

UNIVERSITY OF CAPE COAST

THE LEVEL OF CONTAMINATION OF ALUMINIUM IN
COOKED FOODS FROM ALUMINIUM UTENSILS FROM
KOTOKURABA MARKET IN THE CAPE COAST METROPOLIS IN
CENTRAL REGION, GHANA.



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CENTRAL REGION, GHANA.

BY

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Education of the Faculty of Science and Technology, College of Education
Studies, University of Cape Coast in partial fulfillment of the requirements
for the award of Master of Philosophy degree in Home Economics
Education.

NOVEMBER, 2023

DECLARATION

Candidate's Declaration

I hereby declare that this thesis work is the result of my unique study, and no portion of it has ever been submitted for another degree at this university or elsewhere.

Candidate's Signature: Date:

Name: Lydia Abia Adukpoh

Supervisor's Declaration

I hereby declare that the thesis work preparation and presentation were overseen in compliance with the University of Cape Coast's MPhil thesis work supervision criteria.

Supervisor's Signature: Date:

Name: Dr. (Mrs.) Augusta Adjei Frimpong

ABSTRACT

In Ghana most households and restaurants use aluminum cooking utensils to prepare food, however, few studies have considered the possibility of the leaching of the metal into foods. For this reason, the major goal of this study was to determine the amount of aluminium leached from aluminium-made cooking utensils into selected cooked foods namely: fresh tomatoes, rice, and cocoyam leaves. Three different brands of aluminium cooking utensils were chosen from a local market specifically Kotoburaba market, Cape Coast, in the Central Region of Ghana and labelled A, B, and C to cook the food samples for the study. Samples were purchased from the Kotoburaba market for the nine-week research study. Each week, the samples underwent preparation and an analysis to determine the amount of aluminum present both before and after cooking. The acquired data were analysed using a one-way analysis of variance (ANOVA). The results presented p-values greater than 0.05. Results obtained indicate that cocoyam leaves (nkontomire) recorded the highest amount of leached aluminium metal with $3.54\text{mg/kg} \pm 1.23$ from the utensil, followed closely by tomatoes with $3.16\text{mg/kg} \pm 1.87$ and the least being rice with $2.67\text{mg/kg} \pm 1.38$ over the nine weeks. The type of aluminium utensils used, their prior use, the pH of the food, its form and content, the cooking duration and the presence of silica and other ions such as F^- were considered as conditions that facilitate the leaching of aluminium into foods. This study contributes to improving the knowledge of health practitioners, cooks and food lovers about the health effects of cooking in aluminium manufactured utensils. Food vendors and users of aluminium cooking utensils should be educated on the toxicological effects of using aluminium utensils by FDA and GHS sections in Cape Coast.

KEYWORDS

Aluminum Cooking Utensils

Leaching of Metal

Cooked Food

Aluminum in Food

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DEDICATION

To my lovely mother.

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CHAPTER ONE

INTRODUCTION

Overview

The chapter discusses the study's background, its problem statement, the purpose of the study, the general and specific objectives, the research questions, and the significance of the study. Again, the study's delimitations and limitations are specified to clearly describe the scope of the research and potential drawbacks. The chapter concludes with an explanation of the keywords

Background of the Study

According to Rahman (2009), food is any substance that is consumed to give an organism energy and nutritional support. Animals ingest food orally for growth, health, or pleasure in raw, processed, or formulated forms. Water, lipids, proteins, and carbohydrates make up the majority of food consumed (Whitney & Rolfes, 2015). Foods also contain minerals and vitamins as well as water; which is also been classified as a food (Jacobs & Tapsell, 2007). Water and fibre contain little calories and a low energy density whereas fat is the component of food with the highest energy density (Jacobs & Tapsell, 2007). Processed and non- processed foods has been man's source of survival for a very longtime. In order to make food palatable and safe for mankind, there is the need for it to be cooked and the items in which foods are cooked are sometimes another source of danger to the health of man. The use of aluminium utensils have been a very significant contributor to chemical leaching into food consumed by man.

The production and use of aluminium have increased worldwide, and Ghana is not an exception due to its availability and accessibility. Production and use have seen increases over time because aluminium has been identified as the third most common element in nature which makes up eight percent (8%) of the earth's surface (Mantinez et.al, 2017). Magnesium and potassium occur naturally in water and contribute to the metabolic activities of the human body. Potassium plays a role of helping to maintain normal levels of fluid inside the cells of the body, helps muscles to contract and support normal blood pressure. Magnesium helps the heart maintain a healthy rhythm and is involved in the regulation of blood pressure and cholesterol production (Fronterhouse, 2014). Aluminium on the other hand although abundant in water has limited benefits in the pathway of metabolism (Luis et, al, 2015). This seemingly increases in the use of aluminium could stem from its attractive physical and chemical qualities which include: high tensile strength, low specific gravity, corrosion resistance, malleability, ductility, high thermal and electric conductivity and relatively low purchasing cost (Zotov et al, 2018). Moreover, it has a variation of industrial uses such as; transport, electrical engineering, and manufacturing of household utensils, appliances and packaging materials (Rabinovich, 2013).

The food types that contribute mostly to the daily intake of aluminium include; wheat and wheat-based items, as well as vegetables, cereals, fruits, tea, cereals and coffee (Bratakos et al., 2012; Fekete et al., 2013; González et al., 2010). The food processing, packaging and preservation industries have over the years increased the use of aluminium

due to its availability. Samwel et al (2006), argue that these industries are not the only source of the contribution of aluminium to the human body. According to Anke, Inhat and Stoeppler (2004), aluminium is now popular in the creation of antacids, analgesics, and anti-diarrhoea medications. Another source of aluminium that cannot be overlooked, includes the use of cook wares that are made from aluminium. The use of aluminium cooking utensils provides a significant channel for aluminium metal absorption into foods cooked in aluminium utensils and consequently to consumers (Semwal et al., 2006). In support, Karbouj (2007), acknowledges the possible source of supplementary aluminium in the diet as obtained from aluminium cook wares such as saucepans, foils (wrappers), pressure cookers, skillets, and roasting pans.

Accordingly, the process of aluminium leaching is explained by Alabi and Adeoluwa (2020) as a chemical reaction: $\text{Al}_2\text{O}_3 + 6\text{H}^+ = 2\text{Al}^{3+} + 3\text{H}_2\text{O}$, where Aluminium Oxide (Al_2O_3), acts as a protective layer. Free aluminium in solutions combined with organic acids such as citric, acetic, and oxalic, as well as other complex ligands found in foods such as fluoride and hydroxyl. These reactions often occur concurrently and stimulate each other irrespective of the types of food being prepared because the recipe and methods of preparation play important roles in the leaching levels. Therefore, it is envisaged that investigating the possible contamination of selected cooked foods using aluminium cooking utensils could be of utmost importance to users of aluminium products. This implies intake of aluminium daily is contingent on the types of food consumed.

Statement of Problem

Generally, foods are more prone to aluminum contamination when they are cooked in aluminum cooking utensils (Adelkhani, Nasoodi & Jafari, 2009; Dabonne et.al, 2010; Dan & Ebong, 2013). This is mostly from a chemical reaction that occurs during food preparation (Alabi & Adeoluwa, 2020). Individuals are exposed to large amounts of aluminium after consuming foods cooked in utensils made of aluminium (Sorenson et. al., 1974; Karbouj, 2007). Aluminum ions compete with other ions, such as calcium, iron, magnesium, fluoride, phosphorus, and others, to limit certain metabolic activities in the human body (Mahieu et al., 2004; Kaur and Gill, 2005). Consumption of aluminium in food encourage the incidence of illnesses consisting of Alzheimer's illness, Parkinson's disease, breast cancer and bone disease in humans (Al Zubaidy et. al, 2011; Al Juhaiman, Al-Shihry & Al-Hazimi, 2014).

In Ghana, specifically Cape Coast in the Central Region, most households, restaurants, and community kitchens use aluminium utensil due to its availability and cheap price to prepare foods for human consumption but little is known about the risks associated with it. Studies like Bannerman (1996), conducted on tomato, plantain, cowpea and cocoyam leaves in Kumasi – Ghana have observed significant amount of aluminium in foods cooked with aluminium pots. Indicating, the potential of aluminium utensils to contribute to aluminium intake. Nonetheless, another research worth mentioning conducted in Kumasi by Ankar-Brewoo et al (2020), assessed the concentrations and dietary risk of the toxic metal Aluminium in vended fufu and fried-rice. The study, showed a relatively

high concentration of the aluminium metal in comparison to the PTWI of aluminium.

However in Cape Coast limited research has been done to consider the leaching of aluminium metal into selected foods such as tomato, nkontomire and rice. This study, therefore, sought to investigate the possible contamination of selected foods cooked in aluminium cooking utensils.

Purpose of the Study

To assess the extent to which the aluminium metal can leach into cooked foods from aluminium-made cooking utensils.

Research Objectives

The study sought to:

1. Determine the concentration of aluminium in selected raw and cooked foods
2. Examine the conditions (temperature, pH and cooking time) that may facilitate the leaching of aluminium into cooked foods.
3. Compare levels of aluminium leaching from different types of aluminium cooking utensils.
4. Determine the level of aluminium in prepared foods.

Research Questions

The study's goal is to obtain answers to the following questions.

1. What is the concentration of aluminium in the selected raw and cooked foods?
2. Under which conditions can the leaching of aluminium into foods take place?

3. What are the levels of aluminium leached from the different types of aluminium utensils?
4. Does the level of aluminium in the prepared food match the recommended standards?

Research Hypothesis

H_0 : There is no statistically significant difference in the level of aluminium concentration of the selected cooked foods over the 9 weeks' period.

H_1 : There is a statistically significant difference in the level of aluminium concentration of the selected cooked foods over the 9 weeks' period.

Significance of the Study

This study has investigated the possible aluminium contamination of selected foods prepared using aluminium cooking utensils from the Kotokuraba market in Cape Coast. Elevated concentration of aluminium in foods contributes to metal poisoning in meals and therefore raises the concern about the safety of food. The findings of this study have several important implications for public health and food safety in Cape Coast, Ghana.

The results of this research study finds it beneficial to the households, restaurants and community kitchens. It is also beneficial to stakeholders in the food value chain, including those in the packaging, processing and storage sector in Cape Coast Ghana, where aluminium-made cooking utensils are the mostly used in order to ensure consumer protection. This study helps stakeholders know which kind of foodstuff are prone to leaching aluminium, the concentration of aluminium leached and

the effects of continuous intake of foods prepared in aluminium utensils. Thus, the results of this study has contributed to improving the knowledge of health practitioners, matrons, cooks and food lovers about the health effects of cooking in aluminium utensils. It has also served as a basis for advanced research and has shed more light on the contributing factors for the leaching of aluminium from utensils into food.

The study has identified several factors that contribute to aluminium contamination in cooked foods including, the pH of the food, cooking temperature and the cooking duration. This knowledge can be used to develop interventions to reduce aluminium contamination. Also, the study has highlighted the need for food safety awareness and education among vendors and consumers in Cape Coast. This includes understanding the risks of aluminium contamination and how to reduce it. The research has shown that leaching of aluminium from utensils is real as such it has provided evidence to support the implementation of regulations that limit the use of aluminium utensils and promote the use of safer alternatives.

Overall, the findings of the study can be used to inform public health policy decisions, food safety practices and consumer education initiatives in Cape Coast aimed at reducing aluminium exposure in the population.

Delimitation

The research investigated the level of aluminium contamination in tomatoes, cocoyam leaves (nkontomire) and rice cooked in new, indamaged aluminium utensils purchased from kotokuraba market in Cape Coast, Central region of Ghana.

Limitation

The study conducted used a single aluminium utensil for a particular food sample. However cooking the selected foods in all three purchased aluminium utensils was the idle. Hence this study acknowledges the former as a limitation.

Another limitation in using complexometric back titration was that, certain ions, such as iron and calcium, can interfere with the titration. This method also cannot detect low concentrations of aluminium accurately.

Operational Definition of Terms

Aluminium: Aluminium (Al) is the earth's crust's most copious metal which is extensively distributed representing approximately 8% of its total mineral content (Samwel et al, 2006).

Aluminum utensils: Aluminum utensils refer to kitchen tools and cookware made primarily from aluminum, a lightweight and highly conductive metal. These utensils include a wide range of items such as pots, pans, baking sheets, spoons, ladles, pressure cookers, and more, designed for various cooking and food preparation tasks

Cooked food refers to food items that have undergone a process of preparation and heating to make them safe to eat, enhance their flavor, improve their texture, and increase their nutritional value.

Aluminum in cooked foods refers to the presence of aluminum particles or ions that have leached from aluminum cookware, utensils, or packaging materials into the food during the cooking process.

Organisation of the Study

This research was structured into five sections. Chapter One comprised the background to the study, statement of the problem, objectives of the study, research questions, significance of the study, delimitations, limitations definition of terms and organization of the study.

Chapter Two of the study was concentrated on literature review with more light on research results produced by other writers linked to the issue under study. Chapter Three examined the research methods to be used for the study which included the research design, sampling procedures, population, data collection instrument and methods for data collection and analysis.

The fourth chapter dealt with the results as well as the discussion of the findings. The final chapter comprised the summary, conclusions and recommendations based on the results of the study.

CHAPTER TWO

LITERATURE REVIEW

This chapter examined previous works from academic books, articles, journals, websites and other publications which were related to the study. The review incorporated theoretical framework, conceptual review and empirical review. The theoretical framework was based on the theory: migration from food contact materials (Bradley & Castle, 2021). The conceptual review focused on the concept of leaching. The empirical review examined the findings of this study with other related studies to either confirm or reject conclusions drawn by earlier researchers. The empirical review examined findings under each objective and focused on topics such as aluminium metal, uses and sources of aluminium, toxicological effects of aluminium consumption, “absorption, distribution, metabolism, and excretion” of the aluminium metal in the human system and comparative standards of aluminium which includes: specific release limit (SRL), provisional tolerance weekly intake (PTWI).

Theoretical Framework

The theoretical framework of this study is based on the theory: migration from food contact materials according to Bradley and Castle, (2021). Migration refers to the process by which substances present in materials, including utensils, may transfer to the food or beverage during storage, processing or cooking (Arvanitoyannis & Kotsanopoulos, 2014). () In this study, the migration theory would be used to address how aluminium from utensils migrates into the cooked foods.

Bradley and Castle (2021) are of the view that migration from a material occurs at the interface with the food and factors that cause migration are: the composition of food, the temperature, duration of contact and acidity of the food. According to Bradley and Castle (2021), chemical migration is defined as “the mass transfer from an external source into food by submicroscopic processes.” The extent to which any substance migrates into a foodstuff is controlled by the diffusion processes that are subject to both kinetic and thermodynamic control. However, migration from materials, such as glass, ceramics, or metal occurs only from the surface of the material, and no diffusion of the substances will occur from within these materials to the surface (Bradley & Castle, 2021). The theory of migration is illustrated by the diagram below according to Bradley and Castle (2021).

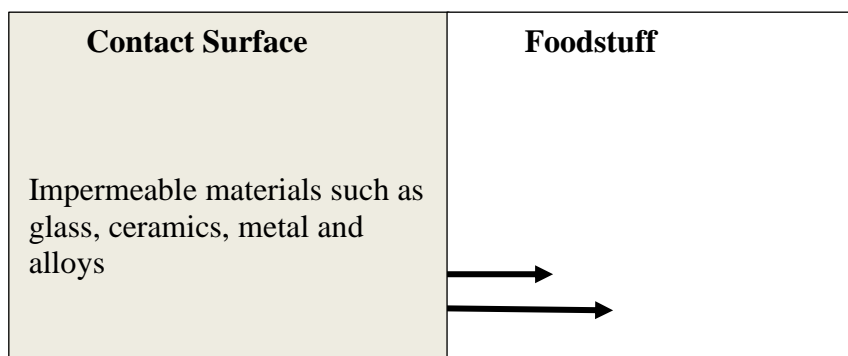


Figure 1: Migration process of contaminant from contact surface into foodstuff.

Source: Bradley and Castle (2021)

Conceptual Review

Leaching of metal in food

The concept associated with this research is generally related to leaching of metal into foods. According to Zheng, Zheng and Chen (2012), generally the leaching of metals into environmental matrix are mostly as a result of acidic conditions. This opportunity contributes to the

contamination and release of toxic metals into the environment. In a research conducted by Zheng, Zheng and Chen (2012), on 'leaching behaviour of heavy metals and transformation of their speciation in polluted soil receiving simulated acid rain', the authors affirm that leaching is one of the concepts underlying the release of aluminium into soil. Zheng et al. (2012), define leaching of metal as the process by which contaminants are transferred from a stabilized matrix to liquid medium, such as water or other solutions. In the research the authors avows that the influence of acid rain (lower pH) is among the contributors to metal pollution of soil. In another vain, Alabi and Adeoluwa (2020) has said that there is contamination of food with aluminium through the concept of leaching when food comes into contact with aluminium utensil. The concept involves the wearing away of aluminium oxide (Al_2O_3) protective layer of aluminium cooking utensils as a result of exposure to acids, high temperature, prolonged cooking duration. The process of aluminium leaching is explained by Alabi and Adeoluwa (2020) as a chemical reaction: $\text{Al}_2\text{O}_3 + 6\text{H}^+ = 2\text{Al}^{3+} + 3\text{H}_2\text{O}$. Other researches have employed the use on analytical methods such as Atomic Absorption Spectroscopy (AAS) and Inductively Coupled Plasma Mass Spectroscopy (ICP/MS). In the determination of aluminium in foods including titration processes like Complexometric Back Titration. This study therefore, adopts the complexometric Back titration method for the determination of aluminium leached into the food employing the outlined concept of leaching presented below, highlighting on the dependent and independent variables.

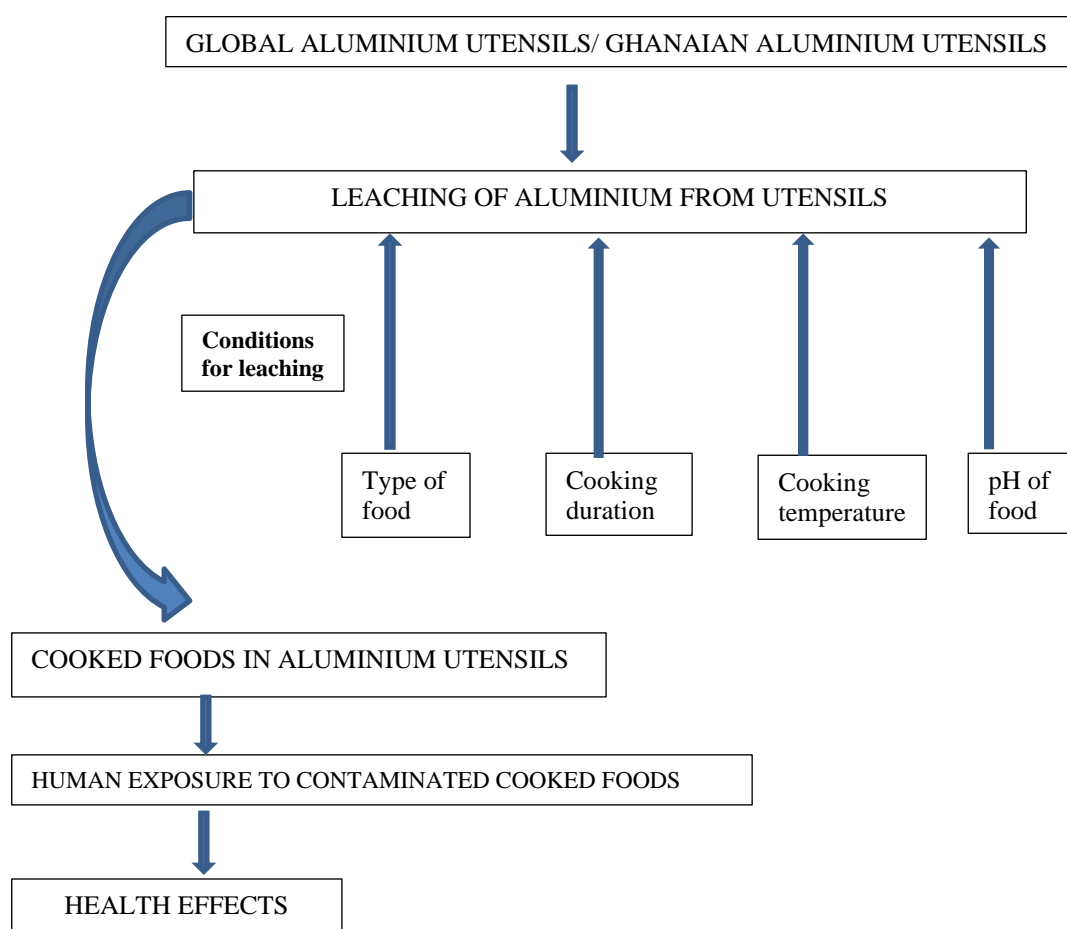


Figure 2: A schematic diagram showing the concept of aluminium leaching from aluminium utensils into cooked foods.

Source: Zheng et al. (2012).

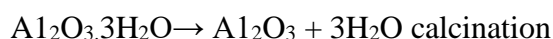
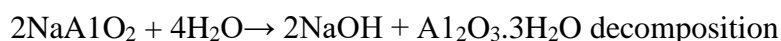
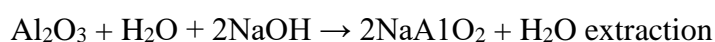
Aluminium metal

Aluminium (Al) is the earth's crust's most copious metal which is extensively distributed representing approximately 8% of its total mineral content (Samwel et al, 2006). It is the third most plentiful element in the universe and the most extensively used metal after steel due to its flexibility (Gupta et al., 2013; Hassan et al, 2008). Aluminium is a metal which has a low weight and appears silvery white. It is a metal obtained from aluminium-containing minerals, predominantly bauxite (Department of Health and Human Services, 2008). Bauxite is transformed to aluminium

(alumina) using the Bayer System. This process produces all the alumina required by the market economy aluminium sector, and its basic features have remained constant since Karl Josef Bayer developed the technology in Germany in 1888 (Macmillan, 2017).

According to Eskin (2008), the global demand for aluminium is estimated to be over 29 million tons per year. There are approximately 7 million tons of recovered aluminium scrap and 22 million tons of virgin aluminium. It is both cost-effective and environmentally friendly to use recycled aluminium. It takes 14,000 kWh to produce 1 tonne of fresh aluminium. On the other hand, remelting and recycling one tonne of aluminium uses only 5% of this. The quality of virgin and recycled aluminium alloys is identical. For foil and conductor cables, pure Al is commonly utilized however, alloying with other elements is required for better strength in some applications. (Eskin, 2008).

Also, with a greater strength-to-weight ratio than steel, Al is one of the lightest engineering metals. By compounding its favourable properties such as strength, lightweight, corrosion resistance, recyclability, and formability, Al is being employed in a growing number of different applications. This group of objects consist of anything from structural resources to lightweight packaging foils (Eskin, 2008). According to (Macmillan, 2017), the following equations can be used to represent the process of aluminium extraction, decomposition and calcination:



Most African households, notably those in Ghana, rely on artisanal kitchen equipment made of aluminium for food preparation. However, even though these containers are frequently used to cook dishes, data on the content of aluminium in food from local kitchen utensils is rare, if not nonexistent. Aluminium was once thought to not affect human health, according to Rajwanshi et al. (1997), and its toxicological report of the evaluation was included in the report of Expert Committee JECFA (FAO/OMS) on additives (WHO Expert Committee on Food Additives, 2007), nevertheless this is no longer the case. Since a transient tolerance of 7 mg/kg of body weight per week has been found for aluminium, collecting and analyzing data on this element appears to be critical.

The aluminium metal have been put to use in different sectors for its vital significance. The ensuing paragraphs discuss what lieterature say about the various use and sources of the aluminium metal and how they have finally found their way into foods

Uses of aluminium

Cooking utensils, food packaging like beverage cans and foil, structural resources for building, vehicles and aeroplanes, and other industrial applications including corrosion-resistant chemical equipment and solid fuel rocket propellants, among others, are all examples of uses for aluminium metal (Alabi & Adeoluwa, 2020). Aluminium compounds including silicate, hydroxide and sulphate are used in a range of applications including treatment of water, processing of food, production of cosmetics (example; antiperspirants), and production of medications (example; antacids) and are also used in industries (WHO, 2003).

Sources of Aluminium Consumption

Food and water: Aluminium is found naturally in drinking water and foods, as well as in many manufactured items. The general public is predominantly exposed to aluminium through food consumption, while modest exposures can also occur through drinking water containing aluminium and breathing ambient air (Department of Health and Human Services, 2008). Niu (2018) is of the view that the amount of aluminium in foods and drinks varies greatly depending on the kind of food, the processing method used, and the geographical areas where food crops are farmed. Small amounts of aluminium can be found in unrefined food items which consist of fresh vegetables, fruits and meat. In certain situations, aluminium complexes may be added to foods such as flour, baking powder, colouring agents, and anticaking agents during processing (El-Sayed & Farag, 2008; Ma et al, 2016).

Aluminium concentrations in natural waters (ponds, lakes, and streams) are typically less than 0.1 milligrams per litre (mg/L). The average person consumes very little aluminium in their drinking water. Water is periodically treated with aluminium salts throughout the process of becoming drinkable water. However, aluminium levels are usually less than 0.1 mg/L. Al concentrations in drinking water have been observed to have reached 0.4-1 mg/L in certain cities (Department of Health and Human Services, 2008). Also, according to (JECFA, 2007), due to the usage of certain chemicals as flocculating agents in the treatment of water meant for human consumption, such as aluminium sulphate and aluminium polychloride, aluminium can be discovered in drinking water. Following

treatment, there is often less than 0.2 mg/L of aluminium in tap water. Therefore, based on a daily intake of 1 L, dietary exposure from treated drinking water may be up to 0.2 mg of aluminium/d, or 0.02 mg of aluminium per kilogram of body weight per day for a child weighing 10 kg.

Medicines: Long-term exposure to aluminium-containing drugs such as phosphate-binders, buffered analgesics and antacids is possible. For someone who takes aluminium-containing medications daily, the amount of aluminium they are exposed to from medications could be much more than from their meals (WHO, 2007).

Parenteral nutrition: Although parenteral nutrition is not the same as oral nutrition of aluminium absorption, it can be used as a dietary substitute for people who have stomach problems. In 1992, researchers discovered that patients receiving parenteral nourishment had higher average plasma aluminium concentrations (0.59 NjM) than those who did not (Thakur, McMillan & Jones, 2014). Furthermore, aluminium concentrations in the blood were greater in babies who received parenteral nutrition (37 Njg/L) than in those who did not (5.2 Njg/L) (Landry, 2014). Many institutions changed their practices after studies revealed that the normal casein hydrolysate solution had more aluminium than similar lipid solutions (Carpenter, Arcaro, & Spink, 2002). Many nations have imposed limits on the amount of aluminium in parenteral solutions as a result of these findings. The US Food and Drug Administration, for example, allows a maximum of 25 Njg Al/ L in substantial size parenteral (Thakur, McMillan, & Jones, 2014). Parenteral nutrition polluted with aluminium has been linked to increased stainable bone aluminium and osteomalacia (Ott et al.,

1983). Al build-up in the liver and portal inflammation caused hepatobiliary dysfunction in rats given a parenteral feeding formula containing the metal (Alemmari et al., 2012). Because neonates have a poorer ability to excrete aluminium than the general population, more research on the pharmacokinetics and dynamics of aluminium should be prioritized (Willhite et al., 2012).

Aluminium from food contacts material: Apart from the sources of Al for human consumption described above, one of the most often used possible sources of increased dietary Al is Al cookware. Cook wares such as pressure cookers, roasting pans, Al skillets, saucepans, pots, foils, wrappers and trays could enhance the quantity of Al in foods. The type of food being processed has a significant impact on the utensils and materials used to make them (Levick, 1980; Trap & Cannon, 1981). Although the primary purpose of utensil design is to satisfy function, safety and hygiene requirements must also be considered. Food preparation and processing utensils must not directly or indirectly transmit toxic compounds, and they must be resistant to corrosion by food, detergent, and other chemicals. They must have no offensive odour, colour, or taste, and their surfaces must be free of pits and fissures to avoid being a source of contamination (Moerman & Partington, 2014).

In the production of food-contact utensils, aluminium and aluminium alloys are often used. Kitchenware is made from a variety of materials, including aluminium, stainless steel, synthetic wood, cast iron, steel, and plastics (Hauser et al., 2004). Before the latter part of the eighteenth century, aluminium products were considered luxury items;

nevertheless, as the cost of production fell, the range of applications grew considerably. Drinking and eating utensils made of aluminium as well as cooking pots and pans, were developed in the early 1900s due to the material features indicated above. Since the end, the military has been the first to embrace it for it to be used as dining utensils, pans, and canteens. As a result of modern food preparation, storage, and packaging procedures, the use of aluminium in food items has increased (Moerman and Partington, 2014). Today, without aluminium, the food industry would be impossible to envisage.

Concerns have been expressed about Al contamination of cooking equipment (Levick, 1980; Koning, 1981; Trapp and Canon, 1981). Pennington (1987) discovered that certain foods have a deleterious effect on Al in certain cook wares. Aluminium in food packaging is becoming more common and accepted (for example, pan masala, pan mixes, drinks, tea, toothpaste, snacks, and hot meals provided on trains and airlines). The type of Al utensils used, their prior use, the pH of the meal, its form and content, the length of time it was cooked, and the existence of salt, sugar, and other ions such as F, Cl, CO₃, and others have been considered as conditions that affect the leaching of aluminium in contact with foods (Jabeen et al, 2016).

Occupational exposure: When compared to people who aren't exposed to aluminium at work, those subjected to the metal in working environments where aluminium welding is done, when aluminium is produced through electrolysis, or in processing industries can have significantly higher internal exposure levels. As a result in these employees, the reference levels

calculated for the general population may be surpassed. Al levels in blood and urine were found to correspond with welding fume aluminium content in longitudinal studies of aluminium welders (Kiesswetter, 2007). The median plasma values, which range from 10 to 14 g/L, are considerably lower than the plasma concentration of 50 g/L, which is thought to be the toxicity limit in people receiving dialysis (Kiesswetter, 2007).

In longitudinal investigations conducted over a 5-year course of study, aluminium welders had average post-shift aluminium level of concentrations of 120 g/L and 13 g/L plasma, compared to industrial employees who were not subjected to the aluminium metal. These alterations were the first group-based subclinical alterations discovered by neuropsychological tests. When early-stage aluminosis was found in aluminium powder production workers, their aluminium concentrations were substantially greater than controls' at 340.5 g/L and 33.5 g/L, respectively (Kraus et al., 2006).

Therapeutic Applications of Aluminum

Antiperspirants

Commercial antiperspirants have contained aluminium compounds as early as 1903. Aluminium salts are utilized in dermatology at concentrations that are substantially higher than those found in antiperspirants sold over the counter due to their antiperspirant effects. These are regarded by the German Dermatological Society (Deutsche Dermatologische Gesellschaft) as a straightforward, suitable, and side-effect-free therapy choice for hyperhidrosis (Zouboulis et al., 2012).

Despite the fact that aluminium is absorbed via the skin (Guillard, Fauconneau, Olichon, Dedieu & Deloncle, 2004), the penetration rate of aluminium chlorohydrate after the dermal application of antiperspirants is incredibly low at just 0.01% and can reach 0.06% in pre-damaged skin. There are no epidemiological research on the use of antiperspirants after shaving the underarms or the use of hair removal products that examine internal exposure.

Vaccination and hyposensitization

Aluminium salts are utilized as active ingredients in the preparations of hyposensitization and vaccines. The immunological effect is enhanced by the assimilation of antigens on weakly permeable aluminium hydroxide. One-time administration of a vaccination recognized in Europe results in absorption of an aluminium prescription of 0.1-0.8 mg (Weisser, Heymans & Keller-Stanislowski, 2015). The dosage of aluminium hydroxide in hyposensitization products authorized for the German market ranges from 0.1 to 1.1 mg. Aluminium exposure from these products is substantially higher than from a single immunization because they are typically given monthly over a three-year period.

After injection, the aluminium salts enter the body; the potential hazards of this are currently the focus of intense debate. The institute of Paul-Ehrlich deemed the “contribution of therapy with aluminium-containing therapeutic allergens to the lifelong buildup of aluminium in the organism as low” and deemed it suitable in light of the beneficial advantages in 2014 (Weisser, Heymans & Keller-Stanislowski, 2017). However, there is a paucity of information regarding the blood or urine

levels of afflicted patients, which would allow for a risk assessment of aluminium's potential for subclinical neurological effects.

Toxicological Effects of Aluminium on Humans

Aluminium consumption can put people's health in danger. Aluminium is said to interfere with a variety of living mechanisms that occurs in the human body. The actual mechanism of aluminium harmfulness, on the other hand, remains unknown. Aluminium is known to be potentially harmful to cells and the nervous system (Stahl, 2017). It's possible that enzyme activity could be interrupted, and mitochondrial function will be compromised. Aluminium has the potential to cause oxidative stress (Kumar & Gill, 2014). Aluminium may have a deleterious impact on three organ systems in particular: the hemopoietic system, the neurological system, and the bones. Aluminium has also been linked to the development of disorders like Alzheimer's dementia and breast cancer (Stahl, 2017).

Some of the diseases that have been linked to aluminium include; softening of the bones known as osteomalacia, aluminosis, anaemia and Hemodialysis encephalopathy (Stahl, 2017; Becaria, Campbell & Bondy, 2002). Aluminium toxicity, which manifests as encephalopathy and osteomalacia, is of special concern. Individuals who are suffering from chronic kidney disease and are also on dialysis or individuals who consume phosphate binders which have aluminium for long durations may have these side effects (Stahl, 2017). Increased aluminium levels in the brain of patients who are on dialysis have been linked to dialysis encephalopathy. The dialysis fluid contains an aluminium component to effectively remove

phosphate from the blood. Aluminium appears to build in the human system and the human brain after which, patients may experience behaviour disorders, enhanced dementia, and melancholy moods as a result of aluminium toxicity (Landry, 2014). Also, the human skeletal structure has been mentioned as a probable focus of aluminium intoxication (Becaria, Campbell & Bondy, 2002).

Long-term exposure to relatively substantial amounts of aluminium, such as taking aluminium-containing antacids, can produce a disproportion in the calcium and phosphate systems, resulting in bone weakening (Spencer et al., 1982). Significant aluminium exposure has been linked to a delay in the growth of bones and also causes anaemia (Landry, 2014). Anaemia caused by aluminium is often macrocytic and hypo chromatic, and it is common in patients who are on dialysis. The specific cause of this occurrence is still unidentified (Landry, 2014). Aluminium lung, also known as aluminosis, is an occupational disease that affects employees who are unprotected from the metal aluminium and aluminium oxide dust and fumes.

Aluminosis is the most well-known of all the aluminium-related health hazards (Schmidt & Grunow, 1991). Aluminium compounds build up in the lungs, impairing the lungs' self-cleaning function. Furthermore, they may trigger inflammatory responses that cause irreparable damage to the tissue in the lungs and respiratory system, leading to fibrosis (Jederlinic et al., 1990). Aluminium's role in breast cancer and the growth of Alzheimer's disease cannot be conclusively proven or refuted due to insufficient and disputable facts. There has been some indication of a link

between aluminium and breast cancer, based on studies indicating that the tissue in the breast of women with tumours may have greater aluminium contents than breast tissue from healthy women to some extent (Darbre, 2001).

Aside from improved detection tools for breast cancer, aluminium-containing antiperspirants and deodorants may potentially have a role. These compounds might contribute to the development of cancer in the breast since they are used so close to the tissue of the breast (Darbre, 2003). It should be mentioned, however, that the research on this subject is conflicting, making it impossible to draw scientifically sound conclusions about aluminium-containing antiperspirants or deodorants and breast cancer. However, in response to this contentious issue, aluminium-free antiperspirants have become more widely available and accessible on the market.

Also, Alzheimer's disease is a disease that causes cognitive problems such as memory loss and loss of direction (Stahl, 2017). Increased aluminium concentrations in particular parts of the brain have been reported (Exley & Vickers, 2014). Aluminium's significance in the development of Alzheimer's disease, like its position in the development of breast cancer, is hotly debated. The neurotoxic potential of aluminium, as well as the likelihood of aluminium bound to transferrin entering the brain, have been hypothesized as probable mechanisms. There is no proof that aluminium is a single cause of Alzheimer's disease, but it may operate as a co-factor that aids in the illness's progression (Stahl, 2017).

Aluminum as a Breast Cancer-causing Factor

The question of whether using antiperspirants that include aluminium can cause breast cancer has been debated for a while (Darbre, 2003). The upper outer quadrants of the breast, which are located near where the chemicals are utilized and where tumors are more frequently detected, also include more glandular tissue (Darbre, 2005). But in recent decades, a rise in this localisation has been noted (Darbre, 2005). However, study of 746 successive breast tissue samples revealed that the proportion of healthy, abnormal, or cancerous tissue change classifications was the same across four dimensions (Klotz et. al, 2017).

Aluminium concentrations were found to be higher in the outside than the inner quadrants of breast tissue in both an examination of malignantly altered tissue and the nipple aspirate fluid from female breast cancer patients (Darbre, 2005). Aluminium, like other minerals, appears to accumulate more heavily in tumor tissue than it does in normal tissue. For instance, when given to rats, 2,7-dimethylbenz[a]anthracene, a compound that is carcinogenic but does not contain aluminium, caused mammary gland tumors with significantly elevated aluminium levels (Darbre, 2005). Moreover, human breast tumor tissue samples revealed considerably greater amounts of other minerals compared to aluminium, such as Cd and Ni, as well as Br, Cl, Zn, Co, Mn, Fe, K, Rb, Na and Ca. Antiperspirant use raises the incidence of breast cancer, according to Darbre's (2005) research.

A study found that prolonged subjection to aluminium chloride altered breast epithelial cells in vitro, causing tumor growth and metastasis in an animal experiment (e.g., through enhanced DNA synthesis and DNA

double-strand breaks). It is believed that this demonstrates cell transition. Breast cancer patients who had combined the use of aluminium-containing deodorants and underarm shaving showed a younger age of disease onset, according to retrospective research (McGrath, 2003), while case-control studies (Fakri, Al-Azzawi, & Al-Tawil, 2006) did not. According to a thorough evaluation of the research, using antiperspirants does not seem to increase the risk of breast cancer (Namer et al., 2008).

Implications of Aluminium on Human Memory Impairment

Since 1921, there has been a connection between Al toxicity and memory problems in humans (Spofforth et al., 1921). Later evidence showed that providing Al to experimental animals intravenously led them to experience convulsions (Chusid et al., 1951). Al is known to be a cause of numerous hemodialysis-related illnesses, such as osteomalacia, microcytic anemia, 2-microglobulin-associated amyloidosis, and dialysis encephalopathy in patients receiving hemodialysis (Wills & Savory, 1989). Alfrey, LeGendre, and Kaehny 1976. Over 20,000 people were exposed to excessive levels of aluminium in Camelford in 1988 as a result of drinking water being unintentionally poisoned with aluminium (Cornwall, UK).

Residents exposed to polluted Al showed a variety of symptoms associated with cerebral impairments in a 10-year follow-up study, including loss of focus and short-term memory (Altmann et al., 1999). In England and Wales, Martyn et al. (1989) found that areas with significant Al drinking water contamination had a higher incidence of Alzheimer's disease (AD). Numerous studies have since produced findings that suggest a connection between AD and Al in drinking water (Flaten, 2001). Frecker

wrote about a region of Norway where there was a connection between a high rate of dementia deaths and high levels of aluminium in the water (Frecker, 1991). Neri and Hewitt observed a positive correlation between Al in drinking water and the incidence of AD in Canada (Neri, 1991). Forbes and McLachlan reported a higher prevalence of AD in Canadian locations with high aluminium and low fluoride levels (Forbes & McLachlan, 1996). Rondeau et al. found that a high daily intake of Al was linked to an increased risk of dementia or cognitive decline in a French cohort study with a 15-year follow-up (Rondeau et al., 2000). According to these researches, when Al penetrates the brain, it has a negative impact on people's memories and contributes to dementia.

Effects of Al In Vitro or in Vivo on the Central Nervous System

Despite being prevalent in the environment, Al is not a component that all living things require, and no enzyme function requires Al. There are around 200 biologically relevant processes in the mammalian central nervous system that Al is thought to interfere with negatively (CNS). Among the crucial procedures for brain development in this area are axonal transport, neurotransmitter synthesis, synaptic transmission, protein phosphorylation or dephosphorylation, protein degradation, gene expression, and inflammatory responses. Al always displays Al^{3+} as its oxidation state. Al^{3+} has affinity for negatively charged, oxygen-supplying ligands. Al^{3+} binds firmly to carboxylates, deprotonated hydroxyl groups, and organic and inorganic phosphates.

These chemical characteristics of Al^{3+} cause it to bind to DNA and RNA phosphate groups, changing DNA architecture and controlling the

expression of several genes essential for brain function. Lukiw et al. found that nanomolar concentrations of Al^{3+} were sufficient to alter neuronal gene expression (Parhad, Krekoski, Mathew & Tran, 1989). Al^{3+} can modify how energy is utilized by attaching to the phosphate groups of ATP and other nucleoside di- and triphosphates. Numerous protein kinases and phosphatases cannot function as a result of Al. The ligand-exchange rate of Al^{3+} is rather low when compared to other metals. Al^{3+} for example, inhibits enzymes with Mg^{2+} cofactors because Mg^{2+} has a 105-fold higher rate of ligand exchange than Al^{3+} .

Al^{3+} also blocks biological activities involving rapid Ca^{2+} exchange since it exchanges at a rate 108 times slower than Ca^{2+} . Due to these features, Al is ineffectual in enzymatic reactions and has a longer half-life in the human body. Strong positive charges and a small ionic radius distinguish Al^{3+} from other metal ions including Ca^{2+} , Zn^{2+} , and Na^{+} . Al^{3+} acts as a cross-linker by aggressively attaching to amino acids that bind metals, such as histidine (His), tyrosine (Tyr), arginine (Arg), and others, as well as to amino acids that have been phosphorylated. This property makes Al^{3+} an efficient tanning agent for leather. Al may attach to various proteins, oligomerize them, change their structure, and stop proteases from degrading them. The self-aggregation and accumulation of highly phosphorylated cytoskeleton proteins, such as neurofilament and microtubule-associated proteins (MAPs), are encouraged by the joining of Al^{3+} to phosphorylated amino acids (Diaz-Nido & Avila, 1990).

As a result, Al causes glial and neuronal cells to undergo apoptosis. Chronic Al treatment reduces long-term potentiation (LTP), a synaptic

information storage mechanism that is well-known as a paradigm of memory systems. Al also has an impact on various enzymes, including those responsible for producing neurotransmitters, which has an effect on the quantity of neurotransmitters. Al^{3+} also inhibits synaptic transmission, neurotransmitter receptors, and voltage-gated Ca^{2+} channels. Finally, Al disrupts a variety of learning and memory-related brain activities, affects emotional response, and creates a deficit in spatial memory. The mechanisms underlying Al-induced memory impairment may include several negative effects (Martyn, 1989).

Neurotoxicity of aluminium

Aluminium (Al^{3+}), which may cross-link proteins, has a great affinity for proteins. Unlike other metals such as zinc, iron and manganese, that are widely prevalent in the body, aluminium is not identified to serve a physiological purpose in humans (Kawahara & Kato-Negishi, 2011). Clinically, substantial neurotoxic consequences have been recorded in patients undergoing dialysis. Aluminium ions were discovered to be the culprits; they had previously been included to the dialysis solution as a phosphate adhesive (Parkinson, Ward & Kerr, 1981). Aluminium levels in patients' blood and brain tissue were greater (Parkinson, Ward & Kerr, 1981). Affected individuals demonstrated bewilderment, memory problems, and, in more severe cases, dementia (Parkinson et al., 1981). The main reasons of these side effects are the biological processes that aluminium in the brain impacts, which are numerous, and the slower clearance of aluminium from the brain compared to other organs (Kawahara & Kato-Negishi, 2011).

Consequently to producing peroxidation and adhering to oppositely charged structural components in neurons, aluminium has the ability to change hippocampal calcium signal pathways that are essential for neuronal plasticity and subsequently, for memory. Cholinergic neurons are predominantly vulnerable to aluminium contamination, which interferes with the production of the neurotransmitter acetylcholine. The last two neurological consequences are of special importance to the Alzheimer's hypothesis, which suggests a connection between aluminium exposure and Alzheimer's disease (Kawahara & Kato-Negishi, 2011). Neurotoxic effects of aluminium might be partially reversed once aluminium contamination was removed from the dialysate (Parkinson et al., 1981).

After occupational exposure of workers to concentrations of approximately 100 g of aluminum per gram of creatinine and approximately 13 g per liter of plasma, neuropsychological tests pertaining to concentration, learning, and memory, for example, changed; in this case, the neurotoxic effect of aluminum is considered causal (Riihimäki et al., 2000). Even with aluminium exposure over this level, no cases of manifest encephalopathy, which includes disorientation, memory loss, and dementia, have been recorded (Riihimäki et al., 2000).

Absorption, distribution, metabolism, and excretion of aluminium

Most individuals don't think twice about how much aluminium they are exposed to every day. The fact that aluminium is present in food sources may have an impact on how we interpret the phrase "exposed to aluminium." In some ways, this statement is true, but it is important to also take into account the other points. The majority of oral consumption of

aluminium comes through food, which makes up around 95% of daily food intake, and water, which makes up 1-2%. These typically supply 4,000–9,000 micrograms of daily consumption (Yokel, Hicks & Florence, 2008). The oral intake may be increased by taking antacids by up to 5,000,000 micrograms. Al may enter the human body through breathing at a rate of 4 to 20 micrograms per day. Last but not least, aluminium may enter the body more easily after being exposed to deodorants with aluminium-containing components by up to 50,000–75,000 micrograms per day. This amount is augmented by inhalation up to 25,000 micrograms in industrial settings (Yokel et al., 2008).

Immunotherapy for allergic disorders employ adjuvants made of aluminium. Aluminium salts are used in over seventy percent of adjuvant-based therapies. Manufacturers vary in how much aluminium salts are included in their vaccines; for instance, low-dose diphtheria vaccines have 170 micrograms of salts per dosage, whereas 850 micrograms of salts are included in each dosage of the high-dose haemophilus influenza type b vaccine (Exley, 2014). A person may be exposed to aluminium intravenously if, for example, their dialysis solution or total parenteral feeding solution contains aluminium, in essence aluminium toxicity is caused by the buildup of aluminium (Yokel & McNamara, 2001).

Other studies also revealed that, only approximately 1 percent of the aluminium consumed through food is absorbed using these oral mechanisms of metal absorption into the human body (Landry, 2014). Although it is well acknowledged that aluminium in drinking water is more easily absorbed, it accounts for a significantly smaller portion of daily

intake (5%) in comparison to food (95%) (Krewski, 2007). Within the human body, factors like the kind of aluminium compound consumed, the food consumed, the age and state of health of the person consuming the goods, particularly renal function, all contribute to the different amounts of aluminium absorption (Krewski, 2007). Increased intestinal absorption has been found in the elderly and immunocompromised people in several trials, raising concerns regarding toxicity in these groups (Ihnat, 2001).

Aluminium and calcium may have absorption mechanisms in common (Greger & Sutherland, 1997). In the presence of calcium channel blockers, rats in research conducted by Cochran, Goddard, and Ludwigson (1990) exhibited a modest decrease in aluminium uptake (Greger, 1993). However, their findings say that “the slight reduction in the rate of Al absorption which was observed cannot safely be assigned to direct closure of calcium channels to Al” because of elevated amounts of verapamil, a calcium link revocation drug employed in the investigation (Cochran et al., 1990).

However, it is claimed that aluminium enters the bloodstream in two ways: passively by diffusion and actively through transferrin-mediated pathways (Hoffman et al., 2014). Antiperspirants and deodorants actively contain aluminium salts like aluminium chlorohydrate (ACH). They work by accumulating in the eccrine sebaceous glands and aluminium hydroxide that is impermeable. Blocking sweat secretion and causing sweat plaques to relax (Flarend et al., 2001). Patients exposed to vapors containing aluminium have pulmonary absorption and respiratory uptake rates of 1.5–2%, respectively (Hoffman et al., 2014).

In furtherance, this investigation did establish that aluminium uptake is energy-dependent (Cochran et al., 1990). Citric acid has been shown to affect aluminium absorption. Higher levels of urine and serum aluminium were discovered in patients using citrate-containing medications with aluminium-containing pharmaceuticals (Greger, 1993). Greger proposes three explanations for the increased absorption of aluminium: first, citrate may rise aluminium solubility in the stomach, making it easier to absorb; second, citrate may help to co-transport aluminium into mucosal cells; and third, citrate may open epithelial tight junctions, allowing aluminium to pass out of the intestinal lumen (Greger, 1993). Aluminium's circulation is best recognized as collecting primarily in the bones and lungs (Krewski et al., 2007).

In a typical setting, the serum aluminium concentration is virtually the same as the total blood aluminium levels. A little less than 1% of plasma aluminium is bound to phosphate and hydroxide, less than 7% to citrate, and transferrin is a binding site for over 90% of the aluminium in plasma. In the body, aluminium is mostly stored in the bones, the lung, the muscle, the liver, and the brain, accounting for around 60% of the total (Hoffman et al., 2014).

Soft tissues, the spleen, liver, kidney, nerve tissues, muscles, and the heart are also damaged typically after intravenous fluid contamination (Greger, 1993). The kidney is where more than 95% of aluminium is eliminated. Actually, only around 2% of aluminium is excreted through bile, with the kidneys being the primary organ through which it is removed from the body. Normal circumstances allow healthy persons to eliminate all

ingested Al. However, when the human body comes into contact with large doses of the aluminium metal, such as during complete parenteral feeding, some of the absorbed aluminium may accumulate because it cannot be eliminated in an initial kinetic profile. The protein binding that restricts aluminium ultrafiltration's ability is one of the causes of this buildup. Depending on the specific exposure, quantity, frequency, and route, aluminium clearance varies. Although the majority of the Al that is absorbed is eliminated within the initial exposure period, the excretion process is thought to take anything from a few hours to a few years (Krewski, 2007).

However, the tissues most affected by orally consumed aluminium include the bones, liver, and blood (Greger, 1993). Currently, it is unknown how aluminium is metabolized in the body once it has been ingested. The most prevalent methods of excretion are bile and the kidneys, which together account for 95% of elimination (Krewski et al., 2007).

Mechanism of aluminium poisoning

The third most prevalent and pervasive element in the planet is aluminium. It can be found in water, soil, and air in nature. Additionally, due to this substance's GRAS (generally recognized as safe) classification from the FDA, it is legal to add it to several foodstuffs and drugs. Current research on ecological toxicity has revealed that Al poses serious risk to people, animals, and plants (Barabasz et al., 2002). Oxidative stress is caused by aluminium overdose and affects the kidney, liver, and brain. Free radical production can be increased and alter the enzymes' ability to act as antioxidants. Any contact with aluminium can alter the operation of a

number of enzymes, alter how proteins are made, how nucleic acids work, and how permeable cell membranes are. Additionally, it may have an impact on the body's metabolism of triglycerides and their plasma levels (Shati & Alamri, 2010).

Aluminium's prooxidant characteristics can result to biological oxidation both in vivo and in vitro. Al causes lipid peroxidation by promoting glutathione (GSH) depletion, glutathione peroxidase (GSH-Px), glutathione S-transferase (GST), and catalase (CAT) activity in renal tissue (Mahieu, 2005). Aluminium builds up in renal tissue, which causes renal-tubular cells to degenerate, alters cellular metabolism, and induces oxidative stress, all of which result in salt and water imbalances, altered p-aminohippuric acid transport, and ultimately, renal toxicity (Mahieu, 2005).

The primary protein compound in the P-type ATPase family found in all living things is sodium-potassium ATPase. It contributes to the preservation of the cell's homeostasis and gradient both inside and outside the cell. Na^+/K^+ ATPase is inhibited by aluminium both in vitro and in vivo (Therien & Blostein, 2000). Reactive oxygen species (ROS) produced by aluminium cause lipid peroxidation and DNA and protein oxidative damage. It results in damaging alterations in the liver cells and rough endoplasmic reticulum (RER) deterioration, which are connected to a reduction in the synthesis of protein in the liver and may affect cellular calcium levels in cells (El-Sayed, Al-Kahtani & Abdel-Moneim, 2011). Most of the liver cells' mitochondria inflate when exposed to aluminium toxicity, and modifications in the porosity of the mitochondrial membrane can intensify the oxidative circumstances of the mitochondrial functional

group, and this may have an impact on the quantity of free calcium in the cell. Malondialdehyde (MDA) and serum alanine aminotransferase (ALT) movement are increased four times and proinflammatory cytokines like TNF- are increased up to seven times above normal levels as a result of the damage to the liver parenchymal cells (El-Sayed et al., 2011).

Al^{3+} may show up in Fe^{3+} sites because of the similarities between the ionic radii of Al^{3+} and Fe^{3+} . Aluminium is thus linked to the protein that transports Fe^{3+} . This process may reduce the binding of Fe^{2+} , increase free intracellular Fe^{2+} , and cause membrane damage by oxidizing membrane lipids (El-Sayed et al., 2011). While signaling the release of endoplasmic reticulum Ca^{2+} stores, emission of Ca^{2+} activators is aided by endoplasmic reticulum activity. In addition to the depletion of Ca^{2+} storage, the release of Ca^{2+} into the cytoplasm influences fundamental cellular processes such the control of gene expression, cell development, and cell apoptosis (Ribeiro et. al, 2000).

Experimental investigations indicate that aluminium compounds may be neurotoxic because of the production of free radicals and ROS. (Pasha & Oglu, 2017). Different organelles, including mitochondria, lysosomes, and nuclei, are impacted by aluminium compounds (Wu et al., 2012). According to a number of studies, exposure to aluminium boosts the generation of ROS, which causes spillage of electron, increased mitochondrial function, and higher electron-chain activity. The cell's structure and function alter as a result of ROS disrupt to the mitochondria, which is its target component. A variety of cellular processes will result from this, such as a change in mitochondrial activity that lowers the

mitochondrial membrane potential (MMP), a critical component of living cells, including brain cells. Reduced MMP results in lower ATP levels, increased electron leakage, and enzyme activity inhibition in the electron transport chain. The membrane of the nerve cell then depolarizes, and calcium ions begin to enter the system. In general, ROS and oxidative stress disrupt neurotransmission and cellular signaling, which leads to cytotoxicity. This process results in brain injury, neurodegeneration, and neural cell death (Sharma & Mishra, 2006).

Osteomalacia is caused by aluminium buildup in the bone marrow. Increased aluminium levels in the body are linked to fewer osteoblasts and osteoclasts as well as less bone marrow remodeling. A side effect of aluminium's suppression of protein synthesis is that it can prevent the bone marrow from producing hemoglobin. Microcytic hypochromic anemia can be brought on by a decrease in hemoglobin production (Jeffery et al., 1996). Polymorphonuclear leukocyte (PMN) buildup and a dose-dependent inflammatory condition were brought on by aluminium deposition in the lungs of rats. In addition, type II cell hyperplasia and a decrease in alveolar lavageable macrophages have been seen, followed by aluminium entry into the lungs (Cohen, 2004).

DNA, immunological signals, and genetic information are lost in the brains of mammals when aluminium is present due to brain transcriptional activation. Aluminium salts may adhere to DNA and RNA and prevent their synthesis. At different times during the cell cycle, DNA is split into single and double strands to achieve this. Additionally, exposure to aluminium compounds has the potential to alter how stable DNA is and

inhibit DNA repair (Lankoff et al., 2006). Aluminium eventually builds up in human neurons after aluminium loading, which prevents hyperphosphorylation and/or protein phosphatase 2A (PP2A) activity. Tau protein, neurofilament protein, and other proteins may become hyperphosphorylated in neurons if PP2A activity is inhibited. An unbalanced interaction between the phosphatases and the kinases that add to the phosphates that take away from the proteins leads to hyperphosphorylation. The neurons become vulnerable as the recently generated hyperphosphorylated proteins gather there (Walton, 2007).

According to certain animal studies, iron levels in the brain rise when aluminium is present. Receptors for transferrin and ferritin mRNAs are among the iron proteins that make up the structure of the iron regulatory elements (IREs). IRP-1 and IRP-2, two proteins coupled to IRE, function as iron sensors. IRP-1 joins with the iron-sulfur aspect to transform into a state that cannot link to the IRE when iron is employed in the cell, while IRP-2 degrades quickly. According to studies, aluminium may stabilize the IRP-2 by limiting its deterioration, which boosts the manufacture of the transferrin receptor and inhibits ferritin formation. Iron that is bound to transferrin and iron that isn't coupled to transferrin are both removed more quickly from human brain cells when aluminium levels are low. The quantity of iron bound to ferritin also decreases. Increased oxidative stress may be indicated by an increase in the cytoplasmic pool of iron.

However, the defensive cell mechanisms, such as catalase, glutathione reductase, glutathione reductase and superoxide dismutase were unaffected by the elevated iron concentration. While the activity of

superoxide dismutase, glutathione peroxidase and catalase reduced when Al was administered orally, peroxidative damage levels noticeably rose. According to studies, even trace levels of Al in the mind may affect the metabolism of iron there, leading to neurodegenerative diseases (Ward, Zhang & Crichton, 2001). Corticoneuronal apoptosis can be brought on by aluminium, and it's conceivable that the SAPK/JNK signal transduction pathway also known as c-jun N-terminal kinase plays a significant function in this instance (Fu et al., 2003). The transcription factor NF- κ B is also involved in several regulatory pathways and is controlled by a variety of signaling events.. The biological processes that the aforementioned signal transductions are involved in include cell division, differentiation, inherent immunity, inflammation, the growth of tumor cells, the production of ROS, and apoptosis. Some of these biological processes will result in inflammatory neurodegeneration, which will subsequently impair the human central nervous system's ability to function normally (Alexandrov et al., 2000).

Detection tests

Since Al is a naturally common element, serum readings may rise as a result of contamination from outside sources during collection, processing, or analysis. Another interesting discovery is the one made clinically when urine and blood specimens are obtained from an individual who has been subjected to aluminum toxicity (Schifman & Luevano, 2018). In general, individuals on dialysis will have blood aluminum levels of fewer than 10 micrograms per liter or less than 60 micrograms per liter (Wechphanich & Thammarat, 2017). At quantities more than 100

micrograms per liter, toxicity sets in. Additionally, aluminum levels in urine that are less than 55 g/g of creatinine are considered harmless for individuals. It is understood that urine concentrations of 100 g/l are necessary for the onset of neurological problems, while urinary aluminium levels of 4 to 6 mol/L (108 to 162 g/L) constitute a threshold for neurological side effects. The amount of aluminium in one's hair, nails, and perspiration can also be used to diagnose metal intoxication (Ogawa Kayama, 2015).

Management of aluminium poisoning

Patients who have been subjected to aluminium should be protected against further pollution in a variety of methods after being identified. All ambient, intravenous, and oral sources of this metal must be eliminated in order to carry out this action. Patients with occupational asthma (potroom) are advised to switch jobs and are given prescriptions for bronchodilators and corticosteroids. Aluminium deposits are collected using aluminium chelators, which lowers the body's metal load (Crisponi, et al., 2010). The standard levels of aluminium in drinking water and dialysis water in European nations are 10 g/L and 200 g/L, respectively. Some experts claim that the amount of aluminium in the dialysis fluid is less than 2 g/L, and they use a double reverse osmosis technology in their dialysis facilities to support this claim (Crisponi et al., 2010). It has various advantages, including preventing aluminium intoxication from the dialysate to blood and removing aluminium from the plasma through hemodialysis, when a double reverse osmosis device is used to produce dialysate (Crisponi et al., 2010).

In situations of aluminium salt poisoning that are both acute and chronic, chelation therapy is advised. Deferoxamine mesylate (desferrioxamine) (DFO) is without a doubt the only chelator with proven beneficial benefits in the case of aluminium intoxication. The use of DFO is advised by the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI) when dialysis patients' aluminium concentrations are between 60 and 200 g/liter. DFO is prescribed to treat both acute and chronic conditions while reducing expected encephalopathy. A dose of 5 mg/kg of DFO (or occasionally a lesser dose) will be administered to chronic dialysis patients before the anticipated start of the dialysis session by more than an hour. In acute situations, a greater dose of 15 mg/kg/day is also given. Dexferoxamine binds to aluminium to generate the chelate aluminexamine, which is eliminated through hemodialysis or urine. In dementia, osteomalacia, and encephalopathy, use duration varies (Smith, 2013).

One of the chelators, calcium disodium ethylenediaminetetraacetic acid (CaNa_2 EDTA or EDTA), has demonstrated a notable reduction in aluminium toxicity. 500 ml of physiological saline with a 2 g/10 ml EDTA dilution was intravenously infused for around two hours. One trial involved giving the medication once a week for ten weeks, while another used it every two weeks for six to twelve months. The findings demonstrated a correlation between the patients' neurological symptoms' improvement and the level of aluminium in their urine samples' decline (Fulgenzi, Vietti & Ferrero, 2014). Ordog (2005), asserted that effects from aluminium poisoning included sinus infections, persistent weariness, loss of feeling

and movement, ataxia, vertigo, memory loss, and chronic discomfort in aluminium plant workers (Ordog, 2005).

Aluminium toxicity effects on leaves and roots of plants

For plants growing in acid soils, aluminium toxicity is a possible growth-restraining factor in many regions of the world (Foy, 1974). Aluminium poisoning can not have obvious signs and symptoms. The foliar signs of phosphorus (P) shortage in plants are similar (overall stunting, small, dark green leaves and late maturity, purpling of stems, leaves, and leaf veins, yellowing and death of leaf tips). Al poisoning can sometimes manifest as a diminished calcium transport issue or an induced calcium (Ca) shortage (curling or rolling of young leaves and collapse of growing points or petioles). In fact, excessive Al causes signs of iron (Fe) insufficiency in wheat, sorghum, and rice (Clark et al., 1981). Aluminium aids in new root formation and seedling establishment but has no effect on seed germination (Nosko et al., 1988). 2-4 days following the start of seed germination, there was a noticeable inhibition of root growth (Bennet, Breen & Fey, 1991).

According to Vanpraag & Weissen (1985), plant species and ecotypes growing on acidic soils had developed a high level of resistance to the effects of aluminium on root growth and absorption during time and phenological evolution. Inhibition of root growth in plants is the main sign of Al poisoning (Bennet, Breen & Fey, 1991). More cellular damage is visible in the roots than in other plant components (Rincon & Gonzales, 1992). Al poisoning was seen in the root system, especially in the root-tips and lateral roots, which swelled and turned brown (Kinraide, 1992). The entire root system has a corraloid appearance and has a lot of lateral roots

that are stubby but lack fine branching (Foy, Chaney & White, 1978). The availability of particular monomeric species of Al to the plant roots seems to be what determines the toxicity (Bartlett & Riego, 1972). As the pH and Al concentrations increase, polymerization of Al may cause losses of phytoactive, monomeric Al, leading to complex formation or chelation with phosphate and organic acids (Bache & Sharp, 1976; Bartlett & Riego, 1972).

According to Wagatsuma et al. (1987), aluminium affects the root cells of different crops. They found that the outer cortex and epidermis cells of maize (an Al-sensitive plant) were damaged in the area just 1cm from the root tip, and that the walls of these cells in barley (a plant highly sensitive to Al) were abnormal and partially detached; more pronounced abnormality and detachment of the cell walls affected almost the entire cortex, and few cortex cells remained alive in oats (an Al-tolerant plant), after 6 days of exposure to the Al treatment. Additionally, they stated that modest levels of Al treatment caused the roots of pea plants to grow longer. Large amounts of aluminium were taken up by the root's tip. In the tip part, the K content dropped as the Al level rose, but the Ca content remained essentially constant. According to Bennet et al. (1985), an anisotropic growth response of cortical cells with a 20-hour root exposure to Al were linked to the disintegration of the outer cells of the root as well as the collapse of the conducting tissue of the stele.

Aluminium interference with Ca, Mg and P

Inhibition of root growth and disruption in root structure, particularly cell wall loosening and secretory activity due to the lack of or

reduction in Ca transport (Edwards, Horton & Kirkpatrick, 1976) and disruption of cellular Ca^{2+} homeostasis, are just a few of the advantages of Ca on plants grown in environments where Al is toxic (Kinraide, Ryan & Kochian, 1992; Huang, Grunes & Kochian, 1992). Most of the mineral elements are affected by Al, and this interference with their absorption, transport, and use efficiency is well known (McColl et al., 1991). According to Huang et al. (1992), Al^{3+} significantly reduced net calcium inflow at the root apex. Additionally, the Ca^{2+} flow was impacted more than the fluxes of the other ions and Al^{3+} decreased Ca^{2+} flow in barley, according to Nichol and Oliveira (1995). One of the most prominent signs of Al toxicity was callose buildup at the root apex (Nichol et al., 1993). Higher cytosolic calcium was usually correlated with increased callose production (Zhang, Hoddinotto & Taylor, 1994).

According to Rhue & Grogan (1976), nutrient solutions with higher Ca concentrations had less variation in Al tolerance between maize inbred lines. In the root of peas, aluminium significantly raised the redox potential, decreased the amount of high-bond energy phosphorous, and increased the amount of mineral P. (Dedov & Klimasshevskii, 1976). Using a variety of plant species, DeGraaf et al. (1997) reported the interaction of Al with minerals. The intake of minerals was affected by high Al concentrations in nutritional solutions; Al frequently interfered with the uptake of divalent cations, especially Ca and Mg (Delhaize & Ryan, 1995). Because of aluminium's interference with P uptake, plants cultivated in nutrient solutions or on acidic soils may lack P (Foy & Brown, 1964).

Al toxicity was linked to a decrease in Ca concentrations in soybean tops and roots (Foy, Fleming, and Armiger, 1969), and Mg concentrations decreased in sorghum with high Al concentrations. According to Clarkson and Sanderson (1971), surface interactions involving the charge on the Al^{3+} ion were the main focus of Ca absorption. Al stress often reduced the concentration of numerous mineral elements, including Ca, Mg, and P, in addition to decreasing plant development. Al stress in Tatum subsoil lowered P and Ca in both Al tolerant Dayton and Al-sensitive Kearney barley cultivars grown under both low and adequate soil moisture condition, according to Krizek & Foy, (1988). Al-damaged plants typically collected Al, P, and Fe in their roots but not in their shoots, and Al stress led to P and Fe deficiency. The displacement of Ca and Mg from the roots by Al and the decreased uptake of Ca, Mg, and P from deeper soil zones by beech and other trees were associated with aluminium injury.

According to Wheeler and Dodd (1995), Al poisoning caused variations in the chemical concentrations and physical symptoms of monocotyledons and dicotyledons. According to Keltjens & Tan (1993), Mg was superior to Ca at reducing Al stress in monocotyledons, whereas the opposite was true for dicotyledons. The effects of manganese and aluminium on development and metal buildup in *Triticum aestivum* were described by Blair & Taylor (1997). Additionally, they showed that when the supply of Al increased, the buildup of Mn in roots and shoots considerably decreased. The effect of Al and Ca on Geraldton wax flower pollen germination and tube growth was described by Zhang et al. (1999). They noticed that trivalent cations including Al^{3+} , La^{3+} , and Gd^{3+} in

micromolar concentrations hindered pollen germination. Rapid tip bursting was caused when the developing pollen tubes were exposed to micromolar concentrations of Al^{3+} and a millimolar concentration Ca^{2+} chelator (ethyleneglycol-bis(beta-aminoethyl ether)-N, N'- tetraacetic acid). By raising either the pH of the solution from 4.5 to 6.0 or the Ca^{2+} concentration from 0.25 to 5 mM, the Al^{3+} treated pollen tube bursting was considerably decreased.

Aluminium as a promoter of plant growth in acidic soil

Low levels of Al boosted growth in citrus and cereal plants (Pilon-Smith et al., 2009). Al has primarily been observed to improve plant development in plants that are native to or have acclimated to acidic soils, such as *Melaleuca cajuputi*, *Arnica montana*, *Deschampsia flexuosa*, and *Hydrangea paniculata* (Pilon-Smith et al., 2009). In Australia, Polynesia, and tropical Asia, *Melastoma malabathricum* L. is a common woody species that collects aluminium and thrives in soil that is low in nutrients and acidity (Osaki et al. 1998). Aluminium increased the amount of dry matter, nutrient absorption, and white, fine, metabolically active roots while decreasing the amount of lignin in roots (Watanabe et al. 2005). Additionally, *M. malabathricum* was shown to grow abnormally when grown for extended periods of time without Al, such as rolling of the leaves. Even though *Miscanthus sinensis* is not an Al accumulator, it is a common species of grass in temperate climates with acidic sulphate soil, and Al supply helps it grow (Kayama 2001).

According to Malkanthi et al. (1995), cowpea showed growth improvement when Al was sprayed at pH 3.8 as opposed to pH 5.5, showing

that Al can help plants develop to some extent. When Al was given to a quartz culture in acidic circumstances, *Eucalyptus gummifera*'s growth multiplied by five over the course of five months (Mullette 1975). Numerous investigations discovered that Al could produce morphological changes in the roots of *Camellia sinensis*, *M. sinensis*, *Quercus acutissima*, *M. malabathricum*, *Eucalyptus viminalis*, and *Symplocos paniculata*, including white colouring and elongation (Ghanati et al. 2005). Other plants, such as *Quercus serrata*, *C. sinensis*, and *Betula pendula* (Kidd and Proctor 2000), showed similar advantages of Al on growth (Xu et al. 2016). Al also lengthened central cap cells, which promoted root elongation in canola. Recent studies have shown that applying Al benefits essential commercial crops like rice and corn (Wang et al. 2015).

Empirical Review

Findings of other researchers on the contamination of foods when cooked using aluminium cooking utensils under the objectives of this study have been presented below.

Determine the concentration of aluminum in selected raw and cooked foods

In a study, Ertl and Goessler (2018) investigated aluminium concentrations in uncooked foodstuff as well as the possible aluminium transfer from aluminium foil into foods. The results of the study showed that uncooked food is not significantly contaminated with aluminium. Furthermore, short time contact to aluminium foil increases the food aluminium concentration only marginally. Nevertheless, as soon as the food is in contact with aluminium foil and at the same time in contact with metals

(alloys) with a higher standard electrode potential than aluminium high aluminium concentration were observed.

Stahl, Taschan and Brunn (2011), conducted a study where analysis of aluminium content of a number of foods and food products was undertaken in order to evaluate the nutritional intake of aluminium. A total of 1,431 samples were analysed within the scope of this study; 65 flours, 37 baking premixes, 107 breads, 60 loaf-shaped yeast fruit cakes, 38 pastries in aluminium trays, 185 salted pretzels and similar savoury biscuits, 24 pastas, 12 herb-teas, 37 cocoa powders, 84 chocolates, 115 confectioneries, 50 malts, 237 beers (177 bottled beers, 40 draught beers and 20 canned beers), 59 fruit juices and fruit juice beverages, 65 wines and fruit wines, 31 ready-cooked meals in aluminium trays, 16 soups from the catering trade, 171 mineral waters and spring waters and 38 diverse products were analysed. According to this study, of all the samples, 77.8% had an aluminium concentration of less than 10mg kg⁻¹. Of the samples, 17.5% had aluminium concentrations between 10mg and 100mg kg⁻¹ and only 4.6% of the samples, had aluminium concentrations greater than 100mg.

Examine the conditions (temperature, pH and cooking time) that may facilitate the leaching of aluminium into cooked foods.

As previously stated, potential sources that expose individuals to aluminium are drinking water, additives added to foods and aluminium cooking equipment (Zendehboodi, 2018). The usage of utensils made of aluminium metal and foils in the kitchen is not regarded to be harmful (Ranau, Oehlenschläger, & Steinhart, 2001). However, other studies propose that it could prime to harmful aluminium metal levels in people

(Weidenhamer et al, 2017). Aluminium utensils and foils are not considered dangerous in the kitchen (Ranau, Oehlenschläger, & Steinhart, 2001).

Using gravimetric and atomic absorption methods, researchers calculated the Al metal components of vegetable extracts (such as onion, green beans, tomato, zucchini, and carrots), boiling animal extracts (such as fish and lamb), fresh milk and evaporated milk (Al Juhaiman, 2012). The surfaces of the kitchenware were examined using a scanning electron microscope. From this, aluminium leaching into various samples was shown to be variable and based on aluminium content, extract type, water quality, sodium chloride, temperature, and contact time (Al Juhaiman, 2012; Al Juhaiman, 2010).

In developed countries, preparing and packaging foods in aluminium foil is a primary cause of aluminium contamination. Aluminium leaching levels in sea foods that have been grilled and baked aluminium foils were also measured (Ranau, Oehlenschläger, & Steinhart, 2001). Their findings imply that aluminium seeped into the meal from the aluminium foil, although the amount of aluminium leached varied depending on a variety of factors, including the length, the composition, heating temperature and pH of the meal, the existence of other substances like salts and organic acids and the metal's dissolution owing to chemical reactions.

Also, an interest in aluminium leaching from cook wares increased with the publication of an article (Tennakone & Wickramanayake, 1987) that claimed that in the presence of 1 g/ml fluoride, there is a thousand-fold increase in aluminium leaching from kitchenware. Fluoride only minimally increased Al leaching in later investigations (Savory et al., 1987;

Fairweather-Tait et al., 1987; Baxter et al., 1988). As a result, the authors of the original study admitted to presenting an inappropriately high amount (Tennakone & Wickramanayake, 1987). It was proven that in the presence of 1 g/ml fluoride, Al leaching increased very slightly, but that leaching became significant at 10 g/ml fluoride and higher (Tennakone & Wickramanayake, 1987). As a result, it seems logical to conclude from various researches that the quantity of aluminium leached in food, beverages like tea and coffee, and drinking water is reliant on a variety of properties such as chemical and physical properties and that it can only reach toxic levels in ordinary humans under specific circumstances.

A research conducted by Samwel et al looked at Leaching of aluminium from utensils made of aluminium, indalium (alloy of aluminium), stainless steel and hard anodised aluminium was studied under different conditions of pH and boiling time. Low pH was found to enhance leaching of aluminium from the utensils. Also an experimental work analysis done by Mohammad et al., (2011) detected a significant level of aluminum in the most of the cooked food from the a two aluminium utensils used for the study. According to this research, the leaching process was increased by the addition of salt and citric acid. These results showed that low pH enhanced the leaching of aluminium.

Compare levels of aluminium leaching from different types of aluminium cooking utensils.

A research conducted by Samwel et al looked at Leaching of aluminium from utensils made of aluminium, indalium (alloy of aluminium), stainless steel and hard anodised aluminium. The leaching was found to be the

highest during first-time preparation (new utensils) of all the foods as compared with second-and third-time preparations using the same utensils. Leaching of aluminium during the preparation of various traditional Indian foods was found to be negligible in hard anodised aluminium utensils, indicating the advantage of using such vessels for food preparation over simple aluminium and indalium utensils.

Mohammad, Al Zubaidy and Bassioni (2011) conducted a study where two aluminium utensil of different origin were chosen from the available local market. Minced meat was used with two types of water, drinking and tap. Two techniques for analysis were used, weight loss (WL) measurement and inductively coupled plasma- mass spectrometry (ICP-MS). The results showed little variation between the whole meat and meat extract solution. The latter was chosen for all experimental work. Different solutions were examined starting from water, different concentrations of meat extract, 40% meat extract solution with tomato juice, citric acid, and table salt. The results of the two measurements were almost consistent. The amount of leaching of aluminum from the two utensils was found to be high in the cooking solutions using all the above additives.

Determine the level of aluminium in prepared foods

Ankar-Brewoo et al (2020) in a research, assessed the concentrations and dietary risk of the toxic metals Al, Fe, and Pb in fufu and fried-rice, two commonly consumed streetvended foods in Kumasi – Ghana. Aluminium concentrations in the prepared foods were between 3.04mg/kg and 18.49mg/kg. This level of concentration compared to the

PTWI of 1mg/kg per body weight according to WHO (2014) is relatively high.

Dordevic et al (2019), in a study looked at the degree of aluminum leakage from aluminum foil during baking process of selected food/meals. The experiment included 11 different types of food (Atlantic salmon *Salmo salar*, mackerel *Scomber scombrus*, duck breasts, cheese Hermelín, tomato, paprika, Carlsbad dumplings, pork roast, pork neck, chicken breasts, and chicken thighs) baked both marinated and not marinated. The aluminum content was measured by AAS and ICP/MS methods. The study clearly showed the occurrence of aluminum contamination of food when it is prepared by baking in aluminum foil.

Comparative standard limits of aluminium intake

Daily Intake of Metal (DIM): The daily intake of metals (DIM) is a measurement of the number of contaminants included in the food consumed each day, expressed in milligram per kilogram (mg/kg) of body weight. It is measured by the metal content of yields and the measure of food consumed by the specific food produced. DIM is determined by multiplying the number of pollutants found in a food sample by the projected quantity of food consumed daily, divided by an individual's average body weight. This index aids in determining the amount of heavy metal ingested each day in a specific dietary product. The Hazard Index (HI) is a statistic that determines the likelihood of unfavourable health effects from a combination of chemical components detected in various dietary samples. The total of the individual hazard quotients for each chemical is used to calculate the potential for damage when more than one chemical is involved

in an exposure. The hazard index is the name given to this total (Caylak, 2012).

Specific Release Limit (SRL): In September 2013, metals and alloys that come into connection with food were the subjects of a resolution enacted by the Council of Europe (Council of Europe, 2013). Specific Release Limits (SRLs) for metals and alloys, including aluminium, were recommended in this resolution. Metal ions should be emitted as little as possible as a general rule (ALARA). An SRL stipulates the maximum quantity of metal ions (in mg) that can be transported from the surface of the contact material to the food (in kg) or food simulant. The toxicological characteristics of the metals in concern are taken into account. Aluminium in consumables has a maximum permitted limit of 5.00 mg/kg of food (Council of Europe, 2013). This value can be achieved, according to data from the food industry's monitoring programs for foods and food simulants, as well as data from Europe Community members (Council of Europe, 2013). The Official control authorities utilize the SRL as well.

Provisional Tolerable Weekly Intake (TWI): The Provisional Tolerable Weekly Intake (PTWI) is a measure of how much of a substance can be consumed over a lifetime without causing significant harm. An intake that exceeds the PTWI does not always imply that one's health is jeopardized. Because the importance of the PTWI is a lifetime exposure, a transient excursion above the PTWI would have no health repercussions if the regular intake over a long period is not surpassed (Herrman &Younes, 1999).

Aluminium has a preliminary tolerable weekly intake (PTWI) of 7.00 mg/kg body weight (BW) per week, according to the Scientific Committee for Food (SCF) and the Joint FAO/WHO Expert Committee on Food Additives (JECFA). This figure was derived from animal studies of aluminium toxicity in dogs, rats, and mice (FAO/WHO, 1989). According to this, a 70 kg adult human may consume up to 490 mg per week, while a 15 kg child might consume up to 105 mg per week.

After an animal study (FAO/WHO, 2006) revealed that dosages lower than 7.0 mg/kg per week can cause harm, the provisional TWI was reduced to 1.0 mg/kg bw in 2006. Based on the results of various studies and the build-up of aluminium in the body, the European Food Safety Authority (EFSA) has set a TWI for aluminium of 1.00 mg/kg BW per week. The European Union now uses this criterion (EFSA, 2008). As a result, a 70 kg adult can consume 70.0 mg of aluminium per week for the remainder of their lives, whereas a 15 kg child can consume 15.0 mg.

It was further stated that the gastrointestinal system absorbs 0.01 per cent to 1% of orally consumed aluminium in healthy controls, and it is mostly removed via the kidney. In 2011, the Joint Food and Agriculture Organization (FAO)/World Health Organization (WHO) Expert Committee on Food Additives (WHO JECFA) set a provisional tolerated weekly intake (PTWI) of aluminium of 2 mg/kg body weight. This PTWI was then updated back to 1mg/kg body weight (WHO, 2014).

Table 1: PTWI of Aluminium Recommended by International Bodies

| International Body | Aluminium Compound | PTWI |
|---------------------------|--|----------------------------|
| JECFA 1989 | Aluminium (foodstuffs incl. additives) | PTWI 7 mg Al/kg/bw/week |
| SCF 1990 | Aluminium (all sources) | PTWI 7 mg Al/kg/bw/week |
| FAO/WHO 2006 | Aluminium (all sources) | PTWI 1mg Al/kg/bw/week |
| JECFA 2007 | Aluminium (all sources) | PTWI 1 mg Al/kg/bw/week |
| EFSA 2008 | Aluminium (all sources) | PTWI 1mg Al/kg/bw/week |
| WHO/JECFA 2011 | Aluminium (all sources) | PTWI 2 mg Al/kg/bw/week |
| WHO 2014 | Aluminium (all sources) | PTWI 1mg Al/kg/bw/week |

JECFA, Joint FAO/WHO Expert Committee on Food Additives; WHO, World Health Organization; PTWI,

Provisional Tolerable Weekly Intake; SCF, Scientific Committee for Food of the European Communities; EFSA,

European Food Safety Authority.

CHAPTER THREE

RESEARC METHODS

The materials and laboratory procedures used in this study are described in detail in this Chapter. The Chapter inculcates the research design used in this study, the data collection procedure, study area and sampling procedures. It inculcates the pre-treatment of food and utensil samples, pre-analysis of food to determine aluminium concentration, cooking of food samples, digestion of food samples for laboratory analysis, determination of aluminium in cooked samples using the complexometric back titration using EDTA titration method and the formulae used in the calculation of aluminium. It also explains the data analysis method that was used in the study.

Research Design

Experimental research design was used in this study. Experiment deals with the process of supporting, rejecting, or validating a hypothesis in order to get insight into the cause and effect of something when certain factors are being manipulated (Apuke, 2017). During an experimental research, the researcher investigates the treatment of an intervention into the study group and then measures the outcomes of the treatment. There are three types of experimental designs: pre-experimental, true experimental, and quasi-experimental (Leedy & Ormrod, 2019).

The pre-experimental design involves an independent variable that does not vary or a control group that is not randomly selected. Campbell and Stanley (1963) endorsed the true experimental design, which provides a higher degree of control in the experiment and produces a higher degree

of validity. The true experimental designs result in a systemic approach to quantitative data collection involving mathematical models in the analyses. Whereas, the quasi-experimental design involves nonrandom selection of study participants. Therefore, control is limited and true experimentation is not possible. Since the variable cannot be controlled, validity may be sacrificed (Apuke, 2017).

In this study, the true experimental design was employed, quantitative data was collected using a systematic approach and also involved the use of a mathematical model.

Study Area

The research took place in the Central Region's Cape Coast metropolis. Out of the twenty-three (23) districts, Cape Coast is the only metropolitan area in the Central Region. It is located between latitudes 5° 20' and 1° 11' to 1° 41' west of the Greenwich Meridian. The Gulf of Guinea borders the Metropolis on the south, the Komenda Edina Eguafo Abrem on the west, the Abura Asebu Kwamankese District on the east, and the Twifu Hemang Lower Denkyira District on the north. It is around 122 square kilometres in size, with the farthest point being Brabedze, about 17 kilometres from Cape Coast, the Metropolis in Central Region. In Cape Coast, the largest market is known as the Kotokoroba Market. It is the commercial centre of the region, where all major trading stores are located around (CCMA, 2022). The samples used for the study were purchased from vendors located in this study area. This research was conducted in this area below.

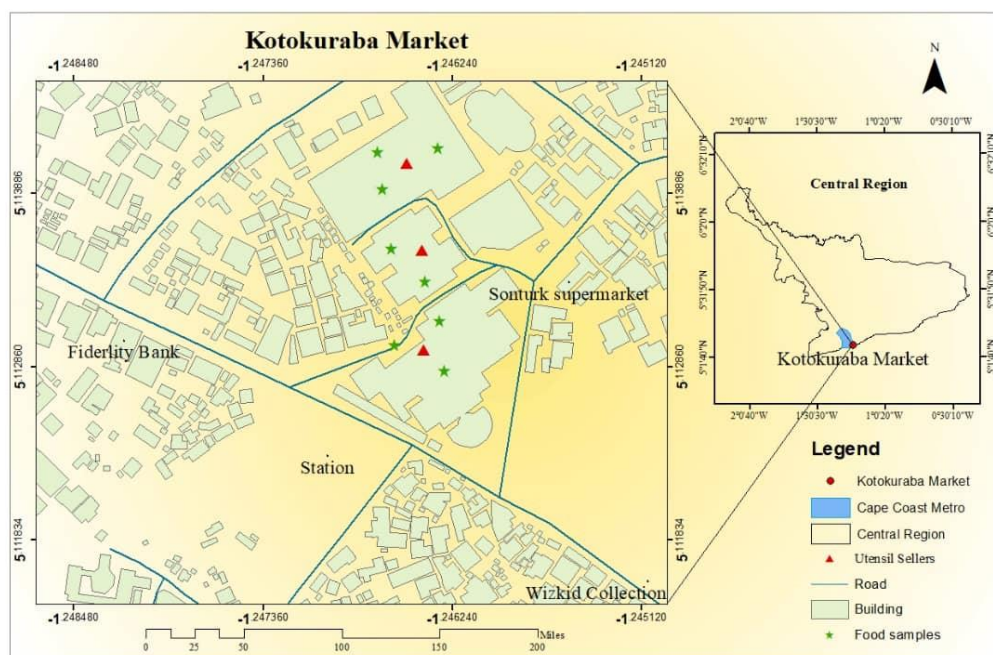


Figure 3: Map of Kotokuraba Market.

Source: CCMA, (2022).

Data Collection

Data was collected on the experimental conditions which included pH, temperature, rate of aluminium leaching, and level of aluminium concentration. After the experimental procedures conducted to measure the pH using pH paper and PC Eutech, the observed pH readings of the samples were reported to the nearest whole number in the laboratory note book. The temperature was measured using a food thermometer during the cooking process. This helped to sustain the temperature used in cooking. Complexometric back titration was used to determine the aluminium concentrations in both the raw and cooked foods. The results of the various experimental titration performed on the selected raw and cooked foods were also recorded in the laboratory note book according to the selected

foods. All analysis were performed in triplicates and the results averaged to indicate the observed aluminium concentrations in the sample.

Sampling Procedure

The purposive sampling procedure was used to select the cooking utensils and the food commodities used for the study. Purposive sampling is a non-probability sampling technique in which units are chosen for the sample based on certain attributes. It takes place when "elements selected for the sample are chosen by the judgment of the researcher." A common believe among researchers is that they can save time and money by employing good judgment to acquire a representative sample (Saunders, Lewis, & Thornhill, 2009).

Three different brands of aluminium cooking utensils ranging from 500ml to 1000ml in sizes were randomly chosen from the Kotoburaba market in the Cape Coast Town in the Central Region of Ghana. The brands of Aluminium utensils used were St Stephen Aluminium (Ship Brand), Floral Aluminium (Oak Brand) and the Cauldron mostly known as iron pot or 'Dadesen' in Ghana. The utensils were labelled A, B, and C respectively for easy identification (Figure 2). The food samples were fresh tomatoes, rice and cocoyam leaves bought from known vendors in the kotokuraba market. Reasons for the selection of these food samples are: fresh tomato is a kind of vegetable which is mostly used nationwide; rice is a cereal which is easily accessible, available and consumed nationwide and cocoyam leaves are mostly used in the preparation of palava sauce which is consumed by most Ghanaians.

The study was conducted for 9 weeks where food samples were bought at the weekly interval and subjected to all sample preparation and aluminium determination analysis.



Figure 4: Cooking utensils used for the research work.

Source: Adukpoh, (2022).



Figure 5: Selected foods cooked in the aluminium cooking utensils.

Source: Adukpoh, (2022).

Determination of pH of Food Samples Before Cooking (Ghana Standards Authority, 2006)

Determination of pH of tomatoes: The fresh tomatoes were washed under running water, chopped on a plastic chopping board and then ground in an earthen ware bowl. A pH paper was then dipped in, the colour change was observed and recorded.

Determination of pH of rice: The washed rice was poured into a beaker, and two to three masses of distilled water (or more if required to give a suitable consistency) were added. It was then heated in a water bath for 30 minutes. In every 5 minutes it was mixed with a rod. After the 30 minutes it was grounded using a clean mortar and pestle. The pH paper was then dipped in the sample, colour was observed and the pH was recorded.

Determination of pH of cocoyam leaves: Freshly washed cocoyam leaves were chopped on a chopping board. It was then placed in an earthen ware bowl where 2 to 3 masses of tap water were added and grounded. The pH paper was then used to check the pH and the result was recorded.

Determination of pH of tap water: 200 ml of tap water was poured into a beaker, the pH paper was then dipped in, the colour change was observed and the pH was recorded.

Sample Preparation and Cooking of Food Commodities in the Kitchen

Procedure: Aluminium utensils A, B and C were thoroughly washed with tap water and set aside. The food samples were also washed under running water to ensure they were clean and free from contamination. Using a plastic chopping board, about 100g of fresh tomatoes were chopped and placed in Utensil A where 150mL-300mL of tap water was added and cooked on a hot plate for 30 minutes at 100⁰ Celcius. Similarly, 100g of cocoyam leaves was weighed and washed. After, thoroughly washing the cocoyam leaves, using a plastic knife it was chopped and placed in pot B where it was cooked using 150-200 mL of tap water for 15 minutes at 100⁰ Celcius. Lastly, 100g of rice was weighed, washed in a bowl of tap water and cooked in Utensil C using 200mL-250mL of Tap water for 45 minutes

at 100⁰ Celcius on a hot plate.

Table 2: The Conditions Used in the Preparation of the Food Samples in the Kitchen

| Food Type | Quantity (grams) | Temperature (°C) | Cooking (minutes) | Time |
|----------------|---------------------|---------------------|----------------------|------|
| Tomatoes | 100 | 100 | 30 | |
| Cocoyam leaves | 100 | 100 | 15 | |
| Rice | 100 | 100 | 45 | |

Source: Adukpoh, (2022).

Laboratory Sample Pre-Treatment

Reagents: Aluminium ions solution (analyte), Eriochrome black T indicator, Standardized 0.01 M zinc sulphate solution(titrant), 0.01 M EDTA solution, Hydrochloric acid

Apparatus: Filter Paper, Erlenmeyer flask, Volumetric flask, Wash bottle, Hot plate, Pipette, Burette

Procedure: Cooked food samples were obtained from the respective utensils. 50g of each sample were weighed exactly to the closest 50 ± 0.0001 g (labelled as w_{sample}). The rice was mashed in a well-dried and clean mortar using a pestle to obtain a fine sample.

Digestion of Food Samples (Yang & Tsai, 2006)

The food samples were transferred to a spotless 250 mL Erlenmeyer flask containing 100 mL deionized water and 6 mL 6MHCL (hydrochloric acid) was added. The combination was boiled gently on a hot plate for about 20 minutes. A stem funnel was placed on the opening of the flask such that the vapour can swiftly condense back into water and wash down the mashed sample that clings to the wall of the flask. Any little mashed sample that

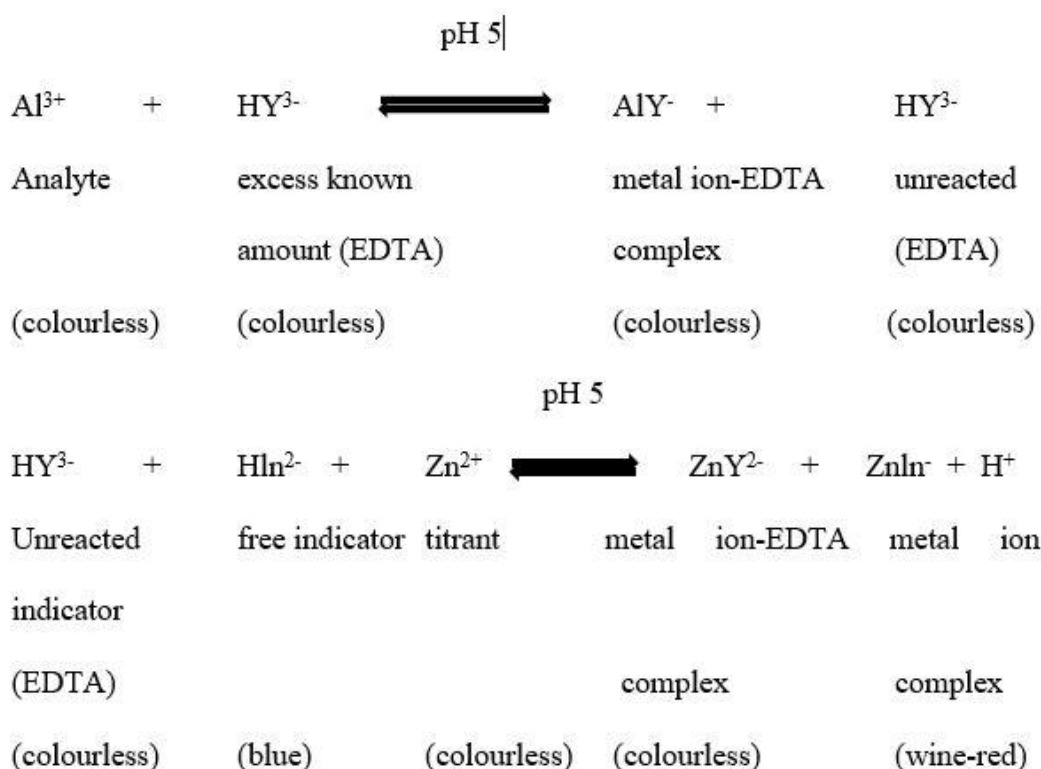
remains affixed to the wall of the flask was washed down with a modest quantity of deionized water and heating was continued. After then, the flask was taken off the hot plate and allowed to cool to room temperature. The combination was then gravity filtered into a volumetric flask with a capacity of 250 mL. After which the flask and residue on the filter paper were washed twice with deionized water to ensure that all metallic ions were transported into the volumetric flask. The solution was then diluted with deionized water to the calibration mark. The Cork was then placed on the flask and the solution was well blended by capsizing and quaking it repetitively. The final step was to label the solution as “The digested food sample solution, 250 mL” (denoted as $V_{\text{food sample}}$).

Determination of Aluminium (Al^{+3}) By Complexometric Back Titration Using EDTA Titration

Principle: Complexometric titration is a type of quantitative analysis used in chemistry laboratories, both general and analytical. Complexometric titration is a type of volumetric analysis in which the development of a coloured complex is utilized to identify the titration's end point. Complexometric titrations are very effective for determining the concentration of a combination of metal ions in solution (Yang and Tsai, 2006). The best approach for identifying aluminium ion is to do a complexometric back titration with a heating environment to encourage chemical bonding between the aluminium ion and EDTA. The reverse titration method is used when a cation forms a steady complex with EDTA in a delayed reaction or when a barometer is prevented from forming a metal ion complex with a greater steady constant than the metal-EDTA

complex. Reverse titration with a standardized zinc sulphate mixture is superior to calcium solution for clearly observing the endpoint in a low pH buffer solution.

In a slow process, complexometric reverse titration is utilized when metallic ions form a steady complex with EDTA or when a metal ion blocks an indicator. This led to no change in colour after the complexometric direct titration because the blocked indicator is unable to release metallic ions. Because these conditions exist in the situation of the aluminium ion, complexometric reverse titration coupled with heating to promote Al-EDTA complexation is the most effective approach for identifying the ion. In this study, an additional quantity of EDTA was added to the Aluminium sample mixture, followed by a pH 10 buffer solution where the Al-EDTA complex can develop. The mixture was heated before the indicator Eriochrome black T, which is blue in its free state was added, to avoid aluminium ions from obstructing the indicator and to enable Al-EDTA complexation. The quantity of unchelated EDTA can be estimated via complexometric back titration with standardized zinc or magnesium mixture. A transformation in colour to a wine-red hue was seen at the end of the process (Yang & Tsai, 2006).



Procedure for the Determination of the Level of Aluminium Concentration in Food Samples Cooked in Aluminium Utensils

The tap water used in cooking the food samples was taken through the same process as the sample to determine the aluminium component. To conceal the development of the Mg-EDTA complex, a 25.00 mL aliquot of the digested meal sample solution was pipetted into a 10 mL acetate-acetic acid pH 5 buffer solution. A burette was used to put a 30.00 mL aliquot of the EDTA standard solution into the flask (labelled as V_{EDTA}) (If EDTA isn't adequate to completely chelate all aluminium ions, a deep blue solution would be observed. To this deep blue solution, add 5.00 or more mL aliquot of the EDTA solution. Return to a boil until the colour turns blue). To speed up the creation of the Al-EDTA complex, it was gently boiled for 5 minutes on a hot plate. The pH was adjusted to 10 after cooling to room temperature,

then 2-3 drops or 30mg of Eriochrome Black T indicator was added and thoroughly mixed. At this point, the solution looked blue. Back-titrated with standardized zinc sulphate solution until the colour changed to wine red at the endpoint. In cases where the wine-red colour shortly turned back to blue, the solution was continuously titrated until a wine-red colour persisted for 3 minutes. The volume used should then be recorded (V_{Zn}).

Note: Because EDTA's capacity to chelate zinc ions is diminished at low pH, a little quantity of EDTA progressively shifts to an active polydentate species in line with Le Chatelier's Principle, resulting in a return to blue colour. To ensure that the Volume of Zinc sulphate (V_{Zn}) readings concur to within 1% of the relative normal deviation, replicate the titration two more times. The concentration of aluminium present in the sample solution and the food sample was determined.

Calculations

$$\text{Concentration of Al}^{3+} \text{ solution (mol L}^{-1}\text{)} = \frac{[M_{EDTA} \cdot (V_{EDTA} - V_{Zn}^{2+})]}{V_{Al}^{3+}}$$

$$\text{Concentration of Al}^{3+} \text{ solution (g L}^{-1}\text{)} = \frac{[M_{EDTA} \cdot (V_{EDTA} - V_{Zn}^{2+})] \cdot At. wt_{Al}}{V_{Al}^{3+}}$$

Where:

M_{EDTA} – Molarity of the EDTA

V_{EDTA} – Volume of the EDTA

V_{Zn}^{2+} – Volume of Zinc sulphate

V_{Al}^{3+} – Volume of Aluminium standard

$At. wt_{Al}$ – Atomic weight of aluminium

Data Analysis

Data were analysed using Analysis of Variance (ANOVA). ANOVA is a statistical method for comparing groups based on differences in the average (mean) of a quantitative (interval or ratio, continuous) measure. Factors are variables that assign respondents to various groups; an ANOVA can have one (one-way) or several factors (a multi-way or factorial design). The term analysis of variance refers to the partitioning of the total variation in the outcome variable into parts explained by the factor(s)—related to differences between groups, so-called explained or between variation—and a part that remains after the factor(s) are taken into account, referred to as unexplained, residual, or within variation (Lavrakas, 2008). Only one independent variable exists in a one-way ANOVA whereas in a two-way ANOVA (sometimes known as a factorial ANOVA) analysis of variance with two independent variables is done (Statistics Solution, 2013). In this research study, one-way ANOVA was employed.

Analysis of variance (ANOVA) is simply an example of the general linear model (GLM) that is commonly used for factorial designs (Henson, 2015). One-way ANOVA compares the means of the dependent variable scores obtained from any number of groups. In GLM terms, ANOVA attempts to explain data (the dependent variable scores) in terms of the experimental conditions (the model) and an error component (Rutherford, 2011). Typically, the researcher applying ANOVA is interested in determining which experimental condition dependent variable score means differ. There is also interest in what proportion of variation in the dependent variable can be attributed to differences between specific experimental

groups or conditions, as defined by the independent variable(s) (Rutherford, 2011). The dependent variable in ANOVA is most likely to be measure on a quantitative scale. However, the ANOVA comparison is drawn between the groups of subjects receiving different experimental conditions and is categorical in nature, even when the experimental conditions differ along a quantitative scale (Rutherford, 2011).

The main reason for adopting a GLM approach to ANOVA is that, it provides conceptual and practical advantages over the traditional approach. The GLM conception divides data into model and error, and it follows that the better the model explains the data, the less the error. Therefore, the set of predictors constituting a GLM can be selected by their ability to reduce the error term. Moreover, as most GLM assumptions concern the error terms, it provides a common means by which the assumptions underlying ANOVA can be assessed (Rutherford, 2011).

Assumptions for ANOVA (Tabachnick & Fidell, 2007).

1. Normality of sampling distribution of means

An assumption of ANOVA is that the sampling distribution of means for each level (or combination of levels) of the independent variable(s) is normal. The assumption is for the sampling distribution, not the raw scores. If the raw scores are normally distributed, the sampling distribution of their means is also normally distributed. However, even if the raw scores are not normally distributed, the Central Limit Theorem assures us that the sampling distribution of means is normally distributed for large enough samples.

2. Independence of errors

The last term in the GLM is e , the error term. Another assumption of analysis is that the errors are independent of one another—that the size of the error for one case is unrelated to the size of the error for cases near in time, near in space, or whatever.

3. Homogeneity of Variance

The ANOVA model tests whether population means from different levels of the independent variable are equal. However, the model assumes that population variances in different levels of the independent variable are equal—that is, it is assumed in ANOVA that the variance of dependent variable scores within each level of the design is a separate estimate of the same population variance.

4. Absence of outlier

An outlier is a score that is unusually far from the mean of its own group and apparently disconnected from the rest of the scores in the group. The effects of outliers are unpredictable; they can hide real statistical significance or create apparent statistical significance in ANOVA. They severely limit the generalizability of the results. For this reason, it is important to screen data sets for outliers prior to analysis. They are sought separately in each level (or combination of levels) of a design.

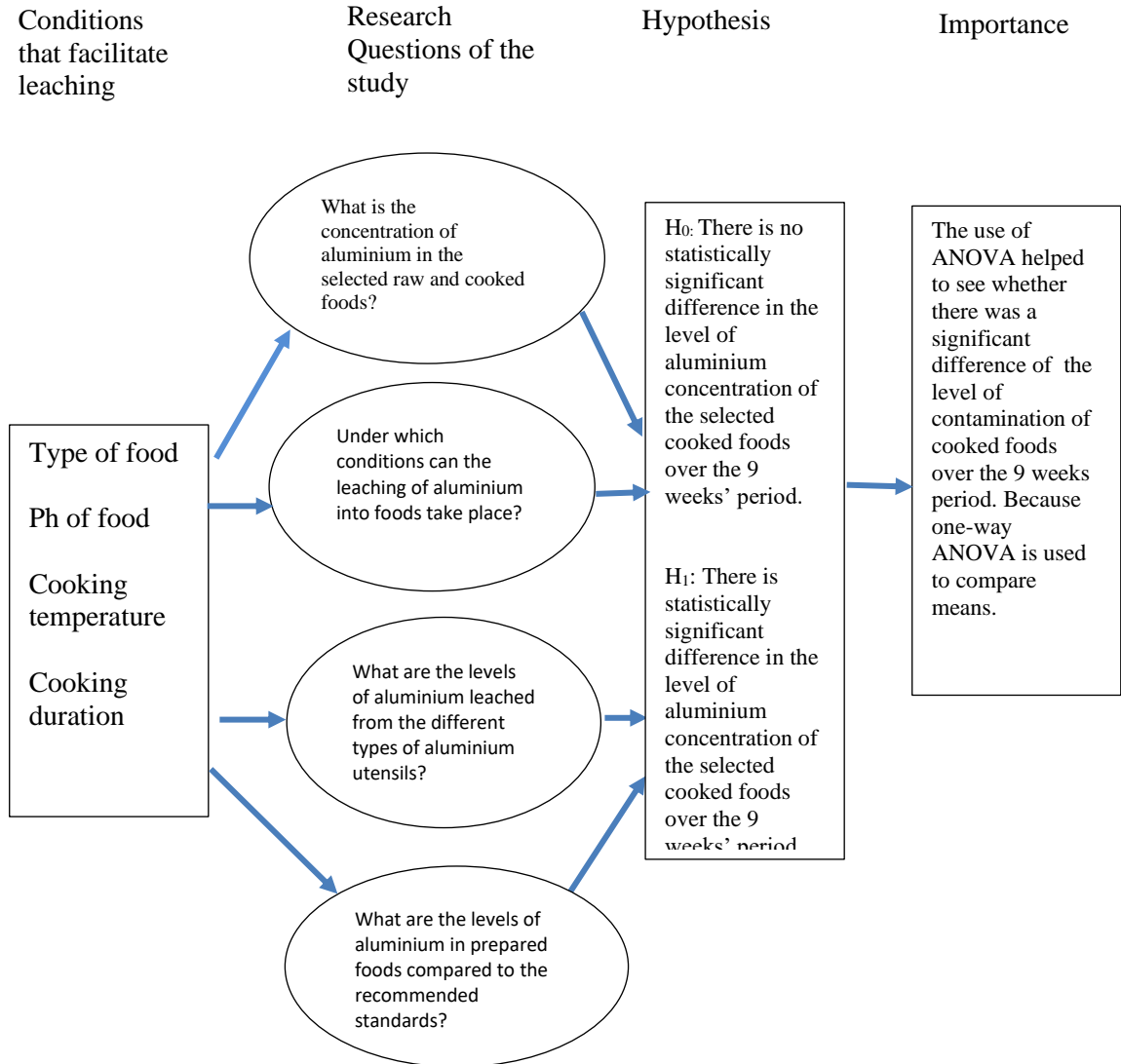


Figure 6: Conceptual framework for conducting the ANOVA (one way).

Source: Tabachnick and Fidell (2007).

CHAPTER FOUR

RESULTS AND DISCUSSION

This chapter of the research thesis put together the results as well as the discussions relating to the specific objectives of this research. This chapter includes the results of the pH of the food samples before and after cooking, the concentration of aluminium in the uncooked food samples, concentration of aluminium in the cooked foods, the levels of aluminium leached from utensil A,B and C over the 9 weeks period of analysis. It also presents the p-values of each sample within the 9 weeks of study.

Results

Table 3: pH of Food and Water Before and After Cooking

| Food samples | pH Before cooking | pH After cooking |
|--------------|-------------------|------------------|
| Tomatoes | 4.0 | 4.0 |
| Rice | 6.0 | 6.0 |
| Nkontomire | 6.0 | 6.0 |
| Tap Water | 7.0 | 7.0 |

*Source:*Adukpoh, (2022).

Table 4: Averaged Concentration of Aluminium in Raw (Uncooked) Food Samples and Water.

| Raw Food Samples and Water | Aluminium Concentration |
|----------------------------|-------------------------|
| Tomatoes | 0.36mg/kg |
| Rice | 0.14mg/kg |
| Nkontomire | 0.90mg/kg |
| Tap water | 0.54mg/l |

*Source:*Adukpoh, (2022).

Table 5: Al³⁺ Concentration in Tomatoes – Utensil A (Week 1-9)

| Samples | Weeks | Mean conc \pm SD | Ph values |
|-----------------------|-------|----------------------|-----------|
| Tomatoes | 1 | 2.88mg/kg \pm 2.03 | 5.0 |
| Tomatoes | 2 | 5.49mg/kg \pm 4.62 | 4.0 |
| Tomatoes | 3 | 4.23mg/kg \pm 1.41 | 4.0 |
| Tomatoes | 4 | 2.76mg/kg \pm 2.29 | 5.0 |
| Tomatoes | 5 | 3.51mg/kg \pm 2.04 | 4.0 |
| Tomatoes | 6 | 1.85mg/kg \pm 2.61 | 5.0 |
| Tomatoes | 7 | 5.76mg/kg \pm 0.62 | 4.0 |
| Tomatoes | 8 | 4.95mg/kg \pm 0.62 | 4.0 |
| Tomatoes | 9 | 5.11mg/kg \pm 0.73 | 4.0 |
| Average Concentration | | 4.06mg/kg \pm 1.87 | |
| PTWI (WHO, 2014) | | 1mg/kg | |

Source: Adukpoh, (2022).

Table 6: Al³⁺ Concentration in Rice – Utensil C (Week 1-9)

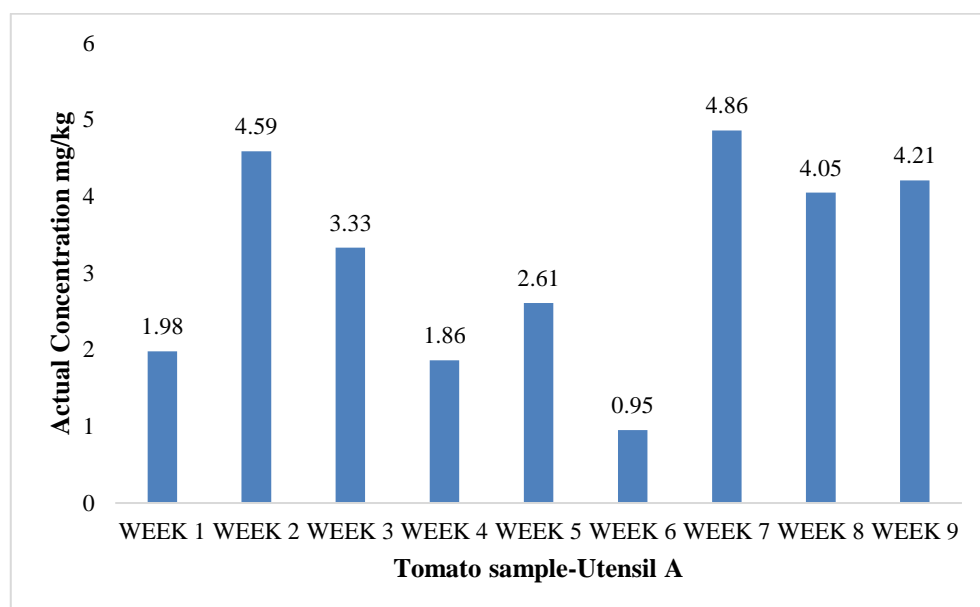
| Samples | Weeks | Mean concentration \pm SD | pH values |
|-----------------------|-------|-----------------------------|-----------|
| Rice | 1 | 3.06mg/kg \pm 0.62 | 6.0 |
| Rice | 2 | 2.25mg/kg \pm 0.68 | 6.0 |
| Rice | 3 | 5.49mg/kg \pm 1.22 | 6.0 |
| Rice | 4 | 3.78mg/kg \pm 1.43 | 6.0 |
| Rice | 5 | 2.77mg/kg \pm 1.09 | 6.0 |
| Rice | 6 | 3.33mg/kg \pm 2.17 | 6.0 |
| Rice | 7 | 4.50mg/kg \pm 4.09 | 6.0 |
| Rice | 8 | 2.48mg/kg \pm 0.54 | 6.0 |
| Rice | 9 | 2.47mg/kg \pm 0.55 | 6.0 |
| Average Concentration | | 3.35mg/kg \pm 1.38 | |
| PTWI (WHO, 2014) | | 1mg/kg | |

Source: Adukpoh, (2022).

Table 7: Al^{3+} Concentration in Nkontomire – Utensil B (Week 1-9)

| Samples | Weeks | Mean concentration \pm SD | pH values |
|-----------------------|-------|-----------------------------|-----------|
| Nkontomire | 1 | 4.32mg/kg \pm 0.54 | 6.0 |
| Nkontomire | 2 | 4.05mg/kg \pm 0.48 | 6.0 |
| Nkontomire | 3 | 3.40mg/kg \pm 1.04 | 6.0 |
| Nkontomire | 4 | 4.50mg/kg \pm 2.30 | 6.0 |
| Nkontomire | 5 | 4.95mg/kg \pm 0.87 | 6.0 |
| Nkontomire | 6 | 4.13mg/kg \pm 0.63 | 6.0 |
| Nkontomire | 7 | 3.15mg/kg \pm 2.45 | 6.0 |
| Nkontomire | 8 | 5.58mg/kg \pm 2.72 | 6.0 |
| Nkontomire | 9 | 3.42mg/kg \pm 2.77 | 6.0 |
| Average Concentration | | 4.17mg/kg \pm 1.23 | |
| PTWI (WHO, 2014) | | 1mg/kg | |

Source: Adukpoh, (2022).

**Figure 7: Concentration of aluminium (Al^{3+}) leached from utensil A into tomatoes.**

Source: Adukpoh, (2022).

In Figure 7, the analysis conducted over nine weeks show that there was a higher concentration of aluminium in tomatoes sample cooked in

week 7 with the aluminium concentration of 4.86mg/kg, followed closely by that of week 2, 9 and 8 with aluminium concentrations of 4.59mg/kg, 4.21mg/kg and 4.05mg/kg respectively whilst week 6 recorded the least with the aluminium concentration of 0.95 mg/kg. The p-value 0.26 calculated, is greater than the critical p-value 0.05. This indicates that there is no statistically significant difference in the result of the concentration of aluminium leached into the food sample throughout the nine weeks, as such we accept the alternate hypothesis and reject the null hypothesis.

Table 8: Anova table showing the p-value for the tomato sample

| ANOVA | | | | | | | |
|---------------------|----------|----|----------|----------|----------|----------|--------|
| Source of Variation | SS | df | MS | F | P-value | F crit | F crit |
| Between Groups | 56.76253 | 9 | 6.306948 | 1.376109 | 0.262755 | 2.392814 | #NUM! |
| Within Groups | 91.66351 | 20 | 4.583175 | | | | |
| Total | 148.426 | 29 | | | | | |

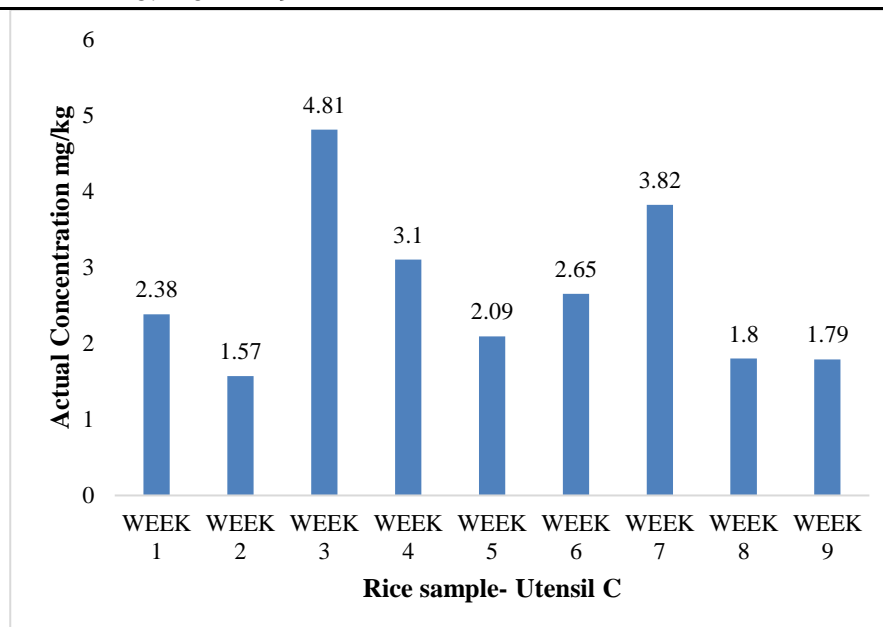


Figure 8: Concentration of aluminium (Al^{3+}) leached from Utensil C into rice sample.

Source, Adukpoh (2022).

In Figure 8, because of the variations in aluminium concentrations over the 9 weeks, it can be observed that there was a higher concentration of aluminium in the rice sample cooked in week 3 with an aluminium concentration of 4.81mg/kg, followed closely by that of week 7, 4 and 6 with aluminium concentrations of 3.82mg/kg, 3.10 mg/ and 2.65mg/kg respectively whilst week 2 recorded the least with an aluminium concentration of 1.57mg/kg. The p-value 0.31 calculated, is greater than the critical p-value 0.05, which indicates that the result of the concentration of aluminium leached into the rice sample throughout the nine weeks showed no significant difference, as such we accept the alternate hypothesis and reject the null hypothesis.

Table 9: Anova Table Showing the p-value for the Rice Sample.

| <i>Source of Variation</i> | <i>SS</i> | <i>Df</i> | <i>MS</i> | <i>F</i> | <i>P-value</i> | <i>F crit</i> |
|----------------------------|-----------|-----------|-----------|----------|----------------|---------------|
| Between Groups | 32.67328 | 9 | 3.630364 | 1.271181 | 0.310872 | 2.392814 |
| Within Groups | 57.118 | 20 | 2.8559 | | | |
| Total | 89.79128 | 29 | | | | |

Source: Adukpoh, (2022).

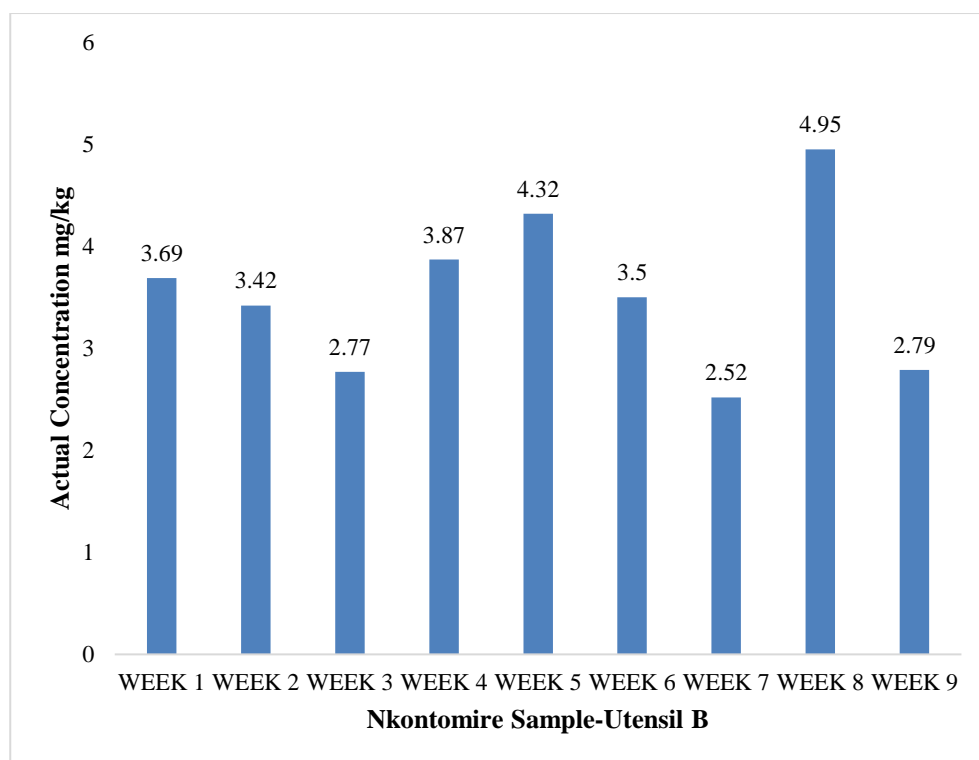


Figure 9: Concentration of aluminium (Al^{3+}) from the utensil B.

Source: Adukpoh, (2022).

Nkontomire sample

In Figure 9, considering the change in concentration over the period, it could be seen that week 8 recorded the highest leaching of aluminium in nkontomire, thus 4.95mg/kg, followed by 5, 4 and 1, having 4.32mg/kg, 3.87mg/kg and 3.69mg/kg respectively. Week 7 recorded the least with an aluminium concentration of 3.15 mg/kg. This could be a result of the variations that occurred during the cooking period. The p-value 0.460 calculated, is greater than the critical p-value 0.05. This indicates that the result of the concentration of aluminium leached into the food sample throughout the nine weeks of research showed no significant difference, as such we accept the alternate hypothesis and reject the null hypothesis.

Table 10: Anova Table Showing p-value for Nkontomire Sample.

| <i>Source of Variation</i> | <i>SS</i> | <i>df</i> | <i>MS</i> | <i>F</i> | <i>P-value</i> | <i>F crit</i> |
|----------------------------|-----------|-----------|-----------|----------|----------------|---------------|
| Between Groups | 27.50847 | 9 | 3.056496 | 1.016157 | 0.460072 | 2.392814 |
| Within Groups | 60.15793 | 20 | 3.007897 | | | |
| Total | 87.6664 | 29 | | | | |

Source: Adukpoh, (2022).

Discussion

Determination of the concentration of aluminium, in the selected cooked, and, uncooked foods

Comparatively, there are pieces of evidence of aluminium leaching in cookware used for this study. Clear evidence for tomatoes seen in table 4, shows that the leaching of aluminium in the tomatoes for the 9 weeks of research showed an approximate high aluminium leach concentration in the food commodity thus 4.2mg/kg, being the average mean concentration for the first 3 weeks, 2.71mg/kg for the next 3 weeks and 5.27mg/kg for the last three weeks. It also showed that the uncooked tomato had an aluminium concentration of 0.36mg.

Also, the raw uncooked rice used in this study had 0.14mg. For the cooked rice, for the first 3 weeks, the average concentration of aluminium was 3.06mg, 2.25mg and 5.49mg. In the second 3 weeks, it was observed that week 4 had 3.78mg, 2.77mg for week 5 and week 6 had 3.33 mg. It could be seen that the first 3 weeks had concentrations higher than the last 3 weeks. The above assertion conforms with Samwel et al, (2006), where similar observations of leaching were shown to be higher when all foods were prepared for the first time (with new utensils) than when they were prepared for the second, third, and subsequent times with the same cook

ware. In the last phase, the concentrations were 4.50mg, 2.48mg and 2.47mg respectively for weeks 7, 8 and 9.

Lastly, the aluminium concentration recorded for nkontomire in the 1st to 3rd weeks were 4.32mg, 4.05mg and 3.40mg respectively with an average of 3.92mg. The aluminium concentration seen in the second three weeks recorded an average of 4.53mg whereas the last three weeks recorded 4.05mg as the average concentration. This shows that the second three weeks recorded a higher concentration of aluminium compared to the first three weeks and the last three weeks. In this study, the aluminium concentration of the raw nkontomire was 0.09mg.

The conditions (temperature, pH, cooking utensils) that may facilitate the leaching of aluminium into foods

In the tomato, sample leaching could have been attributed to the nature of the utensil being new and having the coat used in covering the metal surface this assertion resonates with Semwal et al., (2006). In the third cycle of weeks there was a high leach of aluminium into the tomato this could be attributed to the fact that after cooking, the utensils were being washed which could have accounted for the wearing of the surface coating. Also, tomato is known to be an acidic food because it contains a high amount of citric acid (Al Juhaiman, 2012; Al Juhaiman, 2010; Semwal et al, 2006). In line with, Al Juhaiman, (2012), Al Juhaiman, (2010) and Semwal et al., (2006), the pH recorded values before and after cooking were pH 4.0 and pH 4.0 respectively. These values affirm the already known acidic nature of tomatoes.

The concentration of aluminium recorded in weeks 7-9 was 5.76mg, 4.95mg and 5.11mg which indicates high leaching as compared to the other 6 weeks. There was a decline in aluminium concentration in the 4th, 5th and 6th weeks recording 2.78mg, 3.51mg and 1.85mg respectively. These differences could be a result of variations in soil and growing conditions and or farming practices as well as water used in irrigation had low acidity (Sullivan et al, 1987; Pennington & Jones, 1989; Semwal et al, 2006). In general, aluminium cook wares have been observed to contribute to the daily intake of aluminium when tomatoes are cooked in such wares.

Several reasons could probably cause the presence of aluminium in raw rice. It could be due to rice processing method, irrigation water aluminium level, aluminium level in the soil etc. This assertion has been reported earlier by Niu, (2018), Amarasooriya and Dharmagunawardhane, (2014) and Fekete et al., (2012). Also, the amount of aluminium leached into rice could be a result of the findings presented by Ranau, Oehlenschläger and Steinhart (2001) and Samwel et al., (2006), that aluminium quantity increases with cooking duration at boiling temperature. For this research, the rice was cooked at 100⁰C where it is expected that the water used in cooking will boil. In addition, these results corroborate those found by some authors that showed there is the migration of aluminium in long-cooked foods. In this research, rice was cooked for 45 minutes, which is considered a long cooking duration.

The level of aluminium concentration in nkontomire could be a result of certain factors and conditions. One factor could be the presence of silica which is known to increase the toxicity of aluminium, it has also been

reported to correlate to aluminium toxicity in water (Zatta, 2000) and green leafy vegetables are known to have high levels of silica (Robberecht, Van Dyck, Bosscher, & Van Cauwenbergh, 2008; Njenga, Maina, Kariuki & Mawangi, 2007). Nkontomire being an example of a green leafy vegetable is likely to contain an appreciable amount of silica. Another contributing factor that can be used as a basis for the explanation of the high concentration of aluminium in the cooked nkontomire is high levels of fluoride in the vegetable. It has been recorded by other scholars that the toxicity of aluminium may be because vegetables are known to contain high levels of fluoride which has been noted to enhance aluminium absorption (Njenga, Kariuki & Ndegwa, 2005), therefore the increasing concentration of aluminium in the nkontomire after cooking in an aluminium cooking utensil.

These differences in the level of aluminium concentration could also be a result of variations in soil and farming practices as well as the water used in irrigation could be of low acidity (Niu, 2018; Semwal et al, 2006).

Levels of aluminium leached from different types of aluminium cooking utensils

The brands of Aluminium utensils used were St Stephen Aluminium (Ship Brand), Floral Aluminium (Oak Brand) and Cauldron mostly known as iron pot and 'Dadesen' in Ghana. The utensils were labelled A, B, and C respectively for easy identification. Utensil A was used to cook the tomato food sample, utensil B was used to cook cocoyam leaves and utensil C was used to cook the rice sample. The level of aluminium leached from the different aluminium utensils is shown in Tables 4, 5, and 6 above.

For the tomatoes sample, the actual concentration was determined from the nine different weeks of the study, week 7 recorded the highest leaching of aluminium concentration with $4.86\text{mg/kg} \pm 0.62$, followed closely by weeks 2, 9, and 8 recording $4.59\text{mg/kg} \pm 4.62$, $4.21\text{mg/kg} \pm 0.73$ and $4.05\text{mg/kg} \pm 0.62$ respectively. However, week 6 recorded the least aluminium concentration leaching of $0.95\text{mg/kg} \pm 2.61$ while the rest of the weeks gave intermediate leaching of aluminium.

Also, week 3 recorded the highest leaching of aluminium into the cooked rice sample with a concentration of $4.81\text{mg/kg} \pm 1.22$ followed closely by weeks 7, and 4 recordings of $3.82\text{mg/kg} \pm 4.09$ and $3.10\text{mg/kg} \pm 1.43$ respectively whilst week 2 recorded the least with a concentration of $1.57\text{mg/kg} \pm 0.68$.

Lastly, Nkontomire on the other hand had week 8 recording the highest leaching of aluminium with a concentration of $4.95\text{mg/kg} \pm 2.72$. Weeks 5, 4, and 1 recorded the next highest leaching of aluminium with concentrations of $4.32\text{mg/kg} \pm 0.87$, $3.87\text{mg/kg} \pm 2.30$ and $3.69\text{mg/kg} \pm 0.54$ respectively whereas week 7 recorded the least concentration with $2.52\text{mg/kg} \pm 2.45$.

Averagely, utensil A (used for tomatoes), utensil B (used for nkontomire) and utensil C (used for rice) leached these concentrations of aluminium; $3.16\text{mg/kg} \pm 1.87$, $3.54\text{mg/kg} \pm 1.23$ and $2.67\text{mg/kg} \pm 1.38$ respectively into their respective food samples. From these results, nkontomire cooked with utensil B recorded the highest leaching followed by utensil A and lastly utensil C.

Comparison of the concentration of aluminium within the prepared food with the recommended standards

Consequently, a careful look at the concentrations of leached aluminium ($3.16\text{mg/kg} \pm 1.87$, $3.54\text{mg/kg} \pm 1.23$ and $2.67\text{mg/kg} \pm 1.38$ for tomatoes, nkotomire and rice respectively) in comparison to the PTWI (Provisional Tolerable Weekly Intake) reference limits which are 1mg/kg per body weight according to WHO, 2014, shows that despite the fluctuations in concentrations throughout research, it could be seen that almost all the selected food substances were contaminated with aluminium. Metal ions constitute a variety of public health concerns, including food contamination and have been a principal concern over the years. The results obtained suggest that aluminium contamination in foods is real and its effect cannot be neglected in selected foods (Tomatoes, Rice and Nkontomire).

Aluminium has been discovered in moderate amounts (less than 0.2mg) in most meals (Greger & Sutherland, 1997; Stahl, 2017). According to Agriculture and Agri-food Canada (2001), as cited by Landry (2014), certain studies have identified increased intestinal absorption of aluminium from food amongst the aged and immunocompromised people, raising worries regarding harmfulness in these groups. Hence people who prepare food using aluminium cookware are predisposed to a high concentration of metal ions. Researchers assert that the distribution of aluminium is better understood as it accumulates mostly in the lungs and bones (Krewski et al., 2007). Also, soft tissues (typically after intravenous fluid contamination), the spleen, liver, kidney, nerve tissues, muscles, and the heart are among the high concentration afflicted areas (Saiyed & Yokel, 2005). The kidneys,

which account for 95 per cent of elimination, and bile are the most common routes of excretion (Krewski et al., 2007).

In all the observations made, it could be understood that aluminium cookwares are contributors to the daily intake of aluminium metal when foods are prepared using aluminium cooking utensils.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

The summary of the entire study is presented in this chapter, as well as the key findings and the conclusions drawn from the results obtained from the discussion. The chapter also suggests some recommendations that would help reduce the intake of aluminium from aluminium cooking utensils as it has adverse effects on health. This chapter finally proposes some areas for further studies

Summary

The fundamental goal of this research was to investigate the possible contamination of selected cooked foods in aluminium cookware. Specifically, the study sought to;

1. Determine the concentration of aluminium in selected raw and cooked foods.
2. Examine the conditions (temperature, pH and cooking time) that may facilitate the leaching of aluminium into cooked foods.
3. Compare levels of aluminium leaching from different types of aluminium cooking utensils.
4. Determine the level of aluminium in prepared foods.

The study adopted a quantitative research method and used an experimental design. A reliable analytical method known as the complexometric back titration (Sangale, Daptare and Sonawane, 2014; Yang and Tsai, 2006) was used in this study to determine the contamination of selected cooked foods when cooked using aluminium cooking utensils. Three food samples namely: fresh tomatoes, rice and cocoyam leaves

(nkontomire) and three different brands of aluminium cookware were selected from a known market (kotokoruba market) in Cape Coast, the capital of Ghana's Central Region. Aluminium concentrations were analyzed in both the raw food commodities and after they were cooked in the aluminium cooking utensils over nine (9) weeks.

Key Findings

The study's principal findings are listed below.

- The findings clearly show that using aluminium utensils in the kitchen contributes significantly to total daily aluminium intake through foods, particularly green leafy vegetables and acidic foods. As presented in the chapters above, among the three selected cooked foods, nkontomire recorded the highest concentration. Also, tomatoes recorded an elevated concentration of aluminium from the utensils and this is due to its acidic nature. In addition, rice contributed little aluminium to the total daily intake compared to tomatoes and nkontomire.
- The chemical makeup of raw food commodities and the numerous components utilized, as well as the conditions of preparation, including the duration and temperature of cooking, all influence the amount of aluminium leached as well as the pH of the food sample.
- The Provisional Tolerable Weekly Intake (PTWI) of 1mg/kg body weight (WHO, 2014), was used as the recommended reference intake with which the concentrations of aluminium obtained in this study were compared. The actual concentration of leached metal ions in the food showed that there was an appreciable amount of

aluminium added to the already existing ones in the food and water. Despite the contamination, the p-values (refer to chapter 4) for all the three commodities under study showed p-values greater than the critical p value, hence the concentration of the aluminium metal in the food over the 9 weeks could be described as not significant. This statistical analysis could be well understood by looking at the concentration recorded (refer to tables 2-4)

Conclusions

Determine the concentration of aluminium in selected raw and cooked foods.

In conclusion, the selected raw foods contained an amount of aluminium due to certain conditions such as the soil used for growing, other growing conditions and farming practices as well as certain methods used in processing these selected food commodities. On the other hand the cooked food samples showed concentrations of aluminium higher than the raw foods, which showed that there was a leach from aluminium utensils into the cooked foods.

Examine the conditions (temperature, pH and cooking time) that may facilitate the leaching of aluminium into cooked foods.

The factors examined showed a positive impact and have been recorded to have contributed to the increased level of aluminium metal concentration, these factors were temperature, pH and cooking duration. All foods were cooked at a high temperature of 100⁰ at which leaching of aluminium is possible. From the selected cooked foods, the study showed that acidic foods tend to cause a leach of the aluminium metal from the

utensil into the food, cereals contribute to less aluminium but a longer cooking period can increase its contamination level. Nkontomire on the other hand like any other green-leafy vegetable also had its contamination exceeding required limits as a result of its high silica and fluoride content.

Compare levels of aluminium leaching from different types of aluminium cooking utensils.

In conclusion, utensil A (used for cooking tomatoes), utensil B (used for cooking nkontomire) and utensil C (used for cooking rice) leached these concentrations of aluminium; $3.16\text{mg/kg} \pm 1.87$, $3.54\text{mg/kg} \pm 1.23$ and $2.67\text{mg/kg} \pm 1.38$ respectively. From these results, utensil B recorded the highest leaching followed by utensil A and lastly utensil C.

Determine the level of aluminium in prepared foods.

Lastly, the level of aluminium in prepared foods showed high concentrations when compared with the PTWI (Provisional Tolerable Weekly Intake) reference limit which is 1mg/kg per body weight according to WHO (2014). The level of aluminium leached into the prepared food samples are: $3.16\text{mg/kg} \pm 1.87$, $3.54\text{mg/kg} \pm 1.23$ and $2.67\text{mg/kg} \pm 1.38$ for tomatoes, nkotomire and rice respectively. A careful look at these levels are higher than the recommended weekly level of intake.

However, the fact remains that once daily ingestion of aluminium surpasses the permissible range, it becomes a health hazard. In furtherance, all homes, caterers, restaurant operators, hotels and other food service operators are to be very cautious about the use of aluminium cookware in cooking and as well be enlightened by regulatory authorities on the associated risk of using such utensils. That notwithstanding, the useful

conclusion is that despite cook wares contribution, there are other sources of contamination hence aluminium could still have its route into the body regardless.

Recommendations

The following recommendations are provided based on the study's primary findings and conclusions.

1. Food and Drugs Authority (FDA) and other Environmental Protection Agencies found in Cape Coast should educate the public and various food vendors and food service operations on the public health hazards associated with using aluminium utensils.
2. To avoid aluminium leaching, utensils made of hard anodized aluminium should be recommended for preparing foods.
3. Although the WHO's tentative daily dosage is 1 mg per kilogram of body weight, it's best not to use aluminium utensils for long periods when preparing acidic foods to avoid metal leaching (WHO, 2014). Excessively acidic foods, such as tomatoes, should not be stored in aluminium pots. The acid in certain foods may induce a higher rate of aluminium leaching into the food than usual, resulting in pitting on the pot's surface.
4. A more analytical method such as Atomic Absorption Spectroscopy (AAS) should be used in future studies in relation in aluminium determination in selected cooked foods.
5. Due to the findings of this study with regards to green leafy vegetables containing high silica and fluoride, further studies should consider the determination of aluminium in green leafy vegetables

such as cabbage, cauliflower, spinach, lettuce cooked in different kinds of utensils.

6. Also, further researches should do a comparative study with other kinds of utensils such as stainless steel, steel and copper to cook foods with high acidic content determine the possible leach of the metal into selected foods.

Suggestions for Further Research

Investigate the levels of aluminum leaching from various types of cookware materials, such as stainless steel, steel, copper, and non-stick coatings, when cooking different types of food, especially those with high acidity. Further researchers can conduct a longitudinal study to examine the long-term health effects of chronic exposure to aluminum through diet, particularly focusing on populations with high usage of aluminum cookware.

REFERENCES

- Adelkhani, H., Nasoodi, S., & Jafari, A. H. (2009). A study of the morphology and optical properties of electropolished aluminium in the Vis-IR region. *Int. J. Electrochem. Sci*, 4(2), 238-246.
- Al Juhaiman, L. A. (2010). Estimating Aluminium leaching from Aluminium cook wares in different meat extracts and milk. *Journal of Saudi Chemical Society*, 14(1), 131-137.
- Al Juhaiman, L. A. (2012). Estimating aluminium leaching from aluminium cookware in different vegetable extracts. *International Journal of Electrochemical Science*, 7(8), 7283-7294.
- Al Juhaiman, L. A., Al-Shihry, R. A., & Al-Hazimi, H. M. (2014). Effect of cardamom extract on leaching of aluminium cookware. *International Journal of Electrochemical Science*, 9(1), 1055-1070
- Al Zubaidy, E. A., Mohammad, F. S., & Bassioni, G. (2011). Effect of pH, salinity and temperature on aluminium cookware leaching during food preparation. *Int. J. Electrochem. Sci*, 6(12), 6424-6441
- Alabi, O. A., & Adeoluwa, Y. M. (2020). Production Usage, and Potential Public Health Effects of Aluminium Cookware: A Review. *Annals of Science and Technology*, 5(1), 20-30
- Alemmari, A., Miller, G. G., Bertolo, R. F., Dinesh, C., Brunton, J. A., Arnold, C. J., & Zello, G. A. (2012). Reduced aluminium contamination decreases parenteral nutrition associated liver injury. *Journal of Pediatric Surgery*, 47(5), 889-894

- Alexandrov, P. N., Pogue, A. I., & Lukiw, W. J. (2018). Synergism in aluminium and mercury neurotoxicity. *Integrative Food, Nutrition and Metabolism*, 5(3), 22-34.
- Alfrey, A. C., LeGendre, G. R., & Kaehny, W. D. (1976). The dialysis encephalopathy syndrome: possible aluminium intoxication. *New England Journal of Medicine*, 294(4), 184-188.
- Aliaga, M. and Gunderson, B. (2002) *Interactive Statistics*. Thousand Oaks, CA: Sage Publications.
- Altmann, P., Cunningham, J., Dhanesha, U., Ballard, M., Thompson, J., & Marsh, F. (1999). Disturbance of cerebral function in people exposed to drinking water contaminated with aluminium sulphate: retrospective study of the Camelford water incident. *Bmj*, 319(7213), 807-811.
- Amarasooriya, A. A. G. D., & Dharmagunawardhane, H. A. (2014). Leaching of aluminium and its incorporation to rice during cooking under different fluoride concentrations in water. In *SAITM Research Symposium on Engineering Advancements* (Vol. 3, pp. 213-219)
- Ankar-Brewoo, G., Darko, G., Abaidoo, R., Dalsgaard, A., Johnson, P. N., Ellis, W., & Brimer, L. (2020). Health risks of toxic metals (Al, Fe and Pb) in two common street vended foods, fufu and fried-rice, in Kumasi, Ghana. *Scientific African*, 1(2), 2-11
- Anke, M., Ihnat, M., & Stoeppler, M. (Eds.). (2004). *Elements and their compounds in the environment: occurrence, analysis and biological relevance* (Vol. 3). Weinheim, Germany: Vch Verlagsgesellschaft MbH

- Apuke, O. D. (2017). Quantitative research methods: A synopsis approach. *Kuwait Chapter of Arabian Journal of Business and Management Review*, 33(5471), 1-8
- Arvanitoyannis, I. S., & Kotsanopoulos, K. V. (2014). Migration phenomenon in food packaging. Food–package interactions, mechanisms, types of migrants, testing and relative legislation—a review. *Food and Bioprocess Technology*, 7(1), 21-36
- Bache, B. W., & Sharp, G. S. (1976). Soluble polymeric hydroxy-aluminium ions in acid soils. *Journal of Soil Science*, 27(2), 167-174.
- Bannerman, W. K. (1996). *Determination of aluminium in some raw foods and treated water from Kumasi and its leaching from aluminium pots during cooking* (Kwame Nkrumah University of Science and Technology, Doctoral dissertation)
- Barabasz, W., Albinska, D., Jaskowska, M., & Lipiec, J. (2002). Ecotoxicology of aluminium. *Polish Journal of Environmental Studies*, 11(3), 199-204.
- Bartlett, R. J., & Riego, D. C. (1972). Effect of chelation on the toxicity of aluminium. *Plant and Soil*, 37(2), 419-423.
- Baxter, M., Burrell, J. A., & Massey, R. C. (1988). The effects of fluoride on the leaching of aluminium saucepans during cooking. *Food Additives & Contaminants*, 5(4), 651-656
- Becaria, A., Campbell, A., & Bondy, S. C. (2002). Aluminium as a toxicant. *Toxicology and Industrial Health*, 18(7), 309-320.

- Bennet, R. J., & Breen, C. M. (1991). The aluminium signal: new dimensions to mechanisms of aluminium tolerance. *Plant and Soil*, 134(1), 153-166.
- Bennet, R. J., Breen, C. M., & Fey, M. V. (1985). Aluminium induced changes in the morphology of the quiescent centre, proximal meristem and growth region of the root of *Zea mays*. *South African Journal of Botany*, 51(5), 355-362.
- Blair, L. M., & Taylor, G. J. (1997). The nature of interaction between aluminium and manganese on growth and metal accumulation in *Triticum aestivum*. *Environmental and Experimental Botany*, 37(1), 25-37.
- Bradley, E. L., & Castle, L. (2021). Residues of food contact materials. In *Handbook of Dairy Foods Analysis* (pp. 807-832). CRC Press
- Bratakos, S. M., Lazou, A. E., Bratakos, M. S., & Lazos, E. S. (2012). Aluminium in food and daily dietary intake estimate in Greece. *Food Additives and Contaminants: Part B*, 5(1), 33-44.
- Cape Coast Metropolitan Assembly (CCMA). (2022, February). Cape Coast Metropolitan Assembly: Cape Coast metropolis. Retrieved from: <http://ccma.gov.gh/aboutccma#> 2nd February, 2022
- Carpenter, D. O., Arcaro, K., & Spink, D. C. (2002). Understanding the human health effects of chemical mixtures. *Environmental Health Perspectives*, 110(suppl 1), 25-42.
- Caylak, E. (2012). Health risk assessment for trace metals, polycyclic aromatic hydrocarbons and trihalomethanes in drinking

- water of Cankiri, Turkey. *E-Journal of Chemistry*, 9(4),1976-1991
- Chusid, J. G., Pacella, B. L., Kopeloff, L. M., & Kopeloff, N. (1951). Chronic epilepsy in the monkey following multiple intracerebral injections of alumina cream. *Proceedings of the Society for Experimental Biology and Medicine*, 78(1), 53-54.
- Clark, R. B., Pier, P. A., Knudsen, D., & Maranville, J. W. (1981). Effect of trace element deficiencies and excesses on mineral nutrients in sorghum. *Journal of Plant Nutrition*, 3(1-4), 357-374.
- Clarkson, D. T., & Sanderson, J. (1971). Inhibition of the uptake and long-distance transport of calcium by aluminium and other polyvalent cations. *Journal of Experimental Botany*, 22(4), 837-851.
- Cochran, M., Goddard, G., & Ludwigson, N. (1990). Aluminium absorption by rat duodenum: further evidence of energy-dependent uptake. *Toxicology Letters*, 51(3), 287-294
- Cohen, M. D. (2004). Pulmonary immunotoxicology of select metals: aluminium, arsenic, cadmium, chromium, copper, manganese, nickel, vanadium, and zinc. *Journal of Immunotoxicology*, 1(1), 39-69.
- Creswell, J. W., & Creswell, J. D. (2017). *Research design: Qualitative, quantitative, and mixed methods approaches*. Thousand Oaks, CA: Sage publications
- Crisponi, G., Nurchi, V. M., Faa, G., & Remelli, M. (2010). Human diseases related to aluminium overload. *Monatshefte für Chemie-Chemical Monthly*, 142(4), 331-340.

- Dabonne, S., Koffi, B., Kouadio, E., Koffi, A., Due, E., & Kouame, L. (2010). Traditional utensils: potential sources of poisoning by heavy metals. *British Journal of Pharmacology and Toxicology*, 1(2), 90-92.
- Dan, E. U., & Ebong, G. A. (2013). Impact of cooking utensils on trace metal levels of processed food items. *Ann. Food Sci. Technol*, 14(2), 350-355.
- Darbre, P. D. (2001). Underarm cosmetics are a cause of breast cancer. *European Journal of Cancer Prevention*, 10(5), 389-393
- Darbre, P. D. (2003). Underarm cosmetics and breast cancer. *Journal of Applied Toxicology: An International Journal*, 23(2), 89-95
- Darbre, P. D. (2005). Aluminium, antiperspirants and breast cancer. *Journal of Inorganic Biochemistry*, 99(9), 1912-1919.
- Darbre, P. D. (2005). Recorded quadrant incidence of female breast cancer in Great Britain suggests a disproportionate increase in the upper outer quadrant of the breast. *Anticancer Research*, 25(3C), 2543-2550.
- De Graaf, M. C., Bobbink, R., Verbeek, P. J., & Roleofs, J. G. (1997). Aluminium toxicity and tolerance in three heathland species. *Water, Air, and Soil Pollution*, 98(3), 229-239..
- Dedov, V. M., & Klimasshevskii, E. L. (1976). On the mechanism of genotypic resistance of plants to Al^{3+} . 2. *Effect of Al Ions on pH Redox Potential and the Content of High Energy Phosphorous in Root Tissues of Peas*, *Sib. Vestn. Skh. Nauki*, 3(1), 13-16.

Delhaize, E., & Ryan, P. R. (1995). Aluminium toxicity and tolerance in plants. *Plant Physiology*, 107(2), 315.

Department of Health and Human Services: Division of Toxicology and Environmental medicine, (2008). *Department of health and human sciences-public health service agency for toxic substances and disease registry*, pg 2-7.

Díaz-Nido, J., & Avila, J. (1990). Aluminium induces the in vitro aggregation of bovine brain cytoskeletal proteins. *Neuroscience Letters*, 110(1-2), 221-226.

Dordevic, D., Buchtova, H., Jancikova, S., Macharackova, B., Jarosova, M., Vitez, T., & Kushkevych, I. (2019). Aluminum contamination of food during culinary preparation: Case study with aluminum foil and consumers' preferences. *Food Science & Nutrition*, 7(10), 3349-3360

Edwards, J. H., Horton, B. D., & Kirkpatrick, H. C. (1976). Aluminium toxicity symptoms in peach seedlings. *J. Am. Soc. Hortic. Sci.:(United States)*, 101(2), 22-29.

El-Sayed, S. M., & Farag, S. E. (2008). Investigatory Survey Of The Extent Of Contamination Of Food By Aluminium. *Journal of Food and Dairy Sciences*, 33(8), 5825-5837

El-Sayed, W. M., Al-Kahtani, M. A., & Abdel-Moneim, A. M. (2011). Prophylactic and therapeutic effects of taurine against aluminium-induced acute hepatotoxicity in mice. *Journal of Hazardous Materials*, 192(2), 880-886.

Ertl, K., & Goessler, W. (2018). Aluminium in foodstuff and the influence

of aluminium foil used for food preparation or short time storage.

Food Additives & Contaminants: Part B, 11(2), 153-159

Eskin, D. G. (2008). *Physical metallurgy of direct chill casting of aluminium alloys*. 9(10), 323-340.

European Food Safety Authority (EFSA). (2008). Safety of aluminium from dietary intake. Scientific Opinion of the Panel on Food Additives, Flavourings, Processing Aids and Food Contact Materials (AFC). *EFSA Journal*, 6(7), 754-762

Exley, C. (2014). Aluminium adjuvants and adverse events in sub-cutaneous allergy immunotherapy. *Allergy, Asthma & Clinical Immunology*, 10(1), 1-5.

Exley, C., & Vickers, T. (2014). Elevated brain aluminium and early onset Alzheimer's disease in an individual occupationally exposed to aluminium: a case report. *Journal of Medical Case reports*, 8(1), 1-3.

Fairweather-Tait, S. J., Faulks, R. M., Fatemi, S. J. A., & Moore, G. R. (1987). Aluminium in the diet. *Human Nutrition. Food Sciences and Nutrition*, 41(3-4), 183-192

Fakri, S., Al Azzawi, A., & Al Tawil, N. (2006). Antiperspirant use as a risk factor for breast cancer in Iraq. *EMHJ-Eastern Mediterranean Health Journal*, 12(2), 478-482, 2006.

Fekete, V., Deconinck, E., Bolle, F., & Van Loco, J. (2012). Modelling aluminium leaching into food from different foodware materials with multi-level factorial design of experiments. *Food Additives & Contaminants: Part A*, 29(8), 1322-1333

- Fekete, V., Vandevijvere, S., Bolle, F., & Van Loco, J. (2013). Estimation of dietary aluminium exposure of the Belgian adult population: evaluation of contribution of food and kitchenware. *Food and Chemical Toxicology*, 55, 602-608.
- Flarend, R., Bin, T., Elmore, D., & Hem, S. L. (2001). A preliminary study of the dermal absorption of aluminium from antiperspirants using aluminium. *Food and Chemical Toxicology*, 39(2), 163-168.
- Flaten, T. P. (2001). Aluminium as a risk factor in Alzheimer's disease, with emphasis on drinking water. *Brain Research Bulletin*, 55(2), 187-196.
- Forbes, W. F., & McLachlan, D. R. (1996). Further thoughts on the aluminium- Alzheimer's disease link. *Journal of Epidemiology & Community Health*, 50(4), 401-403.
- Foy, C. D. (1974). Effects of aluminium on plant growth. *The Plant Root and its Environment*. 2(9), 23-40.
- Foy, C. D., & Brown, J. C. (1964). Toxic factors in acid soils: II. Differential aluminium tolerance of plant species. *Soil Science Society of America Journal*, 28(1), 27-32.
- Foy, C. D., Chaney, R. T., & White, M. C. (1978). The physiology of metal toxicity in plants. *Annual Review of Plant Physiology*, 29(1), 511-566.
- Foy, C. D., Fleming, A. L., & Armiger, W. H. (1969). Aluminium Tolerance of Soybean Varieties in Relation to Calcium Nutrition 1. *Agronomy Journal*, 61(4), 505-511.

- Frecker, M. F. (1991). Dementia in Newfoundland: identification of a geographical isolate?. *Journal of Epidemiology & Community Health*, 45(4), 307-311.
- Fronterhouse, J. J. (2014). *Potassium and magnesium roles and food intake analysis*. Oklahoma State University
- Fu, H. J., Hu, Q. S., Lin, Z. N., Ren, T. L., Song, H., Cai, C. K., & Dong, S. Z. (2003). Aluminium-induced apoptosis in cultured cortical neurons and its effect on SAPK/JNK signal transduction pathway. *Brain Research*, 980(1), 11-23.
- Fulgenzi, A., Vietti, D., & Ferrero, M. E. (2014). Aluminium involvement in neurotoxicity. *BioMed Research International*, 1(2), 33-43
- Geographical relation between Alzheimer's disease and aluminium in drinking water. *The Lancet*, 333(8629), 59-62.
- Ghana Standards Authority (2006), Standard Procedure for Analysis of Fruit and Vegetable Products-Determination of pH (GS ISO 1842:1991).
- Ghanati, F., Morita, A., & Yokota, H. (2005). Effects of aluminium on the growth of tea plant and activation of antioxidant system. *Plant and Soil*, 276(1), 133- 141.
- González-Weller, D., Gutiérrez, A. J., Rubio, C., Revert, C., & Hardisson, A. (2010). Dietary intake of aluminium in a Spanish population (Canary Islands). *Journal of Agricultural and Food Chemistry*, 58(19), 10452-10457.
- Greger, J. L. (1993). Aluminium metabolism. *Annual Review of Nutrition*, 13(1), 43-63

- Greger, J. L., Sutherland, J. E., & Yokel, R. (1997). Aluminium exposure and metabolism. *Critical Reviews in Clinical Laboratory Sciences*, 34(5), 439-474.
- Guillard, O., Fauconneau, B., Olichon, D., Dedieu, G., & Deloncle, R. (2004). Hyeraluminemia in a woman using an aluminium-containing antiperspirant for 4 years. *The American Journal of Medicine*, 117(12), 956-959.
- Gupta, N., Gaurav, S. S., & Kumar, A. (2013). Molecular basis of aluminium toxicity in plants: a review. *American Journal of Plant Sciences*, 2(1), 23-35
- Hassan, M. F., Sadek, M. A., Abd-El-Razik, F. H., & Kamel, E. A. (2008). Risk of aluminium toxicity and its relation to some biochemical changes in healthy, diabetic and hyperlepidemic rats. *Egyptian Journal of Natural Toxins*, 5(1-2), 100-120
- Hauser, G., Curiel, G. J., Bellin, H. W., Cnossen, H. J., Hofman, J., Kastelein, J., ... & Timberley, A. W. (2004). Hygienic equipment design criteria. *EHEDG Doc 8, SG Design Principles*
- Henson, R. N. (2015). Analysis of variance (ANOVA). *Brain Mapping: an Encyclopedic Reference*. Amsterdam, Netherlands: Elsevier.
- Herrman, J. L., & Younes, M. (1999). Background to the adi/tdi/ptwi. *Regulatory Toxicology and Pharmacology*, 30(2), 109-113
- Huang, J. W., Grunes, D. L., & Kochian, L. V. (1992). Aluminium effects on the kinetics of calcium uptake into cells of the wheat root apex. *Planta*, 188(3), 414-421.

- Ihnat, M. (2001). Twenty-five years of reference material activity at Agriculture and Agri-Food Canada. *Fresenius' Journal of Analytical Chemistry*, 370(2), 279-285
- Jabeen, S., Ali, B., Ali Khan, M., Bilal Khan, M., & Adnan Hasan, S. (2016). Aluminium intoxication through leaching in food preparation. *Alexandria Science Exchange Journal*, 37(3), 618-626.
- Jacobs, D. R., & Tapsell, L. C. (2007). Food, not nutrients, is the fundamental unit in nutrition. *Nutrition Reviews*, 65(10), 439-450
- JECFA-Joint, F.A.O., WHO Expert Committee on Food Additives & World Health Organization. (1989). *Toxicological evaluation of certain food additives and contaminants. WHO Food Additives Series 24*, pp 113–154
- Jederlinic, P. J., Abraham, J. L., Churg, A., Himmelstein, J. S., Epler, G. R., & Gaensler, E. A. (1990). Pulmonary fibrosis in aluminium oxide workers. *Am Rev Respir Dis*, 142(5), 1179-84
- Jeffery, E. H., Abreo, K., Burgess, E., Cannata, J., & Greger, J. L. (1996). Systemic aluminium toxicity: effects on bone, hematopoietic tissue, and kidney. *Journal of Toxicology and Environmental Health Part A*, 48(6), 649-666.
- Joint FAO/WHO Expert Committee on Food Additives. Meeting, & World Health Organization. (2014). *Safety Evaluation of Certain Food Additives and Contaminants* (Vol. 68). World Health Organization
- Joint, F. A. O., WHO Expert Committee on Food Additives, & World Health Organization. (2007). *Evaluation of certain food additives*

- and contaminants: sixty-eighth report of the Joint FAO/WHO Expert Committee on Food Additives.* World Health Organization Joint, F. A. O., World Health Organization, & WHO Expert Committee on Food Additives. (2011). *Evaluation of certain food additives and contaminants: seventy-third [73rd] report of the Joint FAO/WHO Expert Committee on Food Additives.* World Health Organization
- Karbouj, R. (2007). Aluminium leaching using chelating agents as compositions of food. *Food and Chemical Toxicology*, 45(9), 1688-1693.
- Kaur, A., & Gill, K. D. (2005). Disruption of neuronal calcium homeostasis after chronic aluminium toxicity in rats. *Basic & Clinical Pharmacology & Toxicology*, 96(2), 118-122.
- Kawahara, M., & Kato-Negishi, M. (2011). Link between aluminium and the pathogenesis of Alzheimer's disease: the integration of the aluminium and amyloid cascade hypotheses. *International Journal of Alzheimer's Disease*, 2(1), 112-120.
- Kayama, M. (2001). Comparison of the aluminium tolerance of *Miscanthus sinensis* Anderss. and *Miscanthus sacchariflorus* Benth in hydroculture. *International Journal of Plant Sciences*, 162(5), 1025-1031.
- Keltjens, W. G., & Tan, K. (1993). Interactions between aluminium, magnesium and calcium with different monocotyledonous and dicotyledonous plant species. In *Plant Nutrition—from Genetic Engineering to Field Practice* (pp. 719-722). Springer, Dordrecht.

- Kidd, P. S., & Proctor, J. (2000). Effects of aluminium on the growth and mineral composition of *Betula pendula* Roth. *Journal of Experimental Botany*, 51(347), 1057-1066.
- Kiesswetter, E., Schäper, M., Buchta, M., Schaller, K. H., Rossbach, B., Scherhag, H., ... & Letzel, S. (2007). Longitudinal study on potential neurotoxic effects of aluminium: I. Assessment of exposure and neurobehavioural performance of Al welders in the train and truck construction industry over 4 years. *International Archives of Occupational and Environmental Health*, 81(1), 41-67.
- Kinraide, T. B., Ryan, P. R., & Kochian, L. V. (1992). Interactive effects of Al^{3+} , H^{+} , and other cations on root elongation considered in terms of cell-surface electrical potential. *Plant Physiology*, 99(4), 1461-1468.
- Klotz, K., Weistenhöfer, W., Neff, F., Hartwig, A., van Thriel, C., & Drexler, H. (2017). The health effects of aluminium exposure. *Deutsches Ärzteblatt International*, 114(39), 653-667
- Koning, J. H. (1981). Aluminium pots as a source of dietary aluminium. *The New England Journal of Medicine*, 304(3), 172-173
- Kraus, T., Schaller, K. H., Angerer, J., Hilgers, R. D., & Letzel, S. (2006). Aluminosis—detection of an almost forgotten disease with HRCT. *Journal of Occupational Medicine and Toxicology*, 1(1), 1-9.
- Krewski, D., Yokel, R. A., Nieboer, E., Borchelt, D., Cohen, J., Harry, J. & Rondeau, V. (2007). Human health risk assessment for aluminium,

- aluminium oxide, and aluminium hydroxide. *Journal of Toxicology and Environmental Health, Part B*, 10(1), 1-269.
- Krizek, D. T., & Foy, C. D. (1988). Mineral element concentration of two barley cultivars in relation to water deficit and aluminium toxicity. *Journal of Plant Nutrition*, 11(4), 369-386.
- Kumar, V., & Gill, K. D. (2014). Oxidative stress and mitochondrial dysfunction in aluminium neurotoxicity and its amelioration: a review. *Neurotoxicology*, 41(1), 154-166.
- Landry, K. (2014). Human health effects of dietary aluminium. *Interdisciplinary Journal of Health Sciences*, 4(1), 39-44
- Lankoff, A., Banasik, A., Duma, A., Ochniak, E., Lisowska, H., Kuszewski, T., ... & Wojcik, A. (2006). A comet assay study reveals that aluminium induces DNA damage and inhibits the repair of radiation-induced lesions in human peripheral blood lymphocytes. *Toxicology Letters*, 161(1), 27-36.
- Lavrakas, P. J. (2008). *Encyclopedia of survey research methods*. Thousand Oaks, CA: Sage publications
- Leedy, P. D., & Ormrod, J. E. (2019). *Practical research: Planning and design*. Upper Saddle River, NJ: Pearson.
- Levick, S. E. (1980). Dementia from aluminium pots? *The New England Journal of Medicine*, 303(3), 76-89
- Luis, G., Rubio, C., Revert, C., Espinosa, A., González-Weller, D., Gutiérrez, A. J., & Hardisson, A. (2015). Dietary intake of metals from yogurts analyzed by inductively coupled plasma optical

- emission spectrometry (ICP-OES). *Journal of Food Composition and Analysis*, 39(1), 48-54.
- Ma, N., Liu, Z. P., Yang, D. J., Liang, J., Zhu, J. H., Xu, H. B., & Li, N. (2016). Risk assessment of dietary exposure to aluminium in the Chinese population. *Food Additives & Contaminants: Part A*, 33(10), 1557-1562
- Macmillan, D. W. (Ed.). (2017). *Proceedings of the International Symposium on Quality and Process Control in the Reduction and Casting of Aluminium and Other Light Metals, Winnipeg, Canada, August 23–26, 1987: Proceedings of the Metallurgical Society of the Canadian Institute of Mining and Metallurgy*. Amsterdam, Netherlands: Elsevier
- Mahieu, S. T., Navoni, J., Millen, N., del Carmen Contini, M., Gonzalez, M., & Elías, M. M. (2004). Effects of aluminium on phosphate metabolism in rats: a possible interaction with vitamin D 3 renal production. *Archives of Toxicology*, 78(11), 609-616.
- Mahieu, S., Millen, N., González, M., del Carmen Contini, M., & Elías, M. M. (2005). Alterations of the renal function and oxidative stress in renal tissue from rats chronically treated with aluminium during the initial phase of hepatic regeneration. *Journal of Inorganic Biochemistry*, 99(9), 1858-1864.
- Malkanthi, D. R. R., Yokoyama, K., Yoshida, T., Moritsugu, M., & Matsushita, K. (1995). Effects of low pH and AI on growth and nutrient uptake of several plants. *Soil Science and Plant Nutrition*, 41(1), 161-165.

- Martinez, C. S., Alterman, C. D., Peçanha, F. M., Vassallo, D. V., Mello-Carpes, P. B., Miguel, M., & Wiggers, G. A. (2017). Aluminium exposure at human dietary levels for 60 days reaches a threshold sufficient to promote memory impairment in rats. *Neurotoxicity Research*, 31(1), 20-30.
- Martyn, C. N., Osmond, C., Edwardson, J. A., Barker, D. J. P., Harris, E. C., & Lacey, R. F. (1989). Geographical relation between Alzheimers's disease and aluminium in drinking water. *The Lancet*, 333(8629), 59-62
- McColl, J. G., Waldren, R. P., Wafula, N. J., & Sigunga, D. O. (1991). Aluminium effects on six wheat cultivars in Kenyan soils. *Communications in Soil Science and Plant Analysis*, 22(15), 1701-1719.
- McGrath, K. G. (2003). An earlier age of breast cancer diagnosis related to more frequent use of antiperspirants/deodorants and underarm shaving. *European Journal of Cancer Prevention*, 3(2) 479-485.
- Moerman, F., & Partington, E. (2014). Materials of construction for food processing equipment and services: requirements, strengths and weaknesses. *J Hyg Eng Design*, 6(2), 10-23
- Mohammad, F. S., Al Zubaidy, E. A. H., & Bassioni, G. (2011). Effect of aluminium leaching process of cooking wares on food. *International Journal of Electrochemical Science*, 6(1), 222-230.

- Mooney, S. M., & Miller, M. W. (2000). Expression of bcl-2, bax, and caspase-3 in the brain of the developing rat. *Developmental Brain Research*, 123(2), 103-117.
- Mullette, K. J. (1975). Stimulation of growth in Eucalyptus due to aluminium. *Plant and Soil*, 42(2), 495-499.
- Namer, M., Luporsi, E., Gligorov, J., Lokiec, F., & Spielmann, M. (2008). The use of deodorants/antiperspirants does not constitute a risk factor for breast cancer. *Bulletin du Cancer*, 95(9), 871-880.
- Neri, L., & Hewitt, D. (1991). Aluminium, Alzheimer's disease, and drinking water. *The Lancet*, 338(8763), 390.
- Nichol, B. E., & Oliveira, L. A. (1995). Effects of aluminium on the growth and distribution of calcium in roots of an aluminium-sensitive cultivar of barley (*Hordeum vulgare*). *Canadian Journal of Botany*, 73(12), 1849-1858.
- Nichol, B. E., Oliveira, L. A., Glass, A. D., & Siddiqi, M. Y. (1993). The effects of aluminium on the influx of calcium, potassium, ammonium, nitrate, and phosphate in an aluminium-sensitive cultivar of barley (*Hordeum vulgare* L.). *Plant Physiology*, 101(4), 1263-1266.
- Niu, Q. (2018). Overview of the relationship between aluminium exposure and health of human being. *Neurotoxicity of Aluminium*, 2(1), 1-31.
- Njenga, L. W. and Kariuki, D. N. and Ndegwa, S. 2005. Water labile fluoride in fresh raw vegetable markets from markets in Nairobi, Kenya. *Fluoride* 38(3), 205-205.

- Njenga, L. W., Maina, D. M., Kariuki, D. N., & Mwangi, F. K. (2007). Aluminium exposure from vegetables and fresh raw vegetable juices in Kenya. *Journal Of Food Agriculture and Environment*, 5(1), 8-15
- Nosko, P., Brassard, P., Kramer, J. R., & Kershaw, K. A. (1988). The effect of aluminium on seed germination and early seedling establishment, growth, and respiration of white spruce (*Picea glauca*). *Canadian Journal of Botany*, 66(11), 2305-2310.
- Ogawa, M., & Kayama, F. (2015). A study of the association between urinary aluminium concentration and pre-clinical findings among aluminium-handling and non-handling workers. *Journal of Occupational Medicine and Toxicology*, 10(1), 1-7.
- Ordog, G. (2005). 325 Aluminium Toxicity in Aluminium Factory Workers, Treated with Dmsa Chelation. *BMJ Journal*, 1(3), 33-45
- Osaki, M., Watanabe, T., Ishizawa, T., Nilnond, C., Nuyim, T., Sittibush, C., & Tadano, T. (1998). Nutritional characteristics in leaves of native plants grown in acid sulfate, peat, sandy podzolic, and saline soils distributed in Peninsular Thailand. *Plant and Soil*, 201(2), 175-182.
- Ott, S. M., Maloney, N. A., Klein, G. L., Alfrey, A. C., Ament, M. E., Coburn, J. W., & Sherrard, D. J. (1983). Aluminium is associated with low bone formation in patients receiving chronic parenteral nutrition. *Annals of Internal Medicine*, 98(6), 910-91

- Parhad, I. M., Krekoski, C. A., Mathew, A., & Tran, P. M. (1989). Neuronal gene expression in aluminium myelopathy. *Cellular and Molecular Neurobiology*, 9(1), 123-138
- Parkinson, I. S., Ward, M. K., & Kerr, D. (1981). Dialysis encephalopathy, bone disease and anaemia: the aluminium intoxication syndrome during regular haemodialysis. *Journal of Clinical Pathology*, 34(11), 12-21.
- Pasha, A., & Oglu, A. (2017). Investigation of oxidative effect of aluminium in Albino rats. *MedBioTech Journal*, 1(02), 86-91.
- Pennington, J. A. (1987). Aluminium content of foods and diets. *Food Additives & Contaminants*, 5(2), 161-232.
- Pennington, J. A. T., & Jones, J. W. (1989). Aluminium in health: A critical review. *New York*.
- Pilon-Smits, E. A., Quinn, C. F., Tapken, W., Malagoli, M., & Schiavon, M. (2009). Physiological functions of beneficial elements. *Current Opinion in Plant Biology*, 12(3), 267-274.
- Rabinovich, D. (2013). The allure of aluminium. *Nature Chemistry*, 5(1), 76-76.
- Rahman, M. S. (2009). *Food properties handbook*. Boca Raton, FL: CRC press
- Rajwanshi, P., Singh, V., Gupta, M. K., Kumari, V., Shrivastav, R., Ramanamurthy, M., & Dass, S. (1997). Studies on aluminium leaching from cookware in tea and coffee and estimation of aluminium content in toothpaste, baking powder and paan masala. *Science of the Total Environment*, 193(3), 243-249

- Ranau, R., Oehlenschläger, J., & Steinhart, H. (2001). Aluminium levels of fish fillets baked and grilled in aluminium foil. *Food Chemistry*, 73(1), 1-6
- Rhue, R. D., & Grogan, C. O. (1976). Screening corn for aluminium tolerance. In *Plant adaptation to mineral stress in problem soils. Proceedings of a workshop held at the National Agricultural Library, Beltsville, Maryland, November 22-23, 1976* (pp. 297-310). Cornell Univ. Agricultural Experiment Station.
- Ribeiro, C. P., McKay, R. R., Hosoki, E., Bird, G. S. J., & Putney, J. W. (2000). Effects of elevated cytoplasmic calcium and protein kinase C on endoplasmic reticulum structure and function in HEK293 cells. *Cell Calcium*, 27(3), 175-185.
- Riihimäki, V., Hänninen, H., Akila, R., Kovala, T., Kuosma, E., Paakkulainen, H., ... & Engström, B. (2000). Body burden of aluminium in relation to central nervous system function among metal inert-gas welders. *Scandinavian Journal of Work, Environment & Health*, 2(2), 118-130.
- Rincón, M., & Gonzales, R. A. (1992). Aluminium partitioning in intact roots of aluminium-tolerant and aluminium-sensitive wheat (*Triticum aestivum* L.) cultivars. *Plant Physiology*, 99(3), 1021-1028.
- Robberecht, H., Van Dyck, K., Bosscher, D., & Van Cauwenbergh, R. (2008). Silicon in foods: content and bioavailability. *International Journal of Food Properties*, 11(3), 638-645

- Rondeau, V., Commenges, D., Jacqmin-Gadda, H., & Dartigues, J. F. (2000). Relation between aluminium concentrations in drinking water and Alzheimer's disease: an 8-year follow-up study. *American Journal of Epidemiology*, 152(1), 59-66.
- Rutherford, A. (2011). *ANOVA and ANCOVA: a GLM approach*. John Wiley & Sons
- Saiyed, S. M., & Yokel, R. A. (2005). Aluminium content of some foods and food products in the USA, with aluminium food additives. *Food Additives and Contaminants*, 22(3), 234-244
- Sangale, M., Daptare, S., & Sonawane, V. (2014). Determination of aluminium and magnesium ions in some commercial adsorptive anatacids by complexometric titrations. *International Journal of Advanced Scientific and Technical Research*, 4(4), 452-465
- Saunders, M., Lewis, P., & Thornhill, A. (2009). *Research methods for business students*. Harlow, England: Pearson education
- Savory, J., Nicholson, J. R., & Wills, M. R. (1987). Is aluminium leaching enhanced by fluoride? *Nature*, 327(6118), 107-108
- Schifman, R. B., & Luevano, D. R. (2018). Aluminium toxicity: evaluation of 16-year trend among 14 919 patients and 45 480 results. *Archives of Pathology & Laboratory Medicine*, 142(6), 742-746.
- Semwal, A. D., Padmashree, A., Khan, M. A., Sharma, G. K., & Bawa, A. S. (2006). Leaching of aluminium from utensils during cooking of food. *Journal of the Science of Food and Agriculture*, 86(14), 2425-2430

- Sharma, P., & Mishra, K. P. (2006). Aluminium-induced maternal and developmental toxicity and oxidative stress in rat brain: response to combined administration of Tiron and glutathione. *Reproductive Toxicology*, 21(3), 313-321.
- Shati, A. A., & Alamri, S. A. (2010). Role of saffron (*Crocus sativus* L.) and honey syrup on aluminium-induced hepatotoxicity. *Saudi Med J*, 31(10), 1106-1113.
- Smith, S. W. (2013). The role of chelation in the treatment of other metal poisonings. *Journal of Medical Toxicology*, 9(4), 355-369.
- Sorenson, J. R., Campbell, I. R., Tepper, L. B., & Lingg R. D. (1974). Aluminium in the environment and human health. *Environ Health Perspect*, 8(1), 3-95.
- Spencer, H., Kramer, L., Norris, C., & Osis, D. (1982). Effect of small doses of aluminium-containing antacids on calcium and pHosphorus metabolism. *The American Journal of Clinical Nutrition*, 36(1), 222-234
- Spofforth, J. (1921). Case of aluminium poisoning. *The Lancet*, 197(53), 130-142
- Stahl, T., Falk, S., Rohrbeck, A., Georgii, S., Herzog, C., Wiegand, A., & Brunn, H. (2017). Migration of aluminium from food contact materials to food—a health risk for consumers? Part I of III: exposure to aluminium, release of aluminium, tolerable weekly intake (TWI), toxicological effects of aluminium, study design, and methods. *Environmental Sciences Europe*, 29(1), 1-8.

- Stahl, T., Taschan, H., & Brunn, H. (2011). Aluminium content of selected foods and food products. *Environmental Sciences Europe*, 23(2), 1-11
- Statistics Solutions. (2013). ANOVA. Retrieved from <https://www.statisticssolutions.com/free-resources/directory-of-statistical-analyses/anova/>
- Sullivan, D. M., Kehoe, D. F., & Smith, R. L. (1987). Measurement of trace levels of total aluminium in foods by atomic absorption spectrophotometry. *Journal of the Association of Official Analytical Chemists*, 70(1), 118-120.
- Tabachnick, B. G., & Fidell, L. S. (2007). *Experimental designs using ANOVA* (Vol. 724). Belmont, CA: Thomson/Brooks/Cole.
- Tennakone, K., & Wickramanayake, S. (1987). Aluminium leaching from cooking utensils. *Nature*, 325(6101), 202-202
- Thakur, R. R. S., McMillan, H. L., & Jones, D. S. (2014). Solvent induced pHase inversion-based in situ forming controlled release drug delivery implants. *Journal of Controlled Release*, 176(8), 8-23.
- Therien, A. G., & Blostein, R. (2000). Mechanisms of sodium pump regulation. *American Journal of Physiology-Cell Physiology*, 279(3), 541-566.
- Tomlins, K., & Johnson, P. (2009). Developing food safety strategies and procedures through reduction of food hazards in street-vended foods to improve food security for consumers, street food vendors and input suppliers. Project Final Report.

- Trap, G. A., & Cannon, J. B. (1981). Aluminium pots as a source of dietary aluminium. *N Engl J Med*, 304(77), 172-184
- Van Praag, H. J., Weissen, F., Sougnez-Remy, S., & Carletti, G. (1985). Aluminium effects on spruce and beech seedlings. *Plant and Soil*, 83(3), 339-356
- Wagatsuma, T., Kaneko, M., & Hayasaka, Y. (1987). Destruction process of plant root cells by aluminium. *Soil Science and Plant Nutrition*, 33(2), 161-175.
- Walton, J. R. (2007). An aluminium-based rat model for Alzheimer's disease exhibits oxidative damage, inhibition of PP2A activity, hyperphosphorylated tau, and granulovacuolar degeneration. *Journal of Inorganic Biochemistry*, 101(9), 1275-1284.
- Wang, W., Zhao, X. Q., Hu, Z. M., Shao, J. F., Che, J., Chen, R. F., ... & Shen, R. F. (2015). Aluminium alleviates manganese toxicity to rice by decreasing root symplastic Mn uptake and reducing availability to shoots of Mn stored in roots. *Annals of Botany*, 116(2), 237-246.
- Ward, R. J., Zhang, Y., & Crichton, R. R. (2001). Aluminium toxicity and iron homeostasis. *Journal of Inorganic Biochemistry*, 87(1-2), 9-14.
- Watanabe, T., Jansen, S., & Osaki, M. (2005). The beneficial effect of aluminium and the role of citrate in Al accumulation in *Melastoma malabathricum*. *New Phytologist*, 3(1), 773-780.
- Wechphanich, S., & Thammarat, P. (2017). A survey of metal contamination in blood collection tubes on toxicology assays. *The Bangkok Medical Journal*, 13(2), 5-5.

- Weidenhamer, J. D., Fitzpatrick, M. P., Biro, A. M., Kobunski, P. A., Hudson, M. R., Corbin, R. W., & Gottesfeld, P. (2017). Metal exposures from aluminium cookware: an unrecognized public health risk in developing countries. *Science of the Total Environment*, 579 (2), 805-813
- Weisser, K., Heymans, L., & Keller-Stanislawski, B. (2015). Paul-Ehrlich Institut: Sicherheitsbewertung von Aluminium in Impflösungen. *Bulletin zur Arzneimittelsicherheit*, 3(2), 7-11.
- Wheeler, D. M., & Dodd, M. B. (1995). Effect of aluminium on yield and plant chemical concentrations of some temperate legumes. *Plant and Soil*, 173(1), 133-145
- Whitney, E. N., & Rolfes, S. R. (2015). *Understanding nutrition*. Cengage Learning.
- Willhite, C. C., Ball, G. L., & McLellan, C. J. (2012). Total allowable concentrations of monomeric inorganic aluminium and hydrated aluminium silicates in drinking water. *Critical Reviews in Toxicology*, 42(5), 358-442.
- Williams, C. (2011). Research methods. [JBER]. *Journal of Business & Economics Research*, 5(3), 1-14
- Wills, M. R., & Savory, J. (1989). Aluminium and chronic renal failure: sources, absorption, transport, and toxicity. *Critical Reviews in Clinical Laboratory Sciences*, 27(1), 59-107.
- World Health Organization. (2003). *Aluminium in drinking-water: background document for development of WHO Guidelines for*

- drinking-water quality* (No. WHO/SDE/WSH/03.04/53). World Health Organization
- World Health Organization. (2004). *The World health report: 2004: changing history*. World Health Organization.
- World Health Organization. (2011). Evaluation of certain food additive and contaminants. *World Health Organization Technical Report Series*, (960), 335-347
- Wu, Z., Du, Y., Xue, H., Wu, Y., & Zhou, B. (2012). Aluminium induces neurodegeneration and its toxicity arises from increased iron accumulation and reactive oxygen species (ROS) production. *Neurobiology of Aging*, 33(1), 199-211.
- Xu, Q., Wang, Y., Ding, Z., Song, L., Li, Y., Ma, D., ... & Zhang, H. (2016). Aluminium induced metabolic responses in two tea cultivars. *Plant Physiology and Biochemistry*, 101, 162-172.
- Yang, S. P., & Tsai, R. Y. (2006). Complexometric titration of aluminium and magnesium ions in commercial antacids. An experiment for general and analytical chemistry laboratories. *Journal of Chemical Education*, 83(6), 906-914
- Yokel, R. A., & McNamara, P. J. (2001). Aluminium toxicokinetics: an updated minireview. *Pharmacology & Toxicology: MiniReview*, 88(4), 159-167.
- Yokel, R. A., Hicks, C. L., & Florence, R. L. (2008). Aluminium bioavailability from basic sodium aluminium phosphate, an approved food additive emulsifying agent, incorporated in cheese. *Food and Chemical Toxicology*, 46(6), 2261-2266.

- Zatta, P. (2000). Aluminium and Health: Recommendations. First International Conference in Metals and Brain. From Neurochemistry to Neurodegeneration. University of Padova, Italy, [Retrieved 20–23February 2022] <http://www.bio.unip.it/~zatta/metals/documen2.htm>
- Zendehboodi, Z. (2018). Cytotoxicity and genotoxicity effects of water boiled in aluminium vessels on *Allium cepa* root tip cells. *Journal of Environmental Health Science and Engineering*, 16(2), 337-341
- Zhang, G., Hoddinott, J., & Taylor, G. J. (1994). Characterization of 1, 3- β -D-glucan (callose) synthesis in roots of *Triticum aestivum* in response to aluminium toxicity. *Journal of Plant Physiology*, 144(2), 229-234.
- Zhang, W. H., Rengel, Z., Kuo, J., & Yan, G. (1999). Aluminium effects on pollen germination and tube growth of *Chamelaucium uncinatum*. A comparison with other Ca^{2+} antagonists. *Annals of Botany*, 84(4), 559-564.
- Zheng, S. A., Zheng, X., & Chen, C. (2012). Leaching behavior of heavy metals and transformation of their speciation in polluted soil receiving simulated acid rain. *PloS One*, 7(11), 44-52
- Zotov, R., Meshcheryakov, E., Livanova, A., Minakova, T., Magaev, O., Isupova, L., & Kurzina, I. (2018). Influence of the composition, structure, and physical and chemical properties of aluminium-oxide-based sorbents on water adsorption ability. *Materials*, 11(1), 132-143

Zouboulis, C. C., Bechara, F. G., Fritz, K., Kurzen, H., Liakou, A. I., Marsch, W. C.,... & of Dermatology, E. S. (2012). S1 guideline for the treatment of hidradenitis suppurativa/acne inversa*(number ICD-10 L73. 2). *Journal der Deutschen Dermatologischen Gesellschaft= Journal of the German Society of Dermatology: JDDG*, 10(1), 11-31.

APPENDICES



APPENDIX A: Cook Food Sample going through laboratory analysis



APPENDIX B: Cooking of Tomato Food Sample in Utensil



APPENDIX C: Cooking Nkontomire Food Sample in Utensil



APPENDIX D: Cooking Rice Food Sample in Utensil



APPENDIX E: Tomato Laboratory Analysis After Filtration