carcinogen. Based on early studies, Se was included as a carcinogen in the Delany Clause of the Food Additive Amendment of 1958 (Griffin, 1979). As reviewed by Shapiro, several authors have concluded that there exists only incomplete knowledge on which to base the assumption that Se is carcinogenic (Shapiro, 1973).

Selenium may have an anti-cancer effect as an integral part of glutathione peroxidase (Knekt et al., 1988). Glutathione peroxidase, one of the important enzymes in the prevention of oxidative damage to cellular membranes, contains four Se atoms per mole of protein (Oh et al., 1974). The enzyme participates in the elimination of hydrogen peroxide and other organic hydroperoxides. Such free radicals may damage cellular membranes and thus be promotors in the initiation of carcinogenesis. According to this concept, Se, by its antioxidative effect, has a blocking action in tumorogenesis (Sundstrom et al., 1984). Decreases in glutathione peroxidase correlate with lesions caused by Se deficiency (Oh et al., 1974).

Selenium is required for the mixed function oxidase P-450 system, which is an important system in the elimination of xenobiotics involved in carcinogenesis. Selenium deficiency in rat liver has eliminated the classic induction of P-450 by phenobarbital (Oh et al., 1974). The mechanism of action is not fully understood. The altered P-450 system may be allowing the persistant presence of carcinogenic substances, which otherwise would be eliminated by the P-450 system (Oh et al., 1974).

Some scientists have recommended 250-300 ug Se/day as an "optimal cancer protection level" against the most common cancers such as skin, colon, and mammary cancers (Schrauzer et al., 1977). The aim of experimental studies has been to examine the feasibility of nutritional prophylaxis to protect against cancer. Calculations indicate with remarkable internal consistency that the most common cancer mortality rates should decline significantly if the dietary Se intakes were increased to about twice the U.S. value. This amount would not be harmful and is estimated to lie between 200-300 ug/day for the average adult (Shrauzer et al., 1977). Supplementation of 200ug of organically bound Se for five weeks results in a rise of plasma Se levels which is not associated with an increase in glutathione peroxidase activity (Stead et al., 1985).

The importance of dietary habits is indirectly emphasized by the finding that mean serum Se concentration in patients with total cancer remission increased significantly

although they did not recieve medical Se supplementation (Sundstrom et al., 1984). The risk of developing cancer is higher with low Se intake over a lifetime (Miyamato et al., 1987).

Interactions with other minerals

Complicating aspects of the nutrition of Se are the interrelationships with other minerals. Arsenic, mercury, cadmium, and copper render Se much less toxic than when it is present alone. The presence of arsenic shifts the excretion of Se to the bile (Griffin, 1979). Selenium reduces the toxicity of heavy metal ions like cadmium and mercury (Pories, 1972). Selenium has been reported to protect against cadmium-induced hypertension in rats, although Se given alone results in increased blood pressure (Perry and Erlanger, 1974). Selenium has been shown to protect against cadmium-induced injury of pancreatic B-islet cells (Vahouny, 1982).

The joint administration of Se and arsenic (2 ug/g each) showed that the protecting effects of Se against cancer are significantly counteracted by arsenic, causing a tumor incidence essentially as high as normally observed in controls unsupplemented with Se (Griffin, 1979).

The joint administration of zinc (200 ug/g) and Se (5 ug/g) with drinking water abolishes the cancer-protecting effects of Se and accelerates cancer growth presumably because Se uptake is prevented by zinc (Schrauzer and Ishmael, 1974).

These observations in animals have given rise to epidemiological studies which suggest that trace minerals are also controlling human cancer mortalities. It is unsettling that the combined dietary intakes of arsenic and zinc alone are as a rule higher than those of Se (Shrauzer and Ishmael, 1974).

# ARSENIC

## Nutritional significance

Arsenic (As) is one of the more common toxic trace metals in the environment (Brown et al., 1976). There are many ways in which a person can be exposed to arsenic. It can be ingested in drinking water, food, or medicine; inhaled; or absorbed through the skin through contact with arsenical dusts or solutions. Because small amounts of As are present in the environment, everyone is exposed (Jackson and Grainge, 1975).

Most human foods contain <0.3 ug/g and rarely exceed 1 ug/g on a dry basis, with the exception of seafood, which commonly contains more that 1 ug/g As. The following ranges of As have been reported in foods (dry weight): cereals 0.05 - 0.4 ug/g, vegetables 0.05 - 0.8 ug/g, fruits 0.03 - 1 ug/g, meat 0.005 - .1 ug/g, milk 0.01 - 0.05 ug/g, eggs 0.01 - 0.1 ug/g, and fish 2 - 80 ug/g (Anke, 1986). The total amount of As ingested daily by humans is strongly influenced by the amount of seafood included in the diet. The total diet monitoring program carried out by the FDA since 1967 shows that the average daily intake of As has decreased drastically from about 130 ug/day in 1968 to about 20 ug/day in 1974. It is believed that much of this drop may be due to the decreased use of As-containing pesticides on food crops since the late 1960s. In analyses carried out on individual foods, the highest levels were found in fish, with a mean level of 1.47 ug/g of fish (Jelinek and Corneliussen, 1977). Seafood, especially crustaceans, may contain As in concentrations as high as 100 ug/g (Anke, 1986). Fortunately, almost all of this As is in the form of organoarsenic compounds, which are nontoxic and are not metabolized to toxic forms in the human body (Anke, 1986).

## Bioavailability

For the assessment of toxicity and bioavailability of As, knowledge of the extent of absorption, excretion, and retention of the element is important. There are two

common forms of inorganic As in the environment: arsenite (As 3+) and arsenate (As 5+). Arsenite is considered the more toxic of the two, but arsenate is the more common (Brown et al., 1976). In the 19th century, a number of lethal poisonings occurred in people who were inhaling minute amounts of trimethylarsenic from wallpaper. Trimethylarsenic has been reported as a volatile neurotoxic compound. Excretion of absorbed As is mainly via urine. The biologic half-life of ingested As is about 10 hours. Arsenic concentrates in the skin and is excreted by desquamation of the skin and in sweat (Goyer, 1986). Seafood contains As primarily as arsenobetaine, which is quickly absorbed and rapidly excreted by humans. Humans excrete 74% of 25 mg As ingested from lobster within 48 hours, and As in shrimp is completely excreted by humans within 4 days after consumption (Anke, 1986).

Toxicity

Ingestion of large doses of As (70-180 mg) may be acutely fatal (Goyer, 1986). Large amounts of arsenate uncouple oxidative phosphorylation, resulting in impaired tissue respiration (Brown et al., 1976). It has been proposed that As inhibits energy -linked functions of mitochondria in two ways: competition with phosphate during oxidative phosphorylation and inhibition of energy-linked reduction of NAD (Mitchell et al., 1971).

The symptoms of acute As poisoning in humans by the oral route include gastrointestinal effects, fever, anorexia, hepatomegaly, melanosis, and cardiac arrhythmia. Other features include upper respiratory tract symptoms and peripheral neuropathy (Goyer, 1986). The biological basis for these disturbances is probably an inhibition of a wide range of enzyme systems (Anke, 1986). Hamamoto reported the poisoning of Japanese infants who ingested an average of 3 mg of As per day over a period of 33 days (Anke, 1986). Absorbed As crosses the placenta and is transferred to the fetus, causing embryotoxic and teratogenic effects correlated with the dose reaching

the offspring. For example, a reported case of attempted suicide by arsenite ingestion at 30 weeks of gestation resulted in the death of the infant following premature delivery (Hood et al., 1988).

Cancer

For at least 2500 years, As has been used in medicine. Over the past 150 years, it has been an important drug to treat dermatitis, asthma, syphillis, epilepsy, psoriasis, and amebiasis. Treatment for long periods of time with large amounts of As in the form of Fowler's solution has been clearly correlated with the development of malignant disease, particularly carcinoma of the skin (Jackson and Grainge, 1975). A selected review of the world literature on skin and internal cancer caused by ingestion of inorganic As shows that out of 916 individuals exposed to trivalent As, skin cancer developed in 642 and internal cancer in 58 (Jackson and Grainge, 1975). Clinical evidence that long-term ingestion of As predisposes one to skin cancer is also found in studies of populations whose drinking water has been contaminated with As (12.2 ug/g). In many of these people, Reichenstein's disease developed with gastrointestinal symptoms, mouth ulcers, and melanosis and skin tumors in high incidence. In Argentina, where well water has a high concentration of inorganic As (2.8-4.5 ug/g), Ayerza's disease often develops after a person has drunk the polluted water for five or six years. Keratosis and skin cancers develop, mainly in the trunk and limbs. Fatalities result from liver and kidney ailments (Jackson and Grainge, 1975).

The relationships between ingestion of As with skin cancer and angiosarcoma and inhalation of As-containing particulates and lung cancer establishes As as a human carcinogen. Hepatic angiosarcoma has been considered rare in humans, although it is now known that exposure to inorganic arsenicals may be followed by development of these tumors (Popper et al., 1978). Arsenic has specific effects on the endothelial cells of the blood vessels in the liver, and angiosarcoma of the liver has been reported in vineyard

workers following many years of exposure to As-containing compounds, drinking water, Fowler's solution, wine, and As-containing pesticides. Skin cancers from occupational exposures have been well documented, particularly in the last 20 years. Available data suggest a dose response relationship. Workers engaged in the production of As-containing compounds, where exposure is very high, are reported to have an increased risk of dying from lung cancer (Goyer, 1986).

## ALUMINUM

### Nutritional significance

Aluminum (Al) is the third most abundant naturally occurring element and the most common metallic element. Daily exposure to Al is inevitable due to its abundance and ubiquitous occurrence in nature and its diverse use by man. Food is a source of Al, as it is found in vegetation and in all vertebrae species (Koo and Kaplan, 1988). Early studies suggested that the daily ingestion of Al from food sources varies between 24 and 36mg. However, later evidence points to ingestion of fewer than 3 to 5 mg/day. Aluminum is used as a filler in pickles and cheese and is a major component of baking powder (Alfrey, 1986).

Another possible source of ingested Al is the leaching of this element from cooking utensils during the preparation of food. It is estimated that 20% of the daily intake of Al comes from cooking utensils. One pack of Chinese noodles is estimated to contain 3.3mg of Al, including 2.6mg of Al released from the Al pan (Inoue et al., 1988). The Al content of coffee brewed in a ceramic pot is 0.15 mg/cup as compared with coffee brewed in an Al perculator, which contains 0.55 mg/cup (Lione et al., 1984). However, despite all the exposure, little ingested Al is actually absorbed (Alfrey, 1986). The use of Al in the processing and storing of food increases its Al content but not enough to contribute significantly either to total body burden or toxic effects (Doull, 1982).

There is no physiological role for Al in humans. Trace amounts of this element

are toxic to the nervous system. A growing number of reports suggest that toxic levels of Al can be derived from dietary sources and may accumulate in human tissues (Lione et al., 1984).

## Bioavailability

Normally, absorbed Al is excreted in the urine, so people with full kidney capacity easily eliminate the element. When this route is absent, Al accumulates in tissues and body fluids, giving rise to the clinical conditions of dialysis osteomalacia and dialysis encephalopathy (Gammelgaard and Sandberg, 1989).

The absorption of Al in both normal patients and patients suffering from chronic renal failure was studied. Both groups of patients received approximately 2.5g of Al daily for 23-27 days. In the normal group, the maximum absorption of Al was approximately 97 mg/day (3.9%), while in the renal-failure patients, it was 256 mg/day (10.2%) (Doull, 1982; Gorsky et al., 1979). Balance studies show that subjects excrete more than 96% of ingested Al. This correlates with the fact that tissue Al levels have uniformly been found to be low in normal individuals (Alfrey, 1986).

Small amounts of orally ingested Al are absorbed and deposited in the brain by humans with ostensibly normal renal function. Since Al is found in the environment, human diet, and in many commercial antacid preparations, it is possible that individuals might ingest enough Al during the course of their lives to cause behavioral or neurological impairment (Gorsky et al., 1979).

## Toxicity

Aluminum is unquestionably neurotoxic (Birchall and Chappell, 1988). It is well established that Al is causally implicated in the generally fatal brain disease dialysis encephalopathy, a disease seen mostly in patients with chronic renal failure treated with hemodialysis (Flaten and Odegard, 1988). Aluminum in the water supply is a major source of Al contamination in dialysis fluid, since Al is used as a flocculant during water

purification procedures (Koo and Kaplan, 1988). It is estimated that as much as 2-4g of Al is given to patients during each dialysis (Alfrey, 1986). That Al is neurotoxic in patients with renal failure does not necessarily imply that Al is harmful for persons with normal kidney function since kidney patients are exposed to extremely large amounts of Al and lack the most important route of Al excretion (Flaten and Odegard, 1988).

Abnormally high brain-Al content has been detected in hemodialysis patients who die of encephalopathy. An analysis of cerebral grey matter revealed an Al level of 25 ug/g in dialysis patients as opposed to 2.2 ug/g in control groups of nondialyzed subjects (Gorsky et al., 1979).

There is considerable evidence that Al is somehow related to Alzheimer's disease (Flaten and Odegard, 1988). Aluminosilicates have been identified at the core of senile plaques in Alzheimer's disease. Aluminum has been found to interact with silicic acid, a normal component of plasma, to form aluminosilicate species solubilized by citrate. Aluminum has been detected in neurons bearing neurofibrillary tangles both in Alzheimer's disease and in Parkinson's disease (Birchall and Chappell, 1988).

Aluminum ingestion in chronic hemodialysis patients may result in bone disease suggestive of Al-related osteomalacia. The entry of Al by the gut route is important clinically because two groups of patients, those on long-term hemodialysis and those with chronic peptic ulcer disease, frequently ingest large quantities of Al-containing preparations. The gut barrier is permeable to Al under conditions of high oral intake. Idiopathic osteoporosis, a rare bone disease resulting in bone pain and disabling bone fractures, has been reported to coexist in patients with chronic peptic ulcer disease who are ingesting large quantities of Al-containing antacids (Becker et al., 1977).

The Council of the European Communities has made a resolution for the protection of dialysis patients calling for the minimization of the exposure to Al. However, these limits are not mandatory (Gammelgaard and Sandberg, 1989).

# CADMIUM

## Nutritional significance

One of the trace elements not needed for normal metabolism in the human body is cadmium (Cd). Cadmium is virtually absent from the human body at birth, and accumulates with age up to approximately 50 years. Food is the major source of Cd for humans (Kostial, 1986). Normal daily intake from food in North America and Europe varies but generally averages 30-50 ug/day, with typical foods containing 0.06 ug/g Cd (Whanger, 1982). The greatest concentrations are found in liver and kidney, which contain 0.1 to 1 ug/g (Goyer, 1986). Even greater concentrations are found in shellfish, e.g., oysters, which have been reported to contain up to 5 ug Cd/g (Sharma et al., 1983). Cadmium pollution of soil in which normal crops are grown can originate from sewage sludge, phosphate fertilizers, or industrial waste (Sherlock, 1984).

The WHO/FAO Joint Expert Committee on Food Additives has recommended a provisional tolerable weekly intake of 400-500ug (60-70 ug/day) of Cd. This intake is the daily intake, which during an entire lifetime, appears to be without appreciable risk (Sherlock, 1984). This margin of safety is small and leaves little room for intake from other environmental or occupational sources (Drury and Hammons, 1979).

Because of the potential for accumulation in kidney, there is considerable concern for levels of dietary intake of Cd of the general population. Studies from Sweden have shown a steady increase in Cd content of vegetables over the years. Increase in body burden was determined by an historic autopsy study (Goyer, 1986). Some individuals have consistently higher intakes because they have atypical dietary habits or from food grown in Cd-contaminated soil (Sherlock, 1984).

## Bioavailability

About 5% of Cd ingested by humans is absorbed (Sharma et al., 1983). The rest passes into the feces (Goyer, 1986). The half-life is not known but may be as long as 30 years. About 50 to 75% of the body burden of Cd is in the liver and kidney. With continued retention, there is progressive accumulation in soft tissues (Goyer, 1986).

## Toxicity

Cadmium is toxic to virtually every system of the human body. Acute toxicity may result from ingestion of the relatively high concentrations of Cd as may occur in contaminated foods and beverages. Initial symptoms include nausea, vomiting, diahrrea, muscle cramps, and salivation. When fatal intoxication occurs, these symptoms are followed either by shock due to the loss of liquid and death within 24 hours or by acute renal failure and cardiopulmonary depression and death within 7 to 14 days (Doull, 1982).

Acute exposure to a large Cd dose is known to result in liver injury. This is preceeded by the enhanced formation of metallothionein, a Cd-binding protein, and possibly by changes in cellular glutathione. These effects may contribute to deterioration in cellular metabolic integrity, resulting in subclinical liver dysfunction (Muller et al., 1988).

Of great importance are studies that investigate adverse health effects of chronic exposure at low levels as may be encountered in the environment. People exposed to high levels of Cd via polluted food or water typically develop damaged renal tubules. For the most part, intakes from food are the most important contributors to exposure (Sharma et al., 1983). The principal long-term effects of low-level exposure to Cd are chronic obstructive pulmonary disease and emphysema and chronic renal tubular disease. There also may be effects on the cardiovascular and skeletal systems (Goyer, 1986). Concentrations of 0.41-0.59 ug/g Cd in food would produce severe renal tubular damage by age 50 (Whanger, 1982).

Itai-Itai disease, which is characterized by osteomalacia and is prevalent among elderly women of Japan, is a disease caused by high oral intake of Cd for a long time (Kostial, 1986). Cadmium has been shown to induce hypertension and to cause cardiotoxicity in laboratory rats. Reports from the Soviet Union have shown a four-fold higher incidence of cardiovascular disease in factory workers exposed to cadmium oxide (Jamall and Smith, 1985).

Cancer

Cadmium has been implicated in the increase in the incidence of prostate and other cancers in men exposed to high levels of Cd. A statistically significant difference in the incidence of prostate cancer was found, i.e., 10.6/100,000 population in low- and 53.2/100,000 population in high-Cd-level areas. Cadmium is a known carcinogen for several tissues in animals, e.g., sarcomas, lung cancer, and Leydig cell tumors of the testis (Webber, 1985). Recent reports of lung carcinogenesis in rats following chronic inhalation of Cd are consistent with epidemiological data concerning lung tumor incidence in humans occupationally exposed to this metal (Waalkes et al., 1988). The role and mechanism of Cd action in carcinogenesis is not clear (Webber, 1985).

Interactions with other minerals

In several acute toxicity studies, it has been shown that Se is the most effective Cd antagonist when both agents are administered simultaneously. It has been suggested that Se may be useful in counteracting the effects of Cd-related carcinogenesis in humans since Se has a strong tendency to form complexes with metals (Webber, 1985). Cadmium affects the metabolism of the essential trace metal copper, and copper supplements protect against some of the toxic effects of Cd (Jamall and Smith, 1985). Simultaneously supplementing a diet with zinc, copper, and manganese results in decreased concentrations of Cd (Piscator, 1976).

# ANALYTICAL INSTRUMENTATION

## Atomic absorption spectroscopy

The primary method of mineral analysis for USDA Handbook No. 8 is atomic absorption spectrophotometry (AAS). This is the most widely used method for determining mineral levels in biological samples (Harnley and Wolf, 1984). Attributes are instrument operation simplicity, excellent element specificity and sensitivity, capability of measuring about 70 elements, and moderate cost of basic instrumentation (Alvarez, 1984).

Atomic absorption deals with the excitation of the atom of interest and subsequent measurement of absorbed light energy. Energy in the form of heat within the atomization cell vaporizes and atomizes the sample, producing atoms in the ground state as well as some in the allowed excited energy states possessed by the element. The excited level is reached by absorption of resonance radiation of a specific wavelength from an external source. The measurement of absorbed radiation is proportional to ground state and excited state atomic concentrations and thus to the total number of atoms (or concentration) of the element of interest. The absorbed radiation is characteristic of the element (Alvarez, 1984).

The major drawback is that all of the spectrophotometric methods require sample destruction since the sample must be in solution prior to analysis. This process may introduce substances that interfere with the analysis of the element of interest. It is therefore necessary to use National Bureau of Standards (NBS) reference materials having similar inorganic matrices as test products alongside the sample. Other disadvantages of AAS have historically been its limited calibration range and its inability to analyze more than one element at a time (Harnley and Wolf, 1984).

## X-ray fluorescence

An alternate method of analysis of food samples not routinely used by the Nutrient

Data Bank is x-ray fluorescence (XRF). In XRF, a beam of primary x-rays is directed onto a sample being investigated, causing it to emit secondary (fluorescent) x-rays. These x-rays are characteristic of each element in the sample, and the total number of x-rays is directly proportional to the concentration of the element from which they were produced (Harnley and Wolf, 1984).

X-ray fluorescence has several major advantages over AAS. X-ray fluorescence may be applied directly to the biological sample to provide a non-destructive analysis of mineral levels. This is important since the labor-intensive steps of dissolving and diluting the sample in ash solution are eliminated by simply pressing the sample into a pellet and analyzing by the XRF system. Also, the most valuable food composition data are obtained by samples analyzed as consumed. The multielement analytical capabilities of XRF tremendously increase the information available from each sample without excessively increasing the individual sample cost. In addition, technician time per sample is reduced to 16-24 minutes per sample (Nielson and Kalkwarf, 1977). X-ray fluorescence instruments have traditionally been less available than those for AAS. However, the numbers of instruments have recently been increasing due to their cost compared to AAS instruments and to their potential to become an important approach to mineral assays.

Inductively coupled plasma

A third approach to mineral analysis of environmental samples relies on inductively coupled plasma (ICP) instrumentation. In this technique, radio frequency energy is coupled inductively to a stream of ionized argon gas, causing it to be heated to temperatures of up to 10,000$^0$C. When a sample is introduced into the very hot central region of the plasma, the atoms are excited and emit characteristic radiation. This radiation is measured by a scanning monochromator, which moves rapidly from wavelength to wavelength, locating the peak of the mineral being analyzed and integrating

the intensity for the desired time. The integrated readings are then converted to concentrations by computer (IL Plasma 200 ICP Spectrometer Manual). As with XRF, ICP is capable of multielement analysis. It can determine up to 70 elements, 24 elements in one sample within one minute after plasma torch equilibration. Detection limits are comparable to those for AAS for most elements. However, as with AAS, sample destruction is involved since the sample must be in solution prior to analysis. This may produce chemical interference with accurate mineral analysis. However, due to the long sample residence time and extremely high temperatures of the ICP discharge, chemical interferences are greatly reduced compared to AAS (IL Plasma 200 ICP Spectrometer Manual).

# MATERIALS AND METHODS

## FOOD COLLECTION AND PREPARATION: STUDY ONE

Twenty-one foods were selected from each of 3 to 5 different sources (fresh, processed, home stored, etc.) for a total of 96 food samples. The foods included beets, broccoli, cake, carrots, corn, enriched white bread, green beans, oatmeal, onions, peas, potato, rice, saltines, shrimp, sour cream, spinach, squash, tomato, waffles, whole wheat bread, and zucchini. Foods were prepared for consumption, blended in a glass blender equipped with a stainless steel cutter, weighed, and lyophilized. The lyophilized foods were ground with a mortar and pestle and stored in plastic one-pound containers until sampled for analyses. A 10g aliquot of each ground food sample was transported in individual plastic bags to Salt Lake City (Rogers and Associates Engineering Corp.) for XRF analysis. Canned food samples were drained of fluid before blending. Demineralized water was used whenever water was added for cooking using methods, cooking times, and temperatures recommended for vegetables. Onions were peeled and analyzed raw. Potatoes and winter squash were baked. Moisture in the stored, lyophilized food materials was determined whenever an aliquot was sampled for analysis by oven-drying a separate, equivalent aliquot for 2h in a forced-air oven at $105^{0}$C. Analyzed mineral values were reported on a dry-weight basis, with total moistures reported from the combined lyophilization and oven-drying losses (Nielson et al., 1988).

## FAST FOOD COLLECTION: STUDY TWO

The fast food outlets included common take-out establishments and included breakfast items, lunch items (hamburger, hot dog, taco, pizza, chicken, fish, sandwiches, salad bars, etc.), snack items (ice cream, cakes, candies, chips, etc.), and popular

beverages (soft drinks, coffee, dairy drinks, juices, tea, etc.). The sampling frame included a total of 228 samples. Sample collection of all perishable items involved immediately sealing the item in a heavy-gauge plastic bag, labeling, and freezing the item on dry ice for transport to a storage freezer. Labels included the item, vendor, location, date, and other comments relating to the item description.

Initial collection and freezing was done by Rogers and Associates Engineering Corp., Salt Lake City, UT. After transport to Utah State University, foods were stored in a deep freeze until ready for analytical preparation.

Foods were sampled as whole-serving menu items without separation into components. The items were removed from the deep freeze, weighed, and freeze dried for approximately 48h with the shelf temperature set at $40^0$C. Dried samples were weighed to determine moisture contents. Samples were then manually ground with a porcelain mortar and pestle until homogeneous and stored in plastic containers until ready for analysis. Ten-gram aliquots were weighed into sterile 6-oz Nasco plastic whirl-pak bags and sent to Rogers and Associates Engineering for independent mineral determination by XRF.

## DRY ASHING

Triplicate 2g aliquots of the ground homogeneous sample items were weighed (+/-0.0001g) and placed in porcelain crucibles for ashing. The samples were ashed in a muffle furnace at $550^0$C for at least 48h or until a white ash formed. To any samples that appeared grey or black, 3 drops of nitric acid and 3 drops of 30% hydrogen peroxide were added. The solution was dissolved on a hot plate and reashed at $550^0$C for another 24h. The ash was dissolved in 5 ml of a 1:1 mixture of 6N HCl and demineralized water over a low heat on a hot plate. The solution was then transferred to a 25-ml glass volumetric flask and diluted to volume with demineralized water. The prepared samples

were then stored in plastic 100-ml bottles until ready for analysis.

Between ashings, crucibles were cleaned by soaking in a 1:1 solution of 6N hydrochloric acid and demineralized water in a 4-l Pyrex beaker. The solution was heated over low heat to a slow boil for approximately 12h. Crucibles were individually removed and rinsed thoroughly, using demineralized water, and dried at room temperature.

## WET ASHING

Triplicate 0.5g aliquots of the ground homogeneous sample items were weighed (+/-0.0001g) and placed in 50-ml glass Erlenmyer flasks. To each flask was added 20-30 ml of 70% nitric acid and 2-3 boiling beads. Flasks were placed on a hot plate at low heat for 3-4 days. Nitric acid was added as needed to keep samples wet. To any sample that was not clear after 48h, 0.5 ml 6N HCl per day was added. The clear solution was then transferred to a 25 ml volumetric flask and brought to volume with demineralized water. Prepared samples were then transferred to 100-ml plastic-capped bottles until ready for analysis.

Between ashings, flasks were cleaned by soaking in a 1:2 mixture of 70% nitric acid to demineralized water for approximately 12h. Flasks were removed and rinsed thoroughly with demineralized water and allowed to dry at room temperature.

## ATOMIC ABSORPTION ANALYSIS

Each mineral-ash solution was analyzed in triplicate by AAS (Instrumentation Laboratories Model 457 dual-beam atomic-absorption spectrophotometer). Dry-ashed samples were used to analyze for manganese (Mn), copper (Cu), and zinc (Zn). Wet-ashed samples were used to analyze for iron (Fe) since dry ashing is known to cause Fe losses during oven heating (Clegg et al., 1981). Manganese, Fe, Cu, and Zn standard curves were obtained using stock solutions containing 1000 ppm of the mineral diluted to 4 increments of concentrations covering the working range of the mineral. Flame

atomization was used with an air-acetylene flame and the following wavelengths: Mn, 274 nm; Fe, 245 nm; Cu, 319 nm; and Zn, 213 nm.

The AAS procedure was verified by repeated analysis with NBS standard materials with each set of food samples. The means of these determinations in rice flour (SRM-1568) and wheat flour (SRM-1567) were 19.8 and 7.6 ug/g Mn (20.1+/-0.4 and 8.5+/-0.5 ug/g certified), 8.4 and 17.2 ug/g Fe (8.7+/-0.6 and 18.3 +/-1.0 ug/g certified), 2.5 and 2.8 ug/g Cu (2.2+/-0.3 and 2.0+/-0.3 ug/g certified), and 19.6 and 10.5 ug/g Zn (19.4 +/-1.0 and 10.6+/-1.0 ug/g certified).

## X-RAY FLUORESCENCE ANALYSIS

Multielement mineral determinations were performed on lyophilized sample aliquots sent to Salt Lake City independent of AAS determinations. The samples were analyzed directly by weighing 0.5g aliquots of the dry powder into a 3.2 cm. diameter hardened steel die and pressing self-supporting sample pellets at 2300 $kg/cm^2$ pressure. Four replicate pellets were prepared from each standard reference material, and 3 replicate pellets were prepared from each food. Four analyses were performed on each of the 4 NBS standard reference material pellets, and one analysis was performed on each of the food pellets. Each analysis consisted of collection of 4 separate XRF spectra under vacuum using gadolinium, silver, and germanium (Ge) secondary excitation and 5 kV direct excitation (30, 20, and 10 minutes, respectively, with a Kevex Model 700 spectrometer system). Only the Ge spectrum was used to obtain the Mn, Fe, Cu, and Zn x-ray intensities. The other 3 spectra provided data on additional elements, which were all used in the CEMAS calculation. The CEMAS approach to quantitation relies on fundamental parameters of x-ray physics for quantitation of x-ray intensities. Mineral concentrations were stored directly on disk for subsequent statistical analysis (Nielson et al., 1988).

## INDUCTIVELY COUPLED PLASMA ANALYSIS

An Instrumentation Laboratory Plasma 100/200 ICP Emission Spectrometer was used in the determination of Cd in fast foods since the limit of determination by XRF was too high to generate data of value. Typical foods contain 0.05 ug/g Cd, and the limit of determination by XRF is 5 ug/g. Samples were wet ashed and analyzed with the assistance of a technician trained in mineral analysis by ICP. Calibration curves for a working range of 0.05 - 1.0 ug/g were obtained after each set of 4 samples was analyzed. Inductively coupled plasma procedure was verified by analysis of NBS standard reference materials with each set of food samples. With a lower limit of determination of 0.05 ug/g Cd, ICP showed values for non-fat milk powder (SRM-1549), wheat flour (SRM-1567), and rice flour (SRM-1568) to be below detection limits. Certified values are 0.0005 +/- 0.0002 ug/g for non-fat milk powder, 0.032 +/- 0.007 ug/g for wheat flour, and 0.029 +/- 0.004 ug/g for rice flour.

## STATISTICAL PROCEDURES

Mineral determinations by XRF were compared to 9 NBS standard reference materials in Study One, including powdered milk (SRM - 1549), oyster tissue (SRM - 1566), wheat flour (SRM - 1567), rice flour (SRM - 1568), orchard leaves (SRM - 1571), citrus leaves (SRM - 1572), tomato leaves (SRM - 1573), pine needles (SRM - 1575), and bovine liver (SRM - 1577a). Mean differences, standard deviations of the means, and mean relative bias (mean difference/NBS value x 100) were obtained for Mn, Fe, Cu, and Zn (Nielson et al., 1988).

Values obtained by AAS were compared to independent determinations by XRF for Mn, Fe, Cu, and Zn in the 96 food samples under investigation. Means, standard deviations, and mean differences were calculated. F tests were performed at a 95% confidence interval to determine significance of difference between means obtained by the two methods. Scatter plots were also obtained for these foods to show linear

comparisons between methods.

Detailed quantitative information was then obtained by XRF and AAS on Mn, Fe, Cu and Zn contents for the 228 fast-food items. Means, standard deviations, correlation coefficients ($R^2$), slopes, and intercepts were calculated.

X-ray fluorescence was then used to determine toxic mineral concentrations in NBS standard reference materials. Selenium was determined in oyster tissue (SRM-1566) by 16 determinations and in wheat flour (SRM-1567) by 3 determinations. Similarly, As was determined in citrus leaves (SRM-1572), oyster tissue (SRM-1566), and orchard leaves (SRM-1571) by 16 determinations. Aluminum was determined in pine needles (SRM-1575) and tomato leaves (SRM-1573) by an average of 16 determinations. In the analysis of fast foods by XRF for Mn, Cu, Zn, and Fe, simultaneous data were generated for 43 other minerals including Cd, Al, Se and As. Bar graphs were generated by Harvard Graphics to indicate regions of average daily intake, maximum dietary exposure from fast foods, and toxic levels of mineral. A summarized table of average toxic mineral levels in each type of fast food was then recorded.

# RESULTS AND DISCUSSION

## FIRST OBJECTIVE

One of the most powerful and convincing ways to establish the reliability of an analytical technique is to use quality control standards analyzed by another laboratory. External standards in the form of Certified Reference Materials provide the most comprehensive means for establishing analytical competence and accuracy (Parr, 1985). The National Bureau of Standards provides reference materials intended primarily for use in calibrating instrumentation and evaluating the reliability of analytical methods from the determination of major, minor, and trace elements. The certified values for the element of interest are based on results obtained by reference methods of known accuracy and performed by 2 or more analysts or, alternately from results obtained by 2 or more independent, reliable analytical methods (Rasberry, 1985).

The comparison of data obtained by XRF against NBS-certified concentrations in Table 1 indicates agreement between methods that is consistently within the quoted NBS uncertainties. Student's t test indicates no significant difference between NBS-certified values and values obtained by XRF. The mean of the differences in the analysis ranged from 0.7 ug/g for Cu to 3.4 ug/g for Mn; or, on a relative basis, it ranged from 0.9% for Fe to 2.3% for Mn. These agreements are the main basis for validating the XRF method.

To further establish the validity of XRF, scatter plots were made to compare data from 96 food samples against the most conventional means of analysis, atomic absorption spectroscopy. The comparisons of XRF with AAS yielded correlation coefficients of 0.94 for Mn (Fig. 1), 0.99 for Fe (Fig. 2), 0.93 for Cu (Fig. 3), and 0.91 for Zn (Fig. 4). The comparisons were linear for all 4 elements over the entire ranges of data (1 - 100 ug/g Mn, 3 - 1600 ug/g Fe, 0.5 - 50 ug/g Cu, 2 - 140 ug/g Zn).

Table 1 - Comparison of x-ray flourescence (XRF) mineral measurements with National Bureau of Standards (NBS)-certified concentrations

| | Mineral Concentration, ug/g | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Manganese | | | Iron | | | Copper | | | Zinc | | |
| Sample | XRF[a] | NBS[b] | diff | XRF | NBS | diff | XRF | NBS | diff | XRF | NBS | diff |
| SRM 1549 | | [d] | | | | | | | | 45.2 | 46.1 | -0.9 |
| SRM 1566 | 17.2 | 17.5 | -0.3 | 198 | 195 | 3 | 62.8 | 63.0 | -0.2 | 867 | 852 | 15.0 |
| SRM 1567 | 8.5 | 8.5 | 0.0 | 19.3 | 18.3 | 1 | 2.0 | 2.0 | 0.0 | 10.1 | 10.6 | -0.5 |
| SRM 1568 | 20.6 | 20.1 | 0.5 | 8.2 | 8.7 | -0.5 | 20. | 2.2 | -0.2 | 19.3 | 19.4 | -0.1 |
| SRM 1571 | 90.2 | 91.0 | -0.8 | 291 | 300 | -9 | 12.6 | 12.0 | 0.6 | 23.9 | 25.0 | -1.1 |
| SRM 1572 | 23.2 | 23.0 | 0.2 | 94 | 90 | 4 | 16.5 | 16.5 | 0.0 | 29.2 | 29.0 | 0.2 |
| SRM 1573 | 250 | 238 | 12 | 690 | 690 | 0 | 11.8 | 11.0 | 0.8 | 66.0 | 62.0 | 4.0 |
| SRM 1575 | 691 | 675 | 16 | 208 | 200 | 8 | 2.9 | 3.0 | -0.1 | 60.0 | [e] | |
| SRM 1577 | 9.9 | 9.9 | 0.0 | 202 | 194 | 8 | 163 | 158 | 5.0 | 125 | 123 | 2.0 |
| students's t test | | | NS[f] | | | NS | | | NS | | | NS |
| mean diff | | | 3.4 | | | 1.6 | | | 0.7 | | | 2.3 |
| st dev of mean | | | 2.3 | | | 1.7 | | | 0.6 | | | 1.9 |
| mean rel. bias %[g] | | | 2.3 | | | 0.9 | | | 2.2 | | | 1.6 |

[a] XRF values are means of 16 determinations

[b] NBS-certified concentrations

[c] Difference between XRF mean and NBS value

[d] Some or all determinations were below XRF detection limits

[e] Zn not certified by NBS

[f] No significant difference at 95% confidence level

[g] (Mean difference/NBS value) x 100

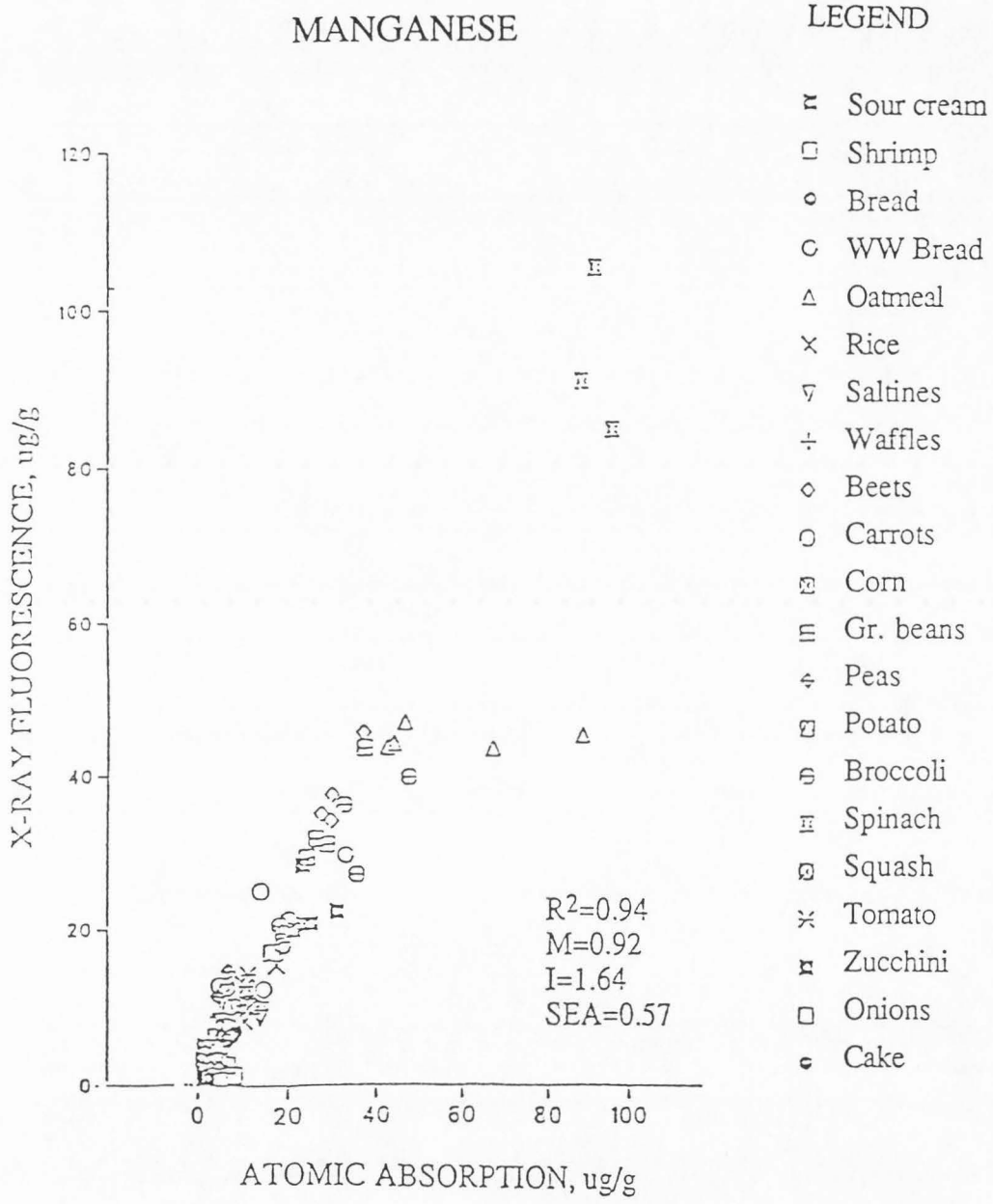


Figure 1. Scatter plot comparison of manganese measurements by x-ray fluorescence (XRF) vs. atomic absorption (AAS) in 96 food samples. Statistical analysis indicate correlation coefficient, slope, intercept, and standard error of analysis between methods.

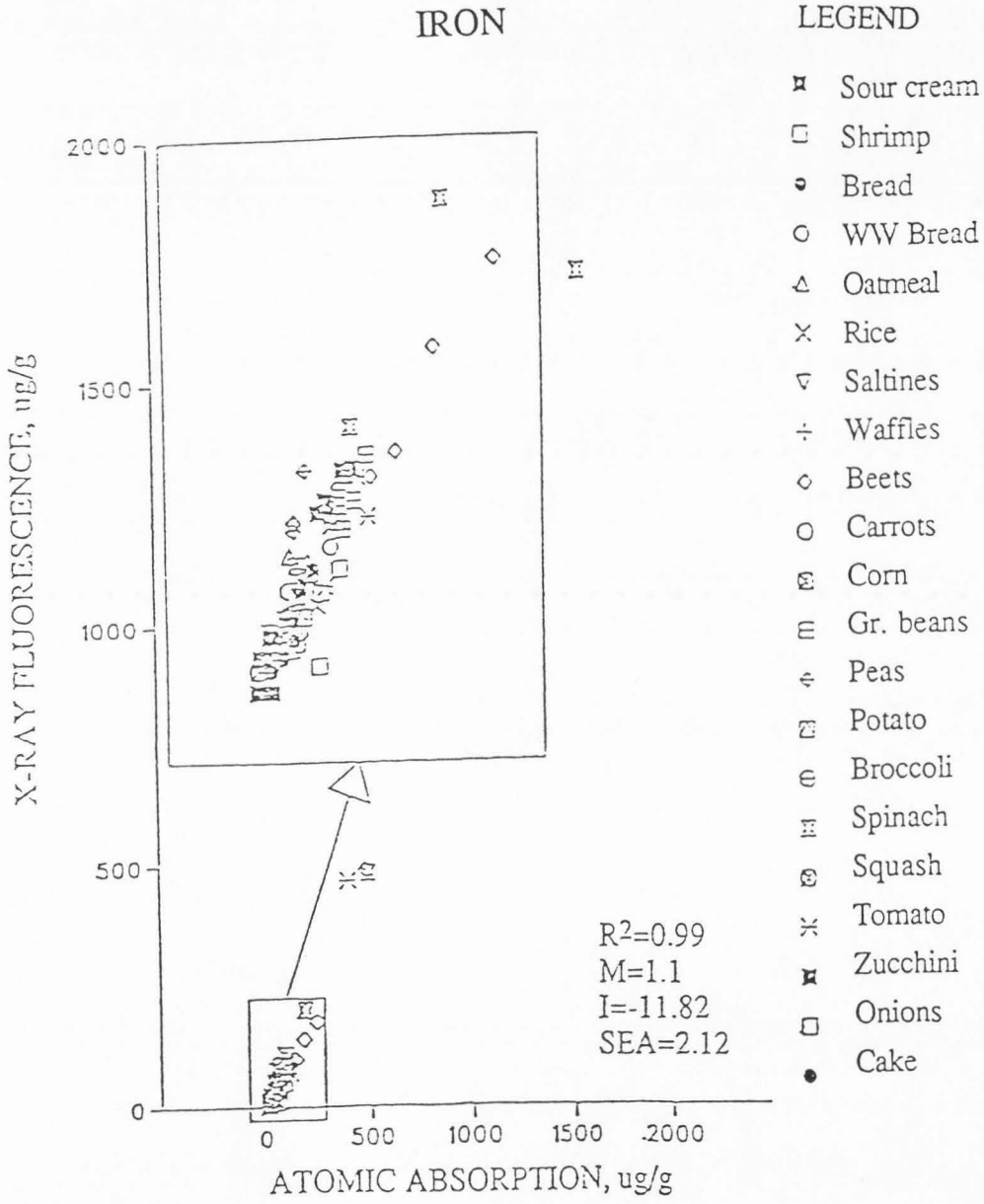


Figure 2. Scatter plot comparison of iron measurements by x-ray fluorescence (XRF) vs. atomic absorption (AAS) in 96 food samples. Statistical analysis indicate correlation coefficient, slope, intercept, and standard error of analysis between methods.

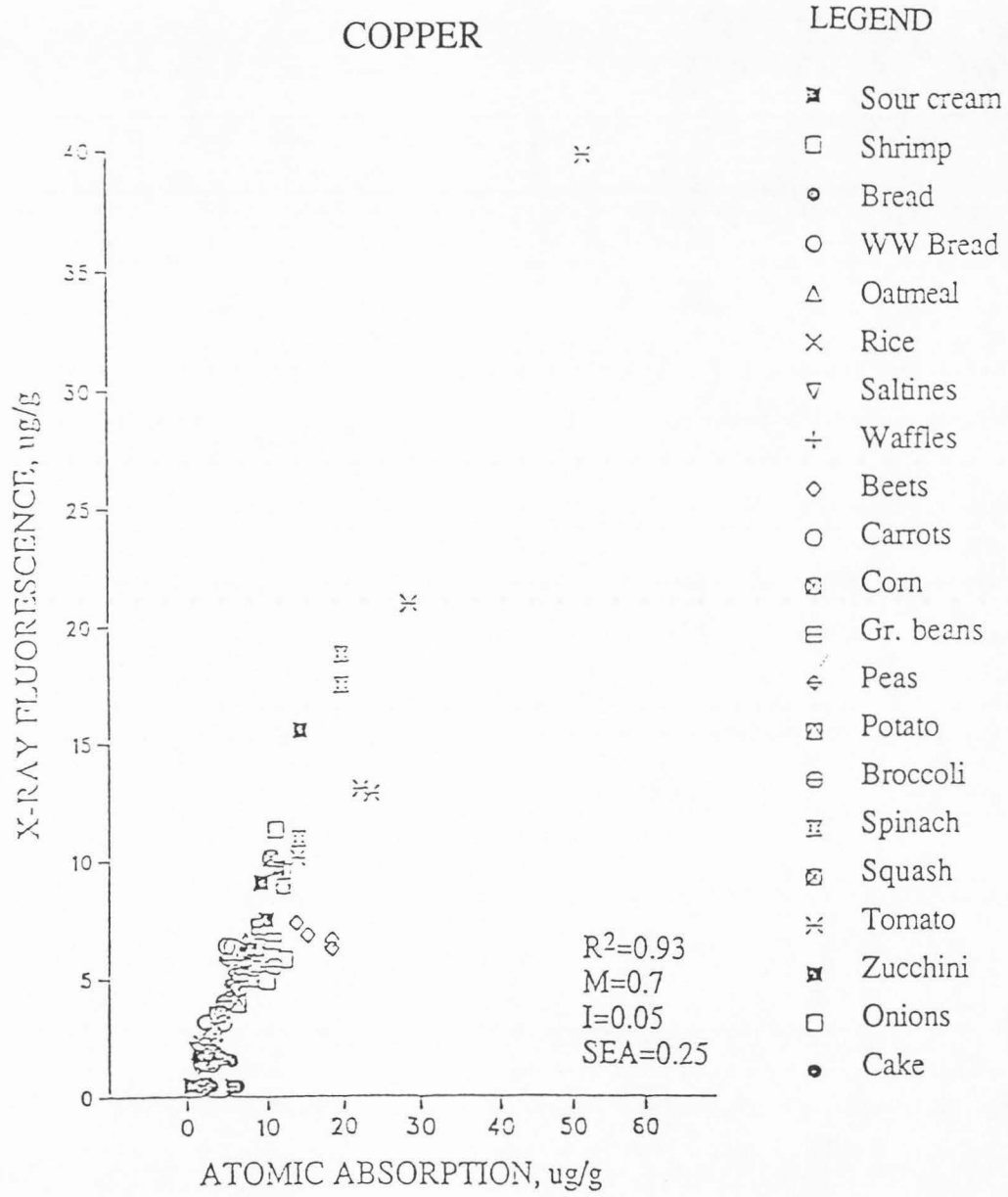


Figure 3. Scatter plot comparison of copper measurements by x-ray fluorescence (XRF) vs. atomic absorption (AAS) in 96 food samples. Statistical analysis indicate correlation coefficient, slope, intercept, and standard error of analysis between methods.

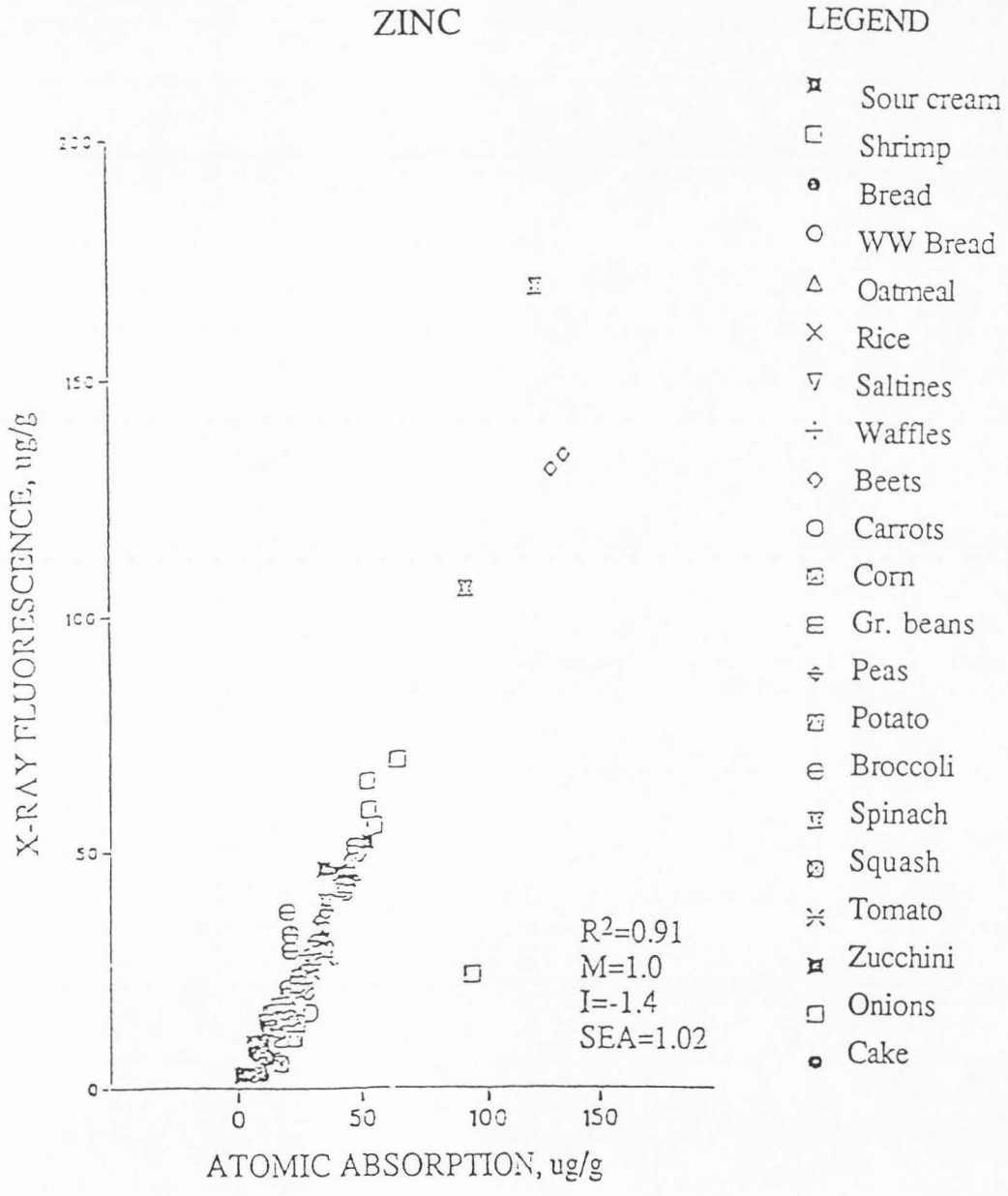


Figure 4. Scatter plot comparison of zinc measurements by x-ray fluorescence (XRF) vs. atomic absorption (AAS) in 96 food samples. Statistical analysis indicate correlation coefficient, slope, intercept, and standard error of analysis between methods.

The scatter plots yielded slopes that were within 10% of unity for Mn, Fe and Zn, with a lower slope (0.7) for Cu suggesting a bias in the copper data.

A statistical comparison of XRF and AAS data is presented in Table 2, with the mean mineral concentrations in each of 96 food samples analyzed. Average overall biases were 0.4 ug/g for Mn, -1.9 ug/g for Fe, -2.1 ug/g for Cu, and -0.8 ug/g for Zn. The sample standard deviations give an estimate of intersample variability. F tests using an alpha of .05 revealed no significant differences among the 4 minerals measured by the 2 methods. These statistical comparisons are a second means of validating XRF since results were obtained by 2 independent analytical methods.

Table 2 - Summary of atomic absorption spectrophotometry (AA) and x-ray flourescence values for manganese, iron, copper, and zinc in 96 food samples (ug/g in dry matter).

| | manganese | | | iron | | | copper | | | zinc | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | AA[a] | XRF[b] | diff[c] | AA | XRF | diff | AA | XRF | diff | AA | XRF | diff |
| mean | 17.7 | 18.2 | 0.4 | 88.7 | 86.8 | -1.9 | 8.0 | 6.0 | -2.1 | 27.3 | 26.6 | -0.8 |
| std dev | 18.7 | 18.2 | 4.6 | 126 | 136 | 16.1 | 6.2 | 4.4 | 2.5 | 20.2 | 23.5 | 5.6 |
| $F_{1,95}$[d] | | | NS | | | NS | | | NS | | | NS |

[a]Atomic absorption values are an average of determinations for each of the 96 food samples.
Each determination is a mean of triplicate analytical values.
[b]X-ray flourescence values are an average of determinations for each of the 96 food samples.
Each determination is a mean of triplicate analytical values.
[c]Average overall difference of values determined by the two methods.
[d]F test indication of significant difference between methods. NS=no significant difference.

Additional comparative values between XRF and AAS were obtained for the 228 fast foods under investigation. Mean values for Mn, Fe, Cu, and Zn are indicated in Appendix 1, Table 7. Correlation coefficients between methods ranged from 0.97 for Fe

and Zn to 0.91 for Cu. The correlation coefficient for Mn was 0.94. The quantitative information presented in Table 7 is important since a complicating factor in toxic element research is interaction. The nutritional or toxic physiology of the trace element is rarely confined to a single element in isolation from others. This can affect minimum requirements and influence maximum tolerances. Tables 3, 4, and 5 show comparisons of XRF mineral measurements with NBS certified values for Se, As, and Al, respectively, again indicating agreement consistent within NBS uncertainties.

As indicated above, XRF has been proven to be a reliable method of multielement analysis of biological materials. High-resolution XRF instruments are now available at prices as low as $30,000 - $40,000, suggesting an initial investment comparable to that for AAS instruments. Consideration should also be given to the unique advantage XRF offers of avoiding sample dissolution, matrix calibrations, and related labor-intensive aspects of mineral analyses commonly encountered in AAS and ICP procedures.

## SECOND OBJECTIVE

X-ray fluorescence was validated as a method in Study One, then used to determine levels of minerals of toxicological interest in fast foods in Study Two. Analytical challenges to the investigator were compounded by the very low concentrations usually involved. The only characteristic that trace elements have in common is that they normally occur in biological samples in very low concentrations.

The increasing use of processed fast foods has resulted in a need for increased toxicological awareness and understanding by nutritionists and other professionals concerned with food safety. Analysis of foods as eaten provides the most meaningful data on the composition of foods. Much of today's analytical data were obtained by analyses of raw foods or components of whole food items. Most chemical compounds are not uniformly distributed throughout the food system. Specific compounds are usually

Table 3 - Comparison of x-ray flourescence (XRF) selenium measurements with National Bureau of Standards (NBS)-certified concentrations

| | | Selenium, ug/g | |
|---|---|---|---|
| Sample | No. obs. | XRF[a] | NBS[b] |
| Oyster tissue (SRM 1566) | 16 | 2.11 | 2.1<br>0.63 |
| Wheat flour (SRM 1567) | 3 | 1.79<br>0.52 | 1.1<br>0.2 |

[a]XRF means and standard deviations

[b]NBS means and standard deviations

Table 4 - Comparison of x-ray flourescence (XRF) arsenic measurements with National Bureau of Standards (NBS)-certified concentrations

| | | Arsenic, ug/g | |
|---|---|---|---|
| Sample | No. obs. | XRF[a] | NBS[b] |
| Citrus leaves (SRM 1572) | 16 | 3.07 | 3.10<br>0.99 |
| Oyster tissue (SRM 1566) | 16 | 12.8 | 13.4<br>1.04 |
| Orchard leaves (SRM 1571) | 16 | 10.4 | 14.0<br>1.2 |

[a]XRF means and standard deviations

[b]NBS means and standard deviations

concentrated in a few foods and occur at very low concentrations in others.

In these regards, XRF has two very important advantages over AAS as an analytical tool for mineral analysis. Information regarding quantitative levels of toxic minerals in foods is greatly lacking and is questionable in accuracy as determined by AAS since sample dissolution by ashing techniques may cause chemical interferences and thus inadequate data on minerals of toxicological importance. X-ray fluorescence by non-destructive analysis can be used to directly analyze foods for accurate mineral content. The second advantage is that XRF, through its multielement capabilities, can be used to screen for high levels of minerals without relying on a preconceived hypothesis to determine the critical elements of determination.

Eating habits in the U.S. have changed dramatically since the turn of the century. Changes in per capita consumption rates of specific food categories could result in a change in the total intake of trace minerals. With the awareness that specific human diseases are induced by high levels of certain trace minerals, it becomes of major public health importance to know not only the mineral level but the maximum level of the mineral

Table 5 - Comparison of x-ray fluorescence (XRF) aluminum measurements with National Bureau of Standards (NBS)-certified concentrations

| | | Aluminum, ug/g | |
|---|---|---|---|
| Sample | No. obs. | XRF[a] | NBS[b] |
| Tomato leaves (SRM 1573) | 16 | 1037 | 1200[c]<br>194 |
| Pine needles (SRM 1575) | 16 | 600<br>40.7 | 545<br>30 |

[a]XRF means and standard deviations
[b]NBS means and standard deviations
[c]Non-certified value

exposure a person can have without risk of disease. Also, the concept of a critical concentration of metal has very important implications with regards to establishing maximum levels that human populations may be exposed to with some margin of safety.

Maximum dietary intake estimates

Excessive exposure to trace metals has been associated with various diseases in humans, and several potential at-risk groups have been identified that would benefit from knowledge of levels of those minerals found in foods that are causally implicated in disease associated with high levels of exposure. Before the dietary intake of any chemical can be estimated, it is essential to know the weight of the diet consumed and the concentration of the chemical in the diet as well as the daily percentage of the intake of that particular food group or item.

An extreme consumption may be defined once the average weight of the adult diet is known and a maximum intake of the food group is estimated. Some individuals may have consistently higher intakes of toxic minerals because they have atypical dietary habits. Food frequency questionaires have indicated a high of 40% of total diet comprised of fast food items for some segments of the population (Nielson, 1985). A hypothetical diet may be designed for a person who is considered to have the highest intake of the food group and therefore a high intake of food contaminants. The concentrations of the contaminant in the diet may be determined by the analysis of individuals foods or by the analyses of food groups comprising like foods, e.g., processed fast foods.

The weight of adult diets used to estimate contaminant intakes in various countries usually lies between 1500 and 3500g; this refers to the weight of food and beverages consumed daily (Sherlock, 1984). Usually intake is estimated assuming a high ingestion of the food and multiplying this by maximum dietary exposure of the mineral in the food. The accuracy of the analyses of the diets and the limits of determination of the analytical methodology are critical if intakes are to be reliably determined.

Cadmium exposure

If the limit of determination for Cd were 0.05 ug/g and findings at or below this level were considered to be 0.05 ug/g for the purpose of estimation, then the estimated range of daily intake from fast foods might be 0.05 ug/g (maximum dietary exposure) x 1500g (minimum weight of adult diet) x .40 (maximum percentage of diet composed of fast foods) = 30ug minimum dietary exposure from diets composed of 40% fast foods. The maximum dietary exposure would then be calculated as 0.05 ug/g (maximum dietary exposure) x 3500g (maximum weight of adult diet) x .40 (maximum percentage of diet composed of fast foods) = 70ug maximum dietary exposure from diets composed of 40% fast foods. The lower limit of determination for Cd by ICP is 0.05 ug/g. The WHO/FAO Joint Expert Committee on Food Additives has recommended a provisional daily intake of 60-70 ug Cd/day (Sherlock, 1984). The results for fast foods show no findings of values over 0.05 ug/g for Cd. Therefore, based on average daily intake (ADI) and maximum provisional limits, Cd appears to be without risk as a component in fast foods. The abbreviation ADI used in Fig. 5 indicates the average intake of Cd in the U.S and should not be confused with allowable daily intake, also abbreviated ADI, which is commonly used in toxicology literature. As Fig. 5 indicates, 200-300 ug Cd/day would be required to induce toxicity. There is, however, a clear need for monitoring of Cd in food since Cd is a toxic mineral at high levels and is related to several diseases including severe renal tubular damage.

Aluminum exposure

For Al, recent evidence points to consumption of 20-30 mg/day in the normal diet (Greger, 1988b). Using the estimate of 1500-3500g as the weight of adult diets per day, we would calculate average foods to contain 5.7-20 ug/g Al. Much higher values were obtained by XRF for Al in fast foods, as indicated in Appendix 1, Table 7. These higher values might be anticipated since aluminum is a major component of baking powder

# CADMIUM

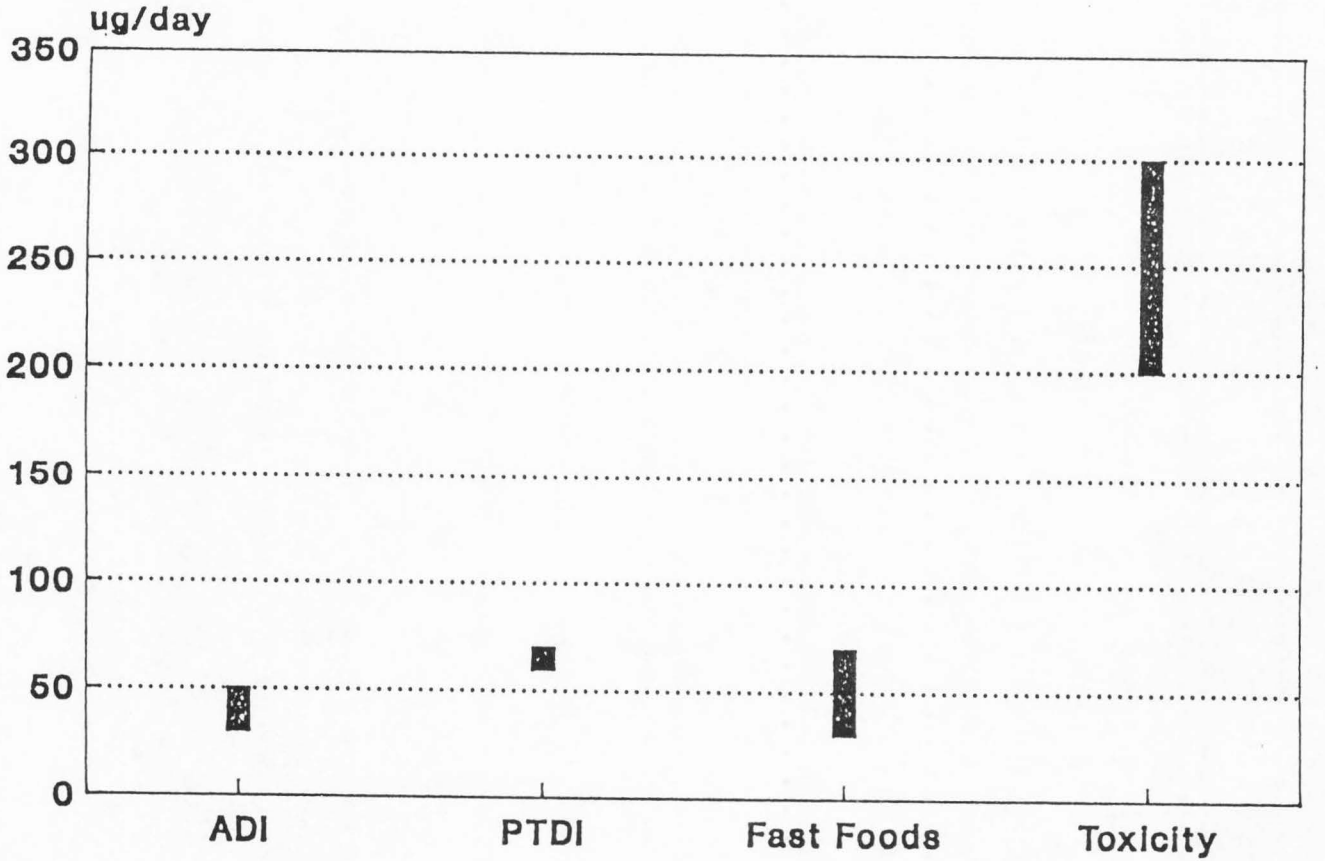


Figure 5. Maximum levels of cadmium exposure in a diet composed of 40% fast foods/day as compared to average daily intake (ADI), provisional tolerable daily intake (PTDI), and levels of toxicity.

(23,000 ug/g), and foods such as pizza, hamburger, burritos, donuts, and rolls are prepared with baking powder as an ingredient (Table 6) (Greger, 1988a). Baked goods prepared with chemical leavening agents and certain processed cheeses are the foods that contribute the greatest amounts of Al to diets of Americans (Greger, 1988b). Other possible sources might be in Al cookware and storage bins typically used in fast food restaurants. Foods stored or cooked in Al pans, trays, or foil accumuate some Al (Greger, 1988b). The variation of Al content in similar foods observed in Table 7 may be explained by the variable degree of storage in Al bins or by the variation in amounts of baking powder used as a cooking ingredient.

To calculate maximum Al exposure from fast foods, we would use the formula previously used for Cd. The highest level of Al detected was 583 ug/g in the 228 fast foods investigated. Other values are indicated in Appendix 1, Table 7. Using a range of 1500-3500g as the weight of adult diets and 40% as the maximum intake of diet consumed as fast foods, we would estimate a range of 320-816mg as the maximum daily exposure of Al consumed from fast foods.

This range of exposure is well above the range of consumption in the normal diet (Fig. 6). Systemic toxicity would be expected in the range of 2000-3000 mg Al/day. With the ever-increasing knowledge of involvement of Al in dialysis encephalopathy and possibly Alzheimer's progression, those at-risk patients should be wary of this high source of Al and favor low Al foods such as grains, fruits, and vegetables (Greger, 1988a). Sensitive individuals, i.e. those on dialysis treatment for impaired kidney function, are already receiving 2000-4000 mg Al/dialysis treatment. Other at-risk groups would include ulcer patients who are receiving 840-5000mg Al and 126-728mg Al daily doses in antacids and buffered analgesics (Greger, 1988b). These patients may suffer long-term consequences from high dietary intakes of Al since they are chronically receiving potentially toxic daily doses pharmacologically.

Table 6 - Summary of toxic mineral levels in each fast food type

| Food type | No. obs. | Ave. size (g) | Se ug/g | As ug/g | Al ug/g | Cd ug/g |
|---|---|---|---|---|---|---|
| Baked beans | 2 | 361 | nd[a] | nd[b] | nd[c] | nd[d] |
| Chili | 2 | 230 | nd | nd | 184.5[e](1)[f] | nd |
| Chef salad | 8 | 327 | nd | nd | 439.2 (1) | nd |
| Clam chowder | 2 | 285 | nd | 0.59 (2) | 272.0 (2) | nd |
| Garden salad | 5 | 245 | nd | nd | 228.0 (1) | nd |
| Potato salad | 5 | 316 | nd | nd | nd | nd |
| Cheese pizza | 8 | 220 | 0.89 (3) | nd | 162.4 (4) | nd |
| Combo pizza | 15 | 276 | 0.79 (4) | nd | 139.4 (13) | nd |
| Bean burrito | 4 | 221 | nd | nd | 150.8 (1) | nd |
| Combo burrito | 8 | 237 | nd | nd | 212.0 (6) | nd |
| Nachos | 5 | 166 | nd | nd | nd | nd |
| Taco | 13 | 87 | nd | nd | 265.5 (1) | nd |
| Chicken nuggets | 7 | 114 | nd | nd | 126.0 (1) | nd |
| Chicken&biscuit | 3 | 246 | nd | nd | 299.8 (2) | nd |
| Fish fillet | 5 | 124 | 0.44 (2) | 3.33 (5) | 116.9 (2) | nd |
| Fish&chips | 1 | 87 | nd | 0.95 (1) | nd | nd |
| Orange juice | 4 | 188 | nd | nd | nd | nd |
| Strawberry shake | 4 | 345 | nd | nd | 174.1 (1) | nd |
| Onion rings | 4 | 92 | nd | nd | nd | nd |
| Hash browns | 2 | 52 | nd | nd | nd | nd |
| French fries | 5 | 92 | nd | nd | nd | nd |
| Apple pie | 4 | 100 | nd | nd | 177.5 (1) | nd |
| Choc. chip cookie | 5 | 65 | nd | nd | 133.4 (2) | nd |
| Choc. sundae | 3 | 179 | nd | nd | nd | nd |
| Ice cream cone | 4 | 123 | nd | nd | nd | nd |
| Donut | 11 | 112 | nd | nd | 155.5 (2) | nd |
| Cinnamon roll | 7 | 161 | 0.44 (1) | nd | 218.7 (4) | nd |
| Country breakfast | 3 | 297 | 0.46 (1) | nd | 346.5 (1) | nd |
| Egg McMuffin | 1 | 143 | 0.66 (1) | nd | nd | nd |
| Sausage biscuit | 4 | 160 | 0.71 (2) | nd | 443.0 (2) | nd |
| Hamburger | 14 | 109 | nd | nd | 164.6 (13) | nd |
| Deluxe burger | 15 | 242 | nd | nd | 138.8 (10) | nd |
| Hot dog | 6 | 108 | nd | nd | 145.5 (1) | nd |
| Classic dog | 4 | 176 | nd | nd | 240.7 (1) | nd |
| Roast beef sand. | 10 | 172 | nd | nd | 196.0 (2) | nd |
| Club sandwich | 1 | 236 | 0.45 (2) | nd | 189.3 (4) | nd |
| Chicken sandwich | 5 | 197 | nd | nd | 185.6 (4) | nd |
| Fish sandwich | 7 | 175 | 0.51 (2) | 1.43 (5) | 139.2 (3) | nd |

[a]All measurements below detection limit of 0.4 ug/g Se by x-ray flourescence

[b]All measurements below detection limit of 0.4 ug/g As by x-ray flourescence

[c]All measurements below detection limit of 90 ug/g Al by x-ray flourescence

[d]All measurements below detection limit of 0.50 ug/g Cd by inductively coupled plasma

[e]Average of measurements above detection limit

[f]Number of detectable measurements from total observations

# ALUMINUM

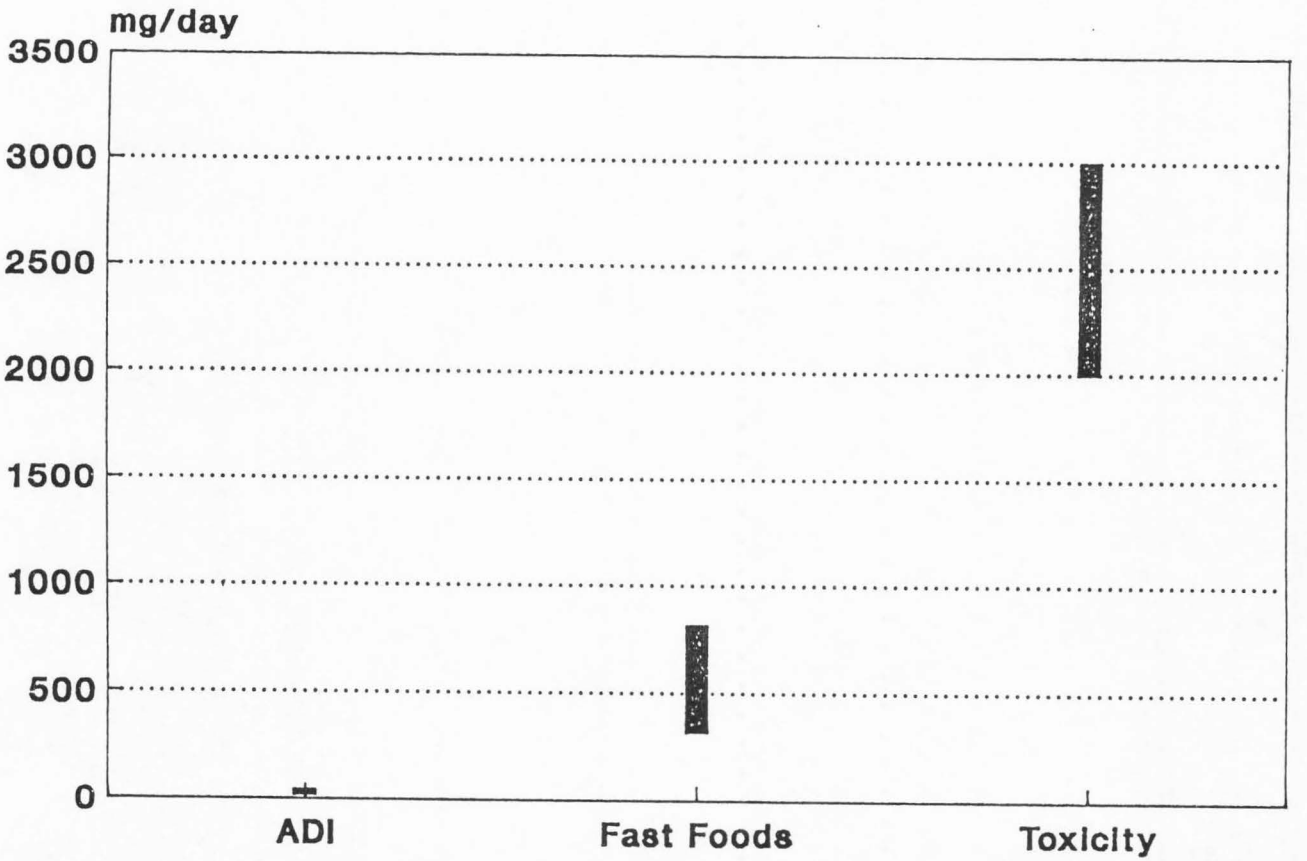


Figure 6. Maximum levels of aluminum exposure in a diet composed of 40% fast foods/day as compared to average daily intake (ADI) and levels of toxicity.

Selenium exposure

Selenium is the only mineral discussed that has a known physiological role and thus an RDA. The RDA for Se has been established as 50-200 ug/day (Lane et al., 1983). The human dietary requirement for Se is in the range of 0.1 to 0.2 ug/g (Levander, 1975). Of the 228 fast food items studied, pizza showed the maximum Se content, particularly those purchased from Peter Piper (Table 7). Possible explanations for variation in Se content of similar foods may be in the source of flour used in cooking since grains and cereals contain variable amounts of Se depending on where they are grown (Lo and Sandi, 1980). Appendix 1,Table 7 shows values ranging from 0.43 to 0.91 ug/g Se. All other foods investigated may be assumed to contain between 0.0 and 0.40 ug/g Se (limit of determination by XRF).

The estimated daily intake range from fast foods is calculated as 0.91ug/g x 1500g x 40% = 546 ug Se/day as the lower limit and 0.91ug/g x 3500g x 40% = 1274ug as the upper limit of exposure. It is doubtful that fast foods as a whole could be considered harmful from the standpoint of Se content since such a small range of the fast food items fall above the low detection limit. As Fig. 7 indicates, mild toxicity would not be expected until levels of 2000-3000 ug Se/day were regularly consumed in the diet. It is even possible to include the recommendation that those foods with high Se content be included regularly in the diet based on experimental data that indicate lower cancer mortality rates with increased Se intakes over an entire lifetime (Miyamato et al., 1987).

Arsenic exposure

Average daily intake of As is estimated as 20-30 ug/day, with most foods containing <0.3 ug/g (Anke, 1986). Of the fast food items studied, maximum As values were consistently found to be above the detection limit in fish products (Table 6). This was expected since foods of marine origin are much richer in As than other foods (Anke, 1986). Values ranged from 0.51 to 7.32 ug/g as shown in Appendix 1, Table 7. All

# SELENIUM

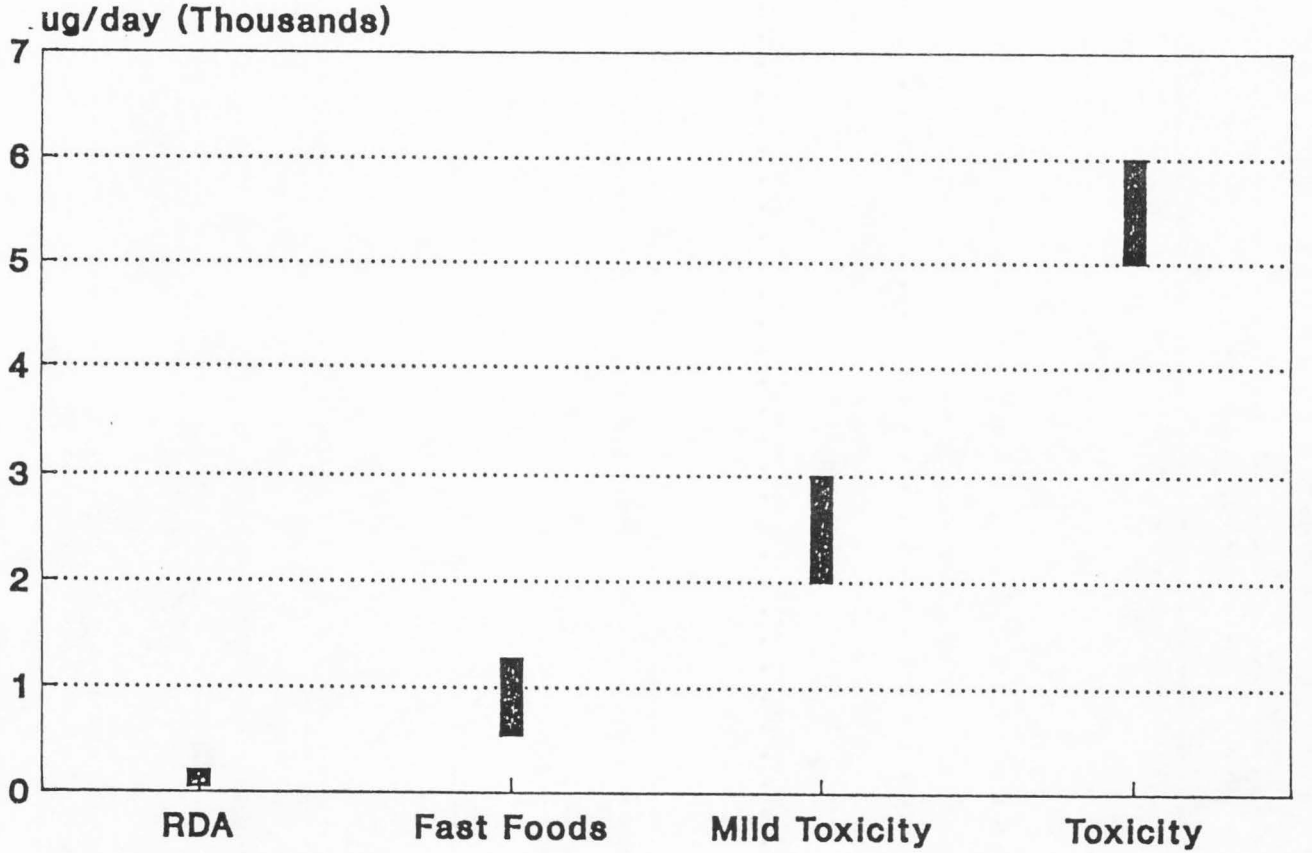


Figure 7. Maximum levels of selenium exposure in a diet composed of 40% fast foods/day as compared to recommended dietary allowance (RDA) and levels of toxicity.

others fell below the detection limit of 0.40 ug/g by XRF.

At the maximum level of 7.32 ug/g and average daily food intake of 1500g, we would calculate a daily intake of 7.32 ug/g x 1500g x .40 = 4.4mg as the minimum level of As exposure and 7.32 ug/g x 3500g x .40 = 10.2mg as the maximum level of As exposure from fast foods. This level of As is unlikely since the majority of the foods analyzed fell into the 0.0 to 0.40 ug/g range, and only the limited category of fish products ranged above detection limits by XRF. Furthermore, toxicity is unlikely since seafood contains As in the form of organoarsenic compounds, which are nontoxic and are not metabolized to toxic forms in the human body (Anke, 1986). Fig. 8 indicates that levels of 18-42 mg As/day would be required for mild toxicity, and levels of 70-180mg would be required for acute toxicity (Goyer, 1986). Those people at high risk of developing skin cancer might consider reducing As exposure via the diet since increasing evidence points to As as a human carcinogen, particularly of the skin.

Possible toxicity from other minerals

It should be mentioned that the minerals, Fe, Mn, Cu, and Zn could be considered toxic at extreme levels of exposure, although they are not accorded priority as toxic minerals by the FDA (Jelinek and Corneliussen, 1977). The main purpose of measuring these levels in fast foods was to use the data as a comparative tool between AAS and XRF to validate XRF as a method of mineral analysis.

Acute Fe intoxication is rare. Doses of 5g have been shown to result in metabolic alterations affecting multiple organ systems. The lethal dose of ferrous sulfate in adults is 12g (Wallack and Winkelstein, 1974). At the average level of 32 ug/g Fe in fast foods (Appendix 1, Table 7), maximum estimated exposure would be 0.1 g Fe/day. Hemochromatosis is a known form of liver disease associated with increased Fe stores in patients with cirrhosis (Grace, 1973). When the Fe burden exceeds the body's capacity for safe storage, the result is widespread damage to the liver, heart, joints, pancreas, and

# ARSENIC

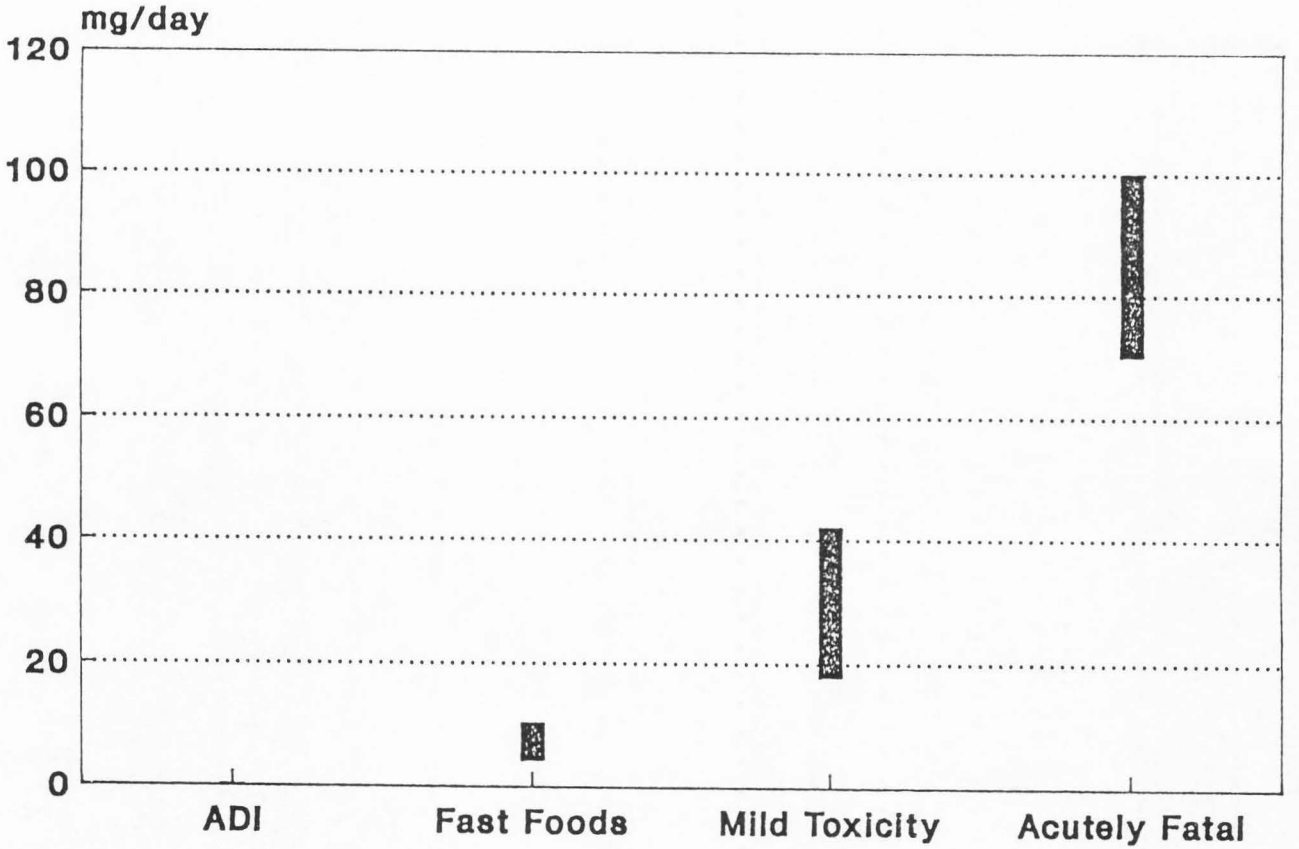


Figure 8. Maximum levels of arsenic exposure in a diet composed of 40% fast foods/day as compared to average daily intake (ADI) and levels of toxicity.

other endocrine organs. It is estimated that 0.25-0.5% of persons in various populations have the genetically determined form of iron overloading called hereditary hemochromatosis (Britton et al., 1987).

Dietary Mn is of low toxicity to animals, and levels greater than 1000 ug/g are needed to produce even slightly toxic signs. The average level of Mn found in fast foods is 5 ug/g (Appendix 1, Table 7). Manganese toxicity has not been observed as a consequence of dietary intake in humans (Levander, 1988).

The lethal dose of Cu in humans is thought to be in the range of 3.5 to 35g, with excesses of 250mg considered emetic (Solomons, 1988). Toxicological considerations are important in terms of accidental acute exposures or industrial exposure (Goyer, 1986). Daily consumption of 10-35mg of Cu is considered to be safe indefinitely (Solomons, 1988). At an average level of 1.8 ug/g (Appendix 1, Table 7), fast foods contribute a maximum of 6.5mg Cu to the diet.

Long-term ingestion of 150 mg Zn/day predisposes humans to anemia, and copper deficiency with 1.6g is considered the lethal dose (Solomons, 1988). Fast foods contain an average of 24 ug/g Zn (Appendix 1, Table 7) and contributes a maximum of 84mg Zn to the diet at the maximum level of exposure.

# CONCLUSION

X-ray fluorescence was established to be an accurate, reliable method for non-destructive multielement analysis of biological samples via determinations of Mn, Fe, Cu, and Zn in a variety of food materials and standard reference materials and by comparison with independent determinations by AAS.

In general, the concentrations of toxic minerals in fast foods are not a hazard to the average consumer. There are, however, certain segments of the population who are at increased risk for toxic mineral overload. The geriatric patient with renal impairment would be prudent to restrict excess sources of Al intake commonly seen in fast foods since Al has been associated with progression of dialysis encephalopathy and Alzheimer's disease, and the major route of Al excretion is missing in these patients.

The concentrations of Cd found in fast foods were low as a whole. This knowledge is important because of the potential for Cd accumulation in the kidney over a lifetime, resulting in severe renal tubular failure. Caution should be exercised to lower the exposure to this mineral in the diet to minimize body burden in high-risk groups.

Arsenic is not believed to pose a hazard since most foods contain the mineral in a nontoxic form, and the majority of fast foods contained arsenic at a level below detection limits. The role of As in the development of skin cancers should be considered when selecting dietary materials known to be high in As, such as seafood.

The human dietary requirement for Se appears to be easily met with the inclusion of fast foods in the diet without risk of toxicity to the consumer.

For the future, food toxicologists should plan to continue to measure toxic minerals in total diet studies to determine whether there are any trends in levels in the average U.S. diet and to follow up on any unusually high findings. Continued surveillance for these minerals in the food supply is necessary to assure optimum food safety.

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# APPENDIX

Table 7 - Quantitative measurements of minerals by atomic absorption (AA), x-ray flourescence (XRF), and inductively coupled plasma (ICP) (ug/g in dry matter).

| Food | Retail Outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Baked beans | KFC | 468 | 48.57 | nd[a] | nd[b] | nd[c] | nd[d] | 25.1 | 25.4 | 3.0 | 2.8 | 9.0 | 9.1 | 60.8 | 59.7 |
| BBQ beans | Smith's | 254 | 50.41 | nd | nd | nd | nd | 21.0 | 25.1 | 4.2 | 3.4 | 10.3 | 9.5 | 54.7 | 54.1 |
| Chili | Naugle's | 129 | 33.73 | nd | nd | 184 | nd | 70.7 | 69.7 | 1.4 | 1.7 | 4.6 | 4.6 | 49.0 | 61.5 |
| Chili | Blimipie's | 332 | 51.73 | nd | nd | nd | nd | 28.6 | 30.7 | 3.6 | 3.0 | 8.2 | 8.6 | 41.0 | 51.4 |
| Chef salad | Wendy's | 333 | 25.69 | nd | nd | nd | nd | 57.9 | 52.4 | 1.2 | 1.1 | 2.3 | 2.7 | 20.1 | 19.2 |
| Chef salad | Wendy's | 359 | 18.99 | nd | nd | nd | nd | 44.1 | 40.5 | nd | nd[e] | 4.8 | 4.4 | 41.0 | 37.8 |
| Chef salad | Wendy's | 338 | 25.32 | nd | nd | nd | nd | 40.2 | 43.2 | nd | nd | 3.9 | 3.5 | 22.5 | 22.0 |
| Chef salad | Wendy's | 408 | 24.95 | nd | nd | nd | nd | 35.6 | 37.9 | nd | nd | nd | nd[f] | 31.7 | 28.1 |
| Chef salad | Hardees | 381 | 40.95 | nd | nd | nd | nd | 41.8 | 41.5 | nd | nd | 6.4 | 6.6 | 22.7 | 22.2 |
| Chef salad | Burger King | 264 | 34.66 | nd | nd | nd | nd | 48.3 | 45.4 | 1.8 | 1.6 | 4.9 | 5.0 | 27.6 | 30.3 |
| Chef salad | Arby's | 266 | 14.21 | nd | nd | 439 | nd | 56.1 | 50.8 | 1.6 | 2.2 | nd | nd | 223 | 198 |
| Chef salad | McDonald's | 270 | 52.79 | nd | nd | nd | nd | 28.9 | 29.7 | nd | nd | nd | nd | 13.2 | 15.2 |
| Chicken soup | Smith's | 202 | 8.64 | nd | nd | nd | nd | 10.3 | 12.8 | nd | nd | 6.7 | 5.9 | 21.2 | 33.1 |
| Clam chowder | Skipper's | 318 | 41.20 | nd | nd | 272 | nd | 45.4 | 49.7 | nd | nd | 4.0 | 4.2 | 28.5 | 28.5 |
| Clam chowder | Blimpie's | 252 | 40.43 | nd | 0.7 | nd | nd | 13.9 | 15.6 | nd | nd | nd | nd | 15.2 | 11.5 |
| Garden salad | McDonald's | 184 | 50.23 | nd | 0.5 | nd | nd | 20.3 | 22.3 | nd | nd | 5.1 | 4.6 | 19.5 | 19.4 |
| Garden salad | Hardees | 337 | 50.42 | nd | nd | nd | nd | 54.5 | 46.7 | nd | nd | 4.4 | 3.9 | 12.6 | 15.9 |
| Garden salad | Burger King | 241 | 22.95 | nd | nd | nd | nd | 47.4 | 42.6 | nd | nd | 5.8 | 5.0 | 20.4 | 22.4 |
| Garden salad | Wendy's | 288 | 9.16 | nd | nd | nd | nd | 22.5 | 23.3 | 4.8 | 4.9 | 9.4 | 13.3 | 36.9 | 37.3 |
| Garden salad | Arby's | 177 | 9.17 | nd | nd | 228 | nd | 22.1 | 27.5 | 1.5 | 1.5 | 4.9 | 5.6 | 22.0 | 35.2 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Potato salad | KFC | 432 | 30.77 | nd | nd | nd | nd | 7.9 | 8.3 | nd | nd | nd | nd | 8.7 | 11.3 |
| Potato salad | Smith's | 257 | 41.30 | nd | nd | nd | nd | 9.9 | 10.2 | nd | nd | 5.9 | 5.7 | 21.4 | 23.2 |
| Potato salad | Smith's | 245 | 31.71 | nd | nd | nd | nd | 6.9 | 8.7 | 0.9 | 1.0 | 5.4 | 5.3 | 16.4 | 15.5 |
| Potato salad | Blimpie's | 211 | 34.65 | nd | nd | nd | nd | 10.8 | 10.9 | nd | nd | 3.2 | 3.2 | 16.6 | 15.9 |
| Potato salad | Albertson's | 435 | 34.47 | nd | nd | nd | nd | 8.2 | 8.5 | nd | nd | 2.5 | 2.9 | 13.7 | 13.2 |
| Cheese pizza | Peter Piper | 176 | 54.61 | 0.8 | nd | 165 | nd | 24.6 | 23.7 | 1.5 | 1.3 | 6.9 | 6.6 | 46.6 | 35.6 |
| Cheese pizza | Peter Piper | 187 | 61.30 | 0.9 | nd | nd | nd | 28.0 | 20.9 | nd | nd | 6.9 | 6.7 | 45.4 | 46.3 |
| Cheese pizza | Peter Piper | 168 | 55.91 | 0.9 | nd | nd | nd | 23.9 | 23.9 | 1.7 | 1.1 | 5.9 | 5.5 | 37.1 | 36.9 |
| Cheese pizza | Pizza Hut | 228 | 59.20 | nd | nd | nd | nd | 35.3 | 32.9 | nd | nd | 2.6 | 2.6 | 24.0 | 27.7 |
| Cheese pizza | Godfather's | 260 | 57.83 | nd | nd | nd | nd | 27.8 | 27.6 | nd | nd | 5.3 | 5.5 | 43.6 | 33.9 |
| Cheese pizza | Godfather's | 261 | 59.59 | nd | nd | 191 | nd | 25.9 | 24.6 | nd | nd | 5.1 | 4.6 | 37.9 | 39.1 |
| Cheese pizza | L.Caeser's | 217 | 59.67 | nd | nd | 113 | nd | 22.7 | 22.7 | 1.1 | 0.9 | 4.8 | 5.2 | 29.3 | 27.6 |
| Cheese pizza | Domino's | 263 | 56.15 | nd | nd | 180 | nd | 24.1 | 16.7 | nd | nd | 4.1 | 4.9 | 40.9 | 41.7 |
| Combo pizza | L.Caeser's | 283 | 54.88 | nd | nd | 119 | nd | 26.1 | 26.3 | 1.0 | 0.9 | 7.3 | 6.3 | 31.6 | 31.9 |
| Combo pizza | L.Caeser's | 262 | 58.73 | 0.6 | nd | 122 | nd | 23.2 | 26.2 | 1.3 | 0.9 | 5.3 | 5.3 | 31.6 | 32.4 |
| Combo pizza | L.Ceaser's | 268 | 49.53 | nd | nd | 171 | nd | 24.8 | 24.5 | 1.1 | 1.6 | 7.0 | 6.7 | 38.8 | 40.1 |
| Combo pizza | Godfather's | 287 | 51.13 | nd | nd | 137 | nd | 33.5 | 33.0 | 1.7 | 1.8 | 5.0 | 5.4 | 50.5 | 38.6 |
| Combo pizza | Godfather's | 261 | 62.46 | nd | nd | nd | nd | 35.0 | 31.8 | 1.9 | 1.6 | 5.8 | 6.6 | 37.9 | 38.4 |
| Combo pizza | Domino's | 360 | 49.15 | nd | nd | 129 | nd | 27.1 | 27.9 | 1.4 | 1.7 | 5.9 | 5.4 | 42.4 | 36.1 |
| Combo pizza | Domino's | 412 | 49.10 | nd | nd | 205 | nd | 31.1 | 31.4 | 1.6 | 1.2 | 4.1 | 4.8 | 34.9 | 35.1 |
| Combo pizza | Pizza Hut | 242 | 54.62 | nd | nd | 125 | nd | 27.2 | 29.9 | 1.6 | 0.9 | 5.7 | 5.0 | 36.1 | 35.1 |
| Combo pizza | Godfather's | 342 | 53.98 | nd | nd | 117 | nd | 30.6 | 33.7 | 1.4 | 1.5 | 5.0 | 5.8 | 41.0 | 40.5 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Special pizza | Peter Piper | 211 | 57.11 | 0.9 | nd | nd | nd | 30.7 | 25.8 | 1.9 | 1.5 | 7.5 | 7.6 | 85.5 | 86.1 |
| Special pizza | Peter Piper | 225 | 48.80 | 0.8 | nd | 142 | nd | 25.2 | 28.6 | 2.9 | 2.1 | 6.8 | 6.2 | 36.9 | 46.2 |
| Special pizza | Peter Piper | 268 | 54.73 | 0.9 | nd | 116 | nd | 28.6 | 28.3 | 1.7 | 1.5 | 7.5 | 7.2 | 37.7 | 36.7 |
| Supreme pizza | Pizza Hut | 246 | 57.80 | nd | nd | 148 | nd | 45.8 | 42.2 | 1.2 | 1.0 | 5.3 | 5.0 | 37.3 | 40.2 |
| Supreme pizza | Pizza Hut | 262 | 55.53 | nd | nd | 138 | nd | 23.3 | 30.9 | nd | nd | 4.7 | 4.0 | 29.9 | 36.5 |
| Supreme pizza | Pizza Hut | 218 | 63.20 | nd | nd | 131 | nd | 25.5 | 24.4 | nd | nd | 4.6 | 4.4 | 42.2 | 32.9 |
| Bean burrito | Taco Bell | 185 | 45.40 | nd | nd | nd | nd | 16.5 | 17.9 | 2.8 | 2.6 | 6.5 | 6.8 | 42.5 | 41.2 |
| Bean burrito | Taco Bell | 182 | 48.96 | nd | nd | 151 | nd | 24.5 | 21.6 | 2.3 | 2.6 | 7.4 | 7.5 | 54.2 | 46.1 |
| Bean burrito | Naugle's | 255 | 47.99 | nd | nd | nd | nd | 23.6 | 27.3 | 4.0 | 3.2 | 8.8 | 8.0 | 42.7 | 40.7 |
| Bean burrito | Naugle's | 263 | 55.76 | nd | nd | nd | nd | 19.0 | 20.1 | 2.9 | 2.4 | 10.. | 18.2 | 37.8 | 46.4 |
| Bean burrito | Lynn Wilson | 150 | 49.35 | nd | nd | nd | nd | 23.9 | 17.9 | nd | nd | 6.2 | 6.6 | 39.4 | 32.8 |
| Combo burrito | Circle K | 275 | 52.59 | nd | nd | 134 | nd | 23.2 | 25.2 | 1.4 | 0.9 | 6.1 | 6.4 | 42.4 | 38.2 |
| Combo burrito | Naugle's | 279 | 53.82 | nd | nd | 190 | nd | 43.6 | 33.4 | 1.9 | 1.8 | 6.2 | 6.0 | 37.3 | 43.6 |
| Combo burrito | Naugle's | 262 | 47.23 | nd | nd | 208 | nd | 43.2 | 42.6 | 1.9 | 1.6 | 6.5 | 6.9 | 49.7 | 43.4 |
| Combo burrito | Taco Time | 227 | 48.83 | nd | nd | 195 | nd | 25.7 | 27.3 | 2.8 | 3.1 | 9.3 | 9.1 | 39.3 | 39.1 |
| Combo burrito | Taco Time | 275 | 46.69 | nd | nd | 210 | nd | 29.5 | 36.6 | 4.9 | 4.0 | 9.8 | 9.2 | 56.9 | 51.2 |
| Combo burrito | Taco Time | 222 | 44.15 | nd | nd | nd | nd | 39.9 | 38.1 | 3.2 | 3.8 | 10.9 | 10.4 | 38.9 | 44.6 |
| Combo burrito | Taco Time | 213 | 40.87 | nd | nd | 336 | nd | 34.6 | 36.4 | 2.6 | 2.8 | 8.8 | 8.9 | 48.2 | 51.6 |
| Nachos/cheese | Naugle's | 121 | 82.80 | nd | nd | nd | nd | 28.7 | 30.5 | nd | nd | 3.6 | 3.3 | 10.4 | 14.8 |
| Nachos/cheese | Seven 11 | 189 | 67.86 | nd | nd | nd | nd | 15.4 | 15.3 | nd | nd | 3.4 | 3.8 | 17.5 | 13.9 |
| Nachos/cheese | Taco Bell | 104 | 49.66 | nd | nd | nd | nd | 18.0 | 18.8 | 1.7 | 0.8 | 4.2 | 4.9 | 12.6 | 11.0 |
| Nachos/cheese | Kmart | 245 | 50.85 | nd | nd | nd | nd | 24.4 | 18.7 | nd | nd | nd | nd | 6.0 | 8.7 |
| Nachos/cheese | Taco Time | 171 | 58.23 | nd | nd | nd | nd | 33.8 | 35.9 | nd | nd | 3.4 | 3.8 | 17.1 | 15.6 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Taco | Taco Bell | 82 | 43.12 | nd | nd | nd | nd | 61.1 | 60.8 | nd | nd | 4.9 | 4.9 | 31.4 | 35.2 |
| Taco | Taco Bell | 75 | 46.40 | nd | nd | nd | nd | 77.0 | 72.8 | nd | nd | 2.7 | 2.8 | 41.5 | 45.7 |
| Taco | Taco Bell | 66 | 45.47 | nd | nd | nd | nd | 56.7 | 56.2 | nd | nd | 2.8 | 3.4 | 39.3 | 30.1 |
| Taco | Taco Bell | 55 | 39.95 | nd | nd | nd | nd | 65.5 | 60.3 | 1.8 | 1.1 | 3.6 | 3.5 | 34.9 | 33.3 |
| Taco | Taco Time | 111 | 37.85 | nd | nd | nd | nd | 56.4 | 54.7 | 3.2 | 3.0 | 8.2 | 8.3 | 37.7 | 39.7 |
| Taco | Taco Time | 84 | 34.52 | nd | nd | 265 | nd | 43.4 | 47.6 | 3.1 | 2.8 | 9.0 | 9.5 | 31.2 | 32.4 |
| Taco | Taco Time | 63 | 38.10 | nd | nd | nd | nd | 42.9 | 46.2 | 3.2 | 2.7 | 8.8 | 8.1 | 28.7 | 37.3 |
| Taco | Taco Time | 98 | 45.32 | nd | nd | nd | nd | 51.5 | 53.7 | 3.6 | 2.9 | 9.2 | 8.0 | 40.2 | 37.8 |
| Taco | Naugle's | 51 | 51.70 | nd | nd | nd | nd | 65.9 | 51.4 | nd | nd | 4.7 | 4.5 | 32.1 | 35.1 |
| Taco | Naugle's | 100 | 41.20 | nd | nd | nd | nd | 61.0 | 59.5 | nd | nd | 4.7 | 4.7 | 34.8 | 36.8 |
| Taco | Naugle's | 82 | 45.14 | nd | nd | nd | nd | 63.5 | 62.3 | nd | nd | 3.8 | 3.6 | 36.4 | 37.8 |
| Taco | Naugle's | 95 | 40.11 | nd | nd | nd | nd | 66.7 | 65.9 | nd | nd | 4.5 | 4.2 | 28.9 | 30.4 |
| Taco | Kmart | 173 | 31.91 | nd | nd | nd | nd | 71.4 | 70.0 | 0.9 | 1.1 | 3.6 | 3.1 | 45.9 | 50.2 |
| Ch. nuggets | KFC | 106 | 51.92 | nd | nd | nd | nd | 18.7 | 16.8 | nd | nd | nd | nd | 15.2 | 12.0 |
| Ch. nuggets | KFC | 144 | 55.74 | nd | nd | nd | nd | 20.3 | 23.2 | nd | nd | 2.3 | 2.9 | 14.3 | 15.9 |
| Ch. nuggets | KFC | 148 | 49.88 | nd | nd | nd | nd | 17.8 | 18.4 | nd | nd | nd | nd | 13.6 | 11.9 |
| Ch. nuggets | McDonald's | 154 | 54.93 | nd | nd | 126 | nd | 15.4 | 13.6 | nd | nd | 2.4 | 2.3 | 13.2 | 13.2 |
| Ch. nuggets | Wendy's | 87 | 56.03 | nd | nd | nd | nd | 9.7 | 8.6 | nd | nd | nd | nd | 17.3 | 15.3 |
| Ch. nuggets | Dairy Queen | 74 | 60.94 | nd | nd | nd | nd | 6.5 | 11.0 | nd | nd | 3.2 | 3.6 | 77.2 | 65.7 |
| Ch. nuggets | Burger King | 86 | 47.29 | nd | nd | nd | nd | 19.4 | 12.5 | nd | nd | 2.5 | 2.7 | 12.4 | 13.1 |
| Chicken biscuit | KFC | 267 | 73.15 | nd | nd | nd | nd | 21.1 | 22.9 | nd | nd | nd | nd | 18.7 | 13.6 |
| Chicken biscuit | KFC | 199 | 75.31 | nd | nd | 230 | nd | 18.7 | 20.2 | nd | nd | nd | nd | 20.8 | 20.8 |
| Chicken biscuit | KFC | 274 | 80.01 | nd | nd | 369 | nd | 13.7 | 20.1 | nd | nd | 3.5 | 3.4 | 18.9 | 18.9 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Fish fillet | Skipper's | 129 | 47.50 | nd | 1.9 | nd | nd | 11.5 | 9.6 | nd | nd | nd | nd | 16.4 | 12.4 |
| Fish fillet | Skipper's | 123 | 50.72 | 0.5 | 2.5 | nd | nd | 11.2 | 10.8 | nd | nd | nd | nd | 11.1 | 13.1 |
| Fish fillet | Skipper's | 158 | 46.35 | nd | 1.9 | 134 | nd | 12.3 | 9.5 | nd | nd | 2.4 | 2.7 | 15.3 | 14.4 |
| Fish fillet | Orange Julius | 79 | 50.22 | nd | 7.3 | 99 | nd | 11.1 | 9.4 | nd | nd | 3.2 | 3.1 | 8.0 | 8.5 |
| Fish fillet | KFC | 133 | 52.82 | 0.4 | 2.9 | nd | nd | 10.9 | 10.7 | nd | nd | nd | nd | 9.6 | 8.1 |
| Fish&chips | A&W | 87 | 76.11 | nd | 0.9 | nd | nd | 11.0 | 9.5 | nd | nd | 4.6 | 4.6 | 16.2 | 14.5 |
| Orange juice | Naugle's | 186 | 10.43 | nd | nd | nd | nd | 2.7 | 4.2 | 2.8 | 2.2 | nd | nd | 17.3 | 10.1 |
| Orange juice | McDonald's | 200 | 12.05 | nd | nd | nd | nd | 2.8 | 3.4 | 2.9 | 2.0 | nd | nd | 9.3 | 9.9 |
| Orange juice | Wendy's | 180 | 17.13 | nd | nd | nd | nd | 3.5 | 3.7 | 2.0 | 2.1 | nd | nd | 7.9 | 11.4 |
| Orange juice | Burger King | 186 | 11.27 | nd | nd | nd | nd | 3.4 | 3.9 | 1.5 | 1.4 | nd | nd | 17.2 | 15.0 |
| Orange julius | Orange Julius | 381 | 8.95 | nd | nd | 144 | nd | 5.4 | 2.9 | nd | nd | nd | nd | 4.7 | 3.7 |
| Straw. shake | Hardees | 346 | 7.65 | nd | nd | nd | nd | 11.9 | 13.2 | nd | nd | nd | nd | 5.4 | 3.5 |
| Straw. shake | McDonald's | 309 | 22.06 | nd | nd | 174 | nd | 14.5 | 13.8 | nd | nd | nd | nd | 4.8 | 6.6 |
| Straw. shake | Wendy's | 328 | 28.05 | nd | nd | nd | nd | 10.0 | 12.3 | nd | nd | nd | nd | 10.4 | 10.4 |
| Straw. shake | Dairy Queen | 397 | 17.19 | nd | nd | nd | nd | 16.9 | 13.9 | nd | nd | nd | nd | 17.3 | 17.6 |
| Onion rings | KFC | 88 | 69.15 | nd | nd | nd | nd | 4.6 | 6.4 | 0.9 | 1.3 | 2.9 | 3.2 | 18.7 | 11.8 |
| Onion rings | Burger King | 59 | 70.67 | nd | nd | nd | nd | 9.8 | 7.4 | nd | nd | 4.2 | 5.2 | 14.3 | 13.7 |
| Onion rings | Arctic Circle | 125 | 54.73 | nd | nd | nd | nd | 7.7 | 7.9 | nd | nd | 4.8 | 5.9 | 13.6 | 12.8 |
| Onion rings | Dairy Queen | 99 | 62.27 | nd | nd | nd | nd | 9.9 | 8.3 | nd | nd | 6.9 | 6.9 | 49.1 | 58.7 |
| Hash browns | McDonald's | 50 | 52.96 | nd | nd | nd | nd | 9.3 | 9.6 | nd | nd | 3.5 | 3.9 | 17.1 | 16.4 |
| Hash browns | Burger King | 55 | 77.72 | nd | nd | nd | nd | 9.2 | 6.7 | nd | nd | nd | nd | 10.6 | 10.7 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| French fries | McDonald's | 77 | 69.82 | nd | nd | nd | nd | 6.5 | 7.2 | 1.5 | 1.6 | 5.6 | 5.2 | 20.7 | 17.3 |
| French fries | Hardees | 112 | 68.98 | nd | nd | nd | nd | 7.1 | 7.4 | 1.4 | 1.9 | 4.8 | 4.3 | 12.2 | 11.5 |
| French fries | Arctic Circle | 117 | 79.16 | nd | nd | nd | nd | 9.0 | 6.9 | nd | nd | 2.8 | 2.7 | 15.2 | 14.4 |
| French fries | Wendy's | 85 | 58.05 | nd | nd | nd | nd | 9.1 | 7.2 | nd | nd | 5.1 | 5.1 | 21.9 | 16.5 |
| French fries | Burger King | 71 | 56.72 | nd | nd | nd | nd | 9.9 | 10.3 | nd | nd | 3.5 | 3.5 | 23.7 | 20.8 |
| Apple pie | Burger King | 117 | 97.36 | nd | nd | 177 | nd | 3.8 | 3.4 | nd | nd | 2.5 | 2.6 | 5.9 | 6.6 |
| Apple pie | McDonald's | 92 | 92.15 | nd | nd | nd | nd | 3.4 | 3.9 | nd | nd | nd | nd | 7.6 | 7.0 |
| Apple pie | Arby's | 93 | 98.42 | nd | nd | nd | nd | 6.6 | 6.2 | nd | nd | 3.3 | 3.9 | 37.5 | 29.0 |
| Apple pie | Hardees | 98 | 99.11 | nd | nd | nd | nd | 3.8 | 3.5 | nd | nd | 3.2 | 2.9 | 5.9 | 6.0 |
| Choc. cookie | Seven 11 | 64 | 93.75 | nd | nd | nd | nd | 6.0 | 8.3 | 1.3 | 1.8 | 4.9 | 4.9 | 25.3 | 21.3 |
| Choc. cookie | Arby's | 59 | 98.70 | nd | nd | nd | nd | 7.9 | 7.6 | 2.0 | 2.0 | 5.9 | 5.6 | 21.3 | 19.3 |
| Choc. cookie | Hardees | 54 | 99.52 | nd | nd | nd | nd | 3.7 | 5.5 | nd | nd | 4.7 | 4.0 | 15.1 | 15.6 |
| Choc. cookie | McDonald's | 64 | 91.78 | nd | nd | 146 | nd | 6.4 | 5.6 | 1.5 | 1.3 | 4.5 | 5.1 | 34.7 | 27.9 |
| Choc. cookie | Grandma's | 84 | 92.96 | nd | nd | 120 | nd | 3.3 | 5.2 | nd | nd | 5.7 | 5.6 | 33.3 | 31.6 |
| Choc. sundae | Arctic C | 164 | 29.36 | nd | nd | nd | nd | 16.2 | 8.6 | nd | nd | nd | nd | 12.7 | 12.3 |
| Choc. sundae | Dairy Queen | 157 | 33.16 | nd | nd | nd | nd | 12.7 | 11.9 | 0.9 | 0.9 | nd | nd | 21.9 | 25.1 |
| Choc. sundae | Dairy Queen | 218 | 23.10 | nd | nd | nd | nd | 12.4 | 12.2 | 1.3 | 1.4 | 4.1 | 3.0 | 22.4 | 29.3 |
| Ice cream cone | Naugle's | 139 | 27.38 | nd | nd | nd | nd | 7.9 | 9.9 | nd | nd | nd | nd | 6.7 | 3.6 |
| Ice cream cone | McDonald's | 78 | 38.35 | nd | nd | nd | nd | 17.5 | 13.7 | nd | nd | nd | nd | 7.0 | 4.7 |
| Ice cream cone | Dairy Queen | 159 | 30.15 | nd | nd | nd | nd | 19.6 | 12.2 | nd | nd | nd | nd | 17.3 | 21.4 |
| Ice cream cone | Arctic Circle | 116 | 29.29 | nd | nd | nd | nd | 12.4 | 11.8 | nd | nd | nd | nd | 4.7 | 3.5 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Cake donut | Winchell's | 85 | 77.40 | nd | nd | nd | nd | 4.8 | 6.6 | nd | nd | 3.1 | 4.4 | 9.1 | 14.9 |
| Cake donut | Winchell's | 51 | 79.07 | nd | nd | nd | nd | 7.6 | 6.6 | nd | nd | 4.4 | 4.1 | 22.8 | 19.2 |
| Cake donut | Winchell's | 82 | 56.57 | nd | nd | nd | nd | 6.7 | 6.3 | nd | nd | 3.9 | 3.9 | 12.7 | 17.8 |
| Glazed donut | Winchell's | 63 | 57.13 | nd | nd | nd | nd | 6.0 | 7.1 | nd | nd | 3.9 | 3.4 | 21.0 | 23.6 |
| Glazed donut | Winchell's | 105 | 35.52 | nd | nd | 149 | nd | 3.2 | 5.8 | nd | nd | 3.7 | 3.9 | 11.6 | 17.7 |
| Glazed donut | Winchell's | 79 | 49.73 | nd | nd | 164 | nd | 6.6 | 6.3 | nd | nd | 4.5 | 4.2 | 18.4 | 18.8 |
| Choc. donut | Seven 11 | 201 | 47.65 | nd | nd | nd | nd | 5.0 | 5.4 | nd | nd | 2.8 | 2.1 | 20.1 | 17.1 |
| Choc. donut | Circle K | 209 | 58.36 | nd | nd | nd | nd | 13.9 | 7.1 | 1.9 | 1.2 | 5.2 | 4.4 | 27.6 | 29.7 |
| Choc. donut | Dolly Madison | 112 | 60.02 | nd | nd | nd | nd | 5.9 | 8.6 | 2.4 | 2.2 | 4.9 | 4.6 | 39.3 | 31.8 |
| Choc. donut | Dolly Madison | 139 | 75.96 | nd | nd | 153 | nd | 4.8 | 5.5 | nd | nd | nd | nd | 17.1 | 17.3 |
| Donut gems | Hostess | 103 | 83.46 | nd | nd | nd | nd | 8.1 | 8.1 | 1.6 | 1.6 | 5.1 | 5.3 | 38.2 | 33.3 |
| Danish pastry | Albertson's | 99 | 76.65 | nd | nd | nd | nd | 10.7 | 9.9 | nd | nd | 3.3 | 3.2 | 23.6 | 18.8 |
| Cinnamon roll | Farmer Jack | 153 | 84.79 | nd | nd | 249 | nd | 7.9 | 8.4 | nd | nd | 4.6 | 4.5 | 27.6 | 32.7 |
| Cinnamon roll | Farmer Jack | 180 | 72.54 | 0.4 | nd | 243 | nd | 5.3 | 8.1 | nd | nd | 6.2 | 6.1 | 31.9 | 35.3 |
| Cinnamon roll | Smith's | 220 | 79.59 | nd | nd | nd | nd | 6.8 | 6.6 | nd | nd | 5.2 | 4.8 | 32.2 | 32.4 |
| Cinnamon roll | Smith's | 213 | 71.72 | nd | nd | nd | nd | 11.5 | 10.6 | 1.7 | 1.5 | 6.7 | 7.5 | 38.2 | 30.9 |
| Cinnamon roll | Albertson's | 110 | 66.02 | nd | nd | 217 | nd | 6.6 | 7.5 | nd | nd | 5.1 | 5.5 | 17.4 | 16.7 |
| Cinnamon roll | Albertson's | 154 | 55.55 | nd | nd | 166 | nd | 8.6 | 8.1 | nd | nd | 4.9 | 5.8 | 27.3 | 24.8 |
| Country brkfst. | Hardees | 356 | 58.12 | 0.5 | nd | 346 | nd | 25.0 | 27.4 | nd | nd | nd | nd | 38.5 | 34.8 |
| Big breakfast | McDonald's | 294 | 53.81 | nd | nd | nd | nd | 28.5 | 23.4 | nd | nd | 2.4 | 2.3 | 38.1 | 33.8 |
| Egg McMuffin | McDonald's | 143 | 66.26 | 0.7 | nd | nd | nd | 22.5 | 25.6 | nd | nd | 4.1 | 3.1 | 39.3 | 41.6 |
| Ham bagel | Burger King | 201 | 76.17 | 0.7 | nd | nd | nd | 33.1 | 30.6 | nd | nd | 3.1 | 3.4 | 42.5 | 36.3 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ham croussant | Circle K | 124 | 77.20 | 0.7 | nd | 303 | nd | 20.8 | 22.9 | nd | nd | 3.8 | 3.1 | 39.9 | 36.3 |
| Sausage&biscuit | Circle K | 122 | 88.19 | nd | nd | nd | nd | 16.6 | 14.7 | nd | nd | 3.0 | 4.5 | 53.4 | 43.7 |
| Sausage&eggs | Burger King | 241 | 48.06 | nd | nd | nd | nd | 20.8 | 25.1 | nd | nd | 2.4 | 3.3 | 34.1 | 36.9 |
| Sunrise biscuit | Hardees | 195 | 70.93 | nd | nd | 583 | nd | 19.0 | 19.6 | nd | nd | nd | nd | 19.6 | 25.2 |
| Hamburger | Wendy's | 105 | 50.43 | nd | nd | 148 | nd | 32.1 | 34.8 | nd | nd | 3.9 | 4.5 | 60.7 | 58.8 |
| Hamburger | Wendy's | 96 | 47.04 | nd | nd | 145 | nd | 38.3 | 38.3 | nd | nd | 4.5 | 4.3 | 56.9 | 51.4 |
| Hamburger | Wendy's | 177 | 48.29 | nd | nd | nd | nd | 46.8 | 47.5 | nd | nd | 2.6 | 3.5 | 56.9 | 57.5 |
| Hamburger | McDonald's | 107 | 56.05 | nd | nd | 185 | nd | 30.7 | 26.2 | nd | nd | 4.9 | 4.6 | 30.7 | 30.7 |
| Hamburger | McDonald's | 102 | 53.81 | nd | nd | 146 | nd | 27.8 | 27.3 | nd | nd | 4.6 | 4.6 | 43.0 | 36.2 |
| Hamburger | Arctic Circle | 99 | 58.94 | nd | nd | 158 | nd | 31.6 | 32.0 | nd | nd | 4.1 | 4.1 | 45.7 | 46.8 |
| Hamburger | Arctic Circle | 107 | 61.18 | nd | nd | 165 | nd | 38.6 | 37.0 | nd | nd | 3.5 | 3.4 | 38.9 | 42.4 |
| Hamburger | Arctic Circle | 100 | 57.76 | nd | nd | 144 | nd | 18.1 | 18.3 | nd | nd | 5.4 | 5.2 | 55.6 | 48.3 |
| Hamburger | Burger King | 110 | 58.84 | nd | nd | 198 | nd | 34.0 | 34.6 | 1.9 | 1.4 | 4.2 | 4.4 | 51.0 | 50.7 |
| Hamburger | Burger King | 107 | 57.75 | nd | nd | 158 | nd | 32.7 | 30.7 | 1.4 | 1.2 | 4.8 | 4.7 | 47.8 | 43.6 |
| Hamburger | Burger King | 107 | 53.54 | nd | nd | 174 | nd | 27.7 | 28.2 | nd | nd | 4.9 | 4.6 | 39.4 | 47.9 |
| Hamburger | Hardees | 113 | 51.79 | nd | nd | 153 | nd | 31.9 | 29.2 | 1.3 | 1.2 | 4.0 | 4.7 | 57.7 | 46.5 |
| Hamburger | Hardees | 97 | 55.06 | nd | nd | 133 | nd | 38.1 | 31.6 | 1.6 | 1.0 | 5.3 | 4.9 | 58.4 | 46.2 |
| Hamburger | Hardees | 105 | 58.32 | nd | nd | 232 | nd | 25.6 | 23.4 | nd | nd | 4.9 | 5.0 | 46.9 | 47.1 |
| Big classic | Wendy's | 239 | 48.43 | nd | nd | 110 | nd | 33.8 | 36.7 | nd | nd | 2.4 | 2.7 | 45.4 | 45.4 |
| Big classic | Wendy's | 249 | 36.83 | nd | nd | nd | nd | 57.7 | 52.5 | 1.6 | 1.4 | 5.1 | 4.8 | 51.4 | 51.7 |
| Big classic | Wendy's | 290 | 50.43 | nd | nd | 146 | nd | 29.2 | 26.6 | nd | nd | 3.7 | 4.1 | 44.5 | 44.2 |
| Whopper | Burger King | 309 | 47.13 | nd | nd | 114 | nd | 29.2 | 29.6 | 1.1 | 1.1 | 4.0 | 3.7 | 48.8 | 39.0 |
| Whopper | Burger King | 297 | 59.40 | nd | nd | nd | nd | 19.5 | 19.6 | 1.5 | 1.5 | 5.5 | 5.8 | 43.9 | 43.2 |
| Whopper | Burger King | 287 | 47.92 | nd | nd | 134 | nd | 32.6 | 34.8 | nd | nd | 2.4 | 2.9 | 30.9 | 38.6 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Big deluxe | Hardees | 240 | 45.73 | nd | nd | nd | nd | 32.3 | 47.1 | nd | nd | 4.9 | 4.7 | 47.8 | 47.2 |
| Big deluxe | Hardees | 231 | 60.96 | nd | nd | nd | nd | 45.7 | 43.7 | 1.6 | 0.9 | 5.8 | 5.9 | 47.1 | 44.9 |
| Big deluxe | Hardees | 254 | 40.35 | nd | nd | 116 | nd | 45.0 | 47.6 | 1.7 | 1.5 | 3.7 | 3.5 | 50.2 | 52.0 |
| Big Mac | McDonald's | 220 | 57.72 | nd | nd | nd | nd | 26.9 | 29.3 | nd | nd | 3.9 | 3.3 | 36.8 | 33.4 |
| Big Mac | McDonald's | 209 | 50.91 | nd | nd | 132 | nd | 52.1 | 50.7 | nd | nd | 3.0 | 3.0 | 38.6 | 36.7 |
| Big Mac | McDonald's | 199 | 50.83 | nd | nd | 154 | nd | 33.4 | 36.5 | nd | nd | 3.5 | 3.1 | 40.2 | 39.4 |
| Bounty burger | Arctic Circle | 214 | 51.02 | nd | nd | 170 | nd | 38.8 | 37.6 | nd | nd | 3.5 | 4.2 | 48.0 | 47.5 |
| Bounty burger | Arctic Circle | 194 | 50.62 | nd | nd | 145 | nd | 29.0 | 27.0 | 1.6 | 1.1 | 4.5 | 4.2 | 54.2 | 51.4 |
| Bounty burger | Arctic Circle | 202 | 61.77 | nd | nd | 166 | nd | 27.6 | 26.6 | nd | nd | 4.4 | 4.4 | 45.3 | 41.2 |
| Hot dog | Dairy Queen | 89 | 70.82 | nd | nd | nd | nd | 16.1 | 21.1 | nd | nd | 2.5 | 2.9 | 29.3 | 31.6 |
| Hot dog | Arctic Circle | 145 | 67.78 | nd | nd | nd | nd | 22.7 | 17.4 | nd | nd | 5.0 | 4.5 | 33.9 | 37.2 |
| Hot dog | Seven 11 | 156 | 52.25 | nd | nd | nd | nd | 32.5 | 31.3 | nd | nd | 4.1 | 4.2 | 45.0 | 45.0 |
| Hot dog | Weinersch. | 88 | 63.30 | nd | nd | nd | nd | 19.5 | 21.2 | nd | nd | 5.2 | 4.6 | 39.7 | 40.4 |
| Hot dog | Weinersch. | 85 | 62.43 | nd | nd | nd | nd | 20.4 | 20.6 | nd | nd | 3.5 | 3.5 | 44.5 | 40.9 |
| Hot dog | Weinersch. | 89 | 52.63 | nd | nd | 145 | nd | 22.6 | 21.6 | nd | nd | 4.5 | 5.2 | 49.5 | 43.4 |
| Classic dog | Weinersch. | 173 | 57.00 | nd | nd | nd | nd | 31.0 | 29.7 | nd | nd | 2.9 | 3.3 | 36.6 | 38.7 |
| Classic dog | Weinersch. | 207 | 51.52 | nd | nd | nd | nd | 46.4 | 40.4 | nd | nd | 4.2 | 4.0 | 35.8 | 36.9 |
| Classic dog | Weinersch. | 182 | 54.21 | nd | nd | nd | nd | 30.1 | 29.4 | nd | nd | 3.0 | 3.0 | 45.7 | 43.7 |
| Roast beef | Circle K | 144 | 49.31 | nd | nd | 241 | nd | 29.3 | 33.2 | nd | nd | 7.2 | 6.3 | 47.5 | 42.6 |
| Roast beef | Arby's | 162 | 51.08 | nd | nd | nd | nd | 48.3 | 49.8 | 1.6 | 1.0 | 4.4 | 3.9 | 57.4 | 51.6 |
| Roast beef | Arby's | 153 | 52.48 | nd | nd | nd | nd | 37.9 | 34.9 | nd | nd | 3.4 | 3.3 | 45.8 | 43.0 |
| Roast beef | Arby's | 142 | 52.57 | nd | nd | 151 | nd | 38.4 | 28.3 | nd | nd | 3.3 | 3.1 | 43.4 | 48.9 |
| Roast beef | Hardees | 169 | 45.51 | nd | nd | nd | nd | 26.8 | 47.8 | nd | nd | 4.0 | 4.3 | 47.4 | 48.8 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Beef&swiss | Subway | 213 | 47.46 | nd | nd | nd | nd | 50.9 | 53.9 | nd | nd | 4.9 | 4.6 | 48.5 | 50.3 |
| Beef&swiss | Arby's | 168 | 46.93 | nd | nd | nd | nd | 31.7 | 35.8 | nd | nd | 3.4 | 3.2 | 37.6 | 42.4 |
| Beef&swiss | Arby's | 164 | 49.55 | nd | nd | nd | nd | 35.6 | 38.5 | nd | nd | 3.8 | 3.9 | 32.5 | 38.4 |
| Beef&swiss | Arby's | 168 | 47.42 | nd | nd | nd | nd | 34.4 | 39.8 | 0.9 | 1.0 | 3.1 | 3.3 | 33.3 | 36.1 |
| Beef&cheese | Seven 11 | 240 | 56.89 | nd | nd | nd | nd | 30.1 | 33.0 | nd | nd | 4.3 | 4.2 | 47.8 | 51.6 |
| Sub sandwich | Kmart | 233 | 44.54 | nd | nd | nd | nd | 20.9 | 18.3 | nd | nd | 4.1 | 4.8 | 35.2 | 39.5 |
| Italian sub | L. Caeser's | 275 | 43.00 | 0.5 | nd | nd | nd | 23.6 | 29.5 | 0.6 | 0.9 | 5.1 | 5.8 | 35.6 | 34.5 |
| Best sandwich | Blimpie's | 245 | 59.80 | 0.4 | nd | 196 | nd | 20.4 | 24.0 | nd | nd | 5.2 | 6.0 | 49.9 | 44.7 |
| Best sandwich | Blimpie's | 226 | 43.17 | nd | nd | 149 | nd | 24.5 | 21.9 | nd | nd | 4.6 | 4.5 | 41.0 | 39.6 |
| Best sandwich | Blimpie's | 272 | 38.57 | nd | nd | 179 | nd | 30.0 | 31.3 | 1.5 | 1.6 | 5.2 | 5.8 | 45.0 | 43.6 |
| BMT sandwich | Subway | 203 | 43.39 | nd | nd | nd | nd | 26.7 | 28.1 | nd | nd | 4.7 | 4.6 | 32.4 | 34.4 |
| BMT sandwich | Subway | 233 | 43.16 | nd | nd | nd | nd | 33.7 | 30.2 | nd | nd | 4.5 | 4.6 | 37.5 | 37.7 |
| BMT sandwich | Subway | 228 | 38.23 | nd | nd | 233 | nd | 29.8 | 26.8 | nd | nd | 3.5 | 4.6 | 36.2 | 33.7 |
| Club sandwich | Blimpie's | 249 | 36.68 | nd | nd | nd | nd | 24.7 | 22.8 | nd | nd | 6.2 | 6.1 | 46.3 | 48.1 |
| Club sandwich | Blimpie's | 244 | 43.52 | nd | nd | nd | nd | 28.7 | 22.8 | nd | nd | 8.4 | 7.0 | 60.8 | 52.6 |
| Club sandwich | Subway | 200 | 39.83 | nd | nd | nd | nd | 46.1 | 40.7 | nd | nd | nd | nd | 33.7 | 35.6 |
| Club sandwich | Subway | 229 | 33.85 | nd | nd | nd | nd | 29.5 | 33.2 | nd | nd | 4.6 | 4.9 | 31.7 | 35.8 |
| Chicken sand. | Arctic Circle | 203 | 64.05 | nd | nd | nd | nd | 8.2 | 9.4 | nd | nd | 4.3 | 5.3 | 26.4 | 32.0 |
| Chicken sand. | Dairy Queen | 205 | 53.03 | nd | nd | 116 | nd | 9.4 | 9.8 | nd | nd | 3.9 | 3.9 | 39.0 | 48.5 |
| Chicken sand. | Wendy's | 195 | 49.47 | nd | nd | 134 | nd | 7.8 | 10.0 | nd | nd | 5.6 | 4.5 | 24.5 | 27.9 |
| Chicken sand. | Burger King | 194 | 53.80 | nd | nd | 343 | nd | 13.3 | 11.6 | nd | nd | 4.3 | 3.7 | 20.0 | 28.0 |
| Chicken sand. | Hardees | 190 | 53.17 | nd | nd | 149 | nd | 7.2 | 8.9 | nd | nd | 4.9 | 4.7 | 26.4 | 25.3 |

(continued)

Table 7 (continued)

| Food | Retail outlet | Serving size (g) | DM % | Se XRF | As XRF | Al XRF | Cd ICP | Zn AA | Zn XRF | Cu AA | Cu XRF | Mn AA | Mn XRF | Fe AA | Fe XRF |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Fish sandwich | McDonald | 138 | 58.26 | 0.5 | 0.9 | 153 | nd | 11.3 | 12.3 | nd | nd | 4.8 | 4.9 | 24.2 | 21.8 |
| Fish sand./cheese | Skipper's | 182 | 60.95 | nd | nd | nd | nd | 16.3 | 15.9 | nd | nd | 4.2 | 3.6 | 18.2 | 18.9 |
| Fish sand./cheese | Skipper's | 163 | 58.93 | 0.6 | 0.9 | 135 | nd | 19.1 | 16.3 | nd | nd | nd | nd | 21.9 | 28.1 |
| Fish sand./cheese | Skipper's | 158 | 52.86 | nd | nd | 129 | nd | 23.3 | 28.0 | nd | nd | 2.6 | 2.5 | 20.9 | 22.2 |
| Fish sand./cheese | Arctic Circle | 176 | 54.67 | nd | 3.3 | nd | nd | 8.7 | 10.0 | nd | nd | 4.2 | 4.9 | 28.7 | 27.2 |
| Fish sand./cheese | Hardees | 219 | 53.37 | nd | 0.6 | nd | nd | 10.6 | 12.0 | nd | nd | 7.8 | 8.1 | 37.8 | 35.3 |
| Fish sandwich | Burger King | 195 | 48.89 | nd | 1.3 | nd | nd | 8.6 | 9.0 | nd | nd | 4.1 | 3.8 | 20.7 | 22.9 |
| | | | | | | Mean | | 24.6 | 24.5 | 1.9 | 1.8 | 4.9 | 4.9 | 32.6 | 32.2 |
| | | | | | | No. samples | | | 228 | | 74 | | 192 | | 228 |
| | | | | | | Correlation ($R^2$) | | | .97 | | .91 | | .94 | | .97 |
| | | | | | | Slope | | | .95 | | .83 | | .93 | | .90 |
| | | | | | | Intercept | | | 1.03 | | .15 | | .36 | | 2.8 |

[a]All measurements below detection limit of 0.40 ug/g Se by XRF
[b]All measurements below detection limit of 0.40 ug/g As by XRF
[c]All measurements below detection limit of 90 ug/g Al by XRF
[d]All measurements below detection limit of 0.05 ug/g Cd by ICP
[e]All measurements below detection limit of 0.7 ug/g Cu by XRF
[f]All measurements below detection limit of 0.8 ug/g Mn by XRF